This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE

MEDICAL LIBRARY

SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
SEMPER VIRENS
1885

AMERICAN BANK NOTE CO. LITHO

PLATE I.

Normal infant at Term: 7/10 Natural Size
Case 3.

PEDIATRICS

THE

HYGIENIC AND MEDICAL TREATMENT

OF

CHILDREN

BY

THOMAS MORGAN ROTCH, M.D.

PROFESSOR OF THE DISEASES OF CHILDREN, HARVARD UNIVERSITY

ILLUSTRATED

PHILADELPHIA
J. B. LIPPINCOTT COMPANY
1896

Copyright, 1895,
by
J. B. Lippincott Company.

Electrotyped and Printed by J. B. Lippincott Company, Philadelphia, Pa., U. S. A.

342
190

TO

NELSON SLATER BARTLETT,

IN RECOGNITION OF HIS INTEREST AND ENTHUSIASM
IN PROMOTING

THE STUDY OF PEDIATRICS.

17852

PREFACE.

A FEW words are perhaps needed to explain what I have undertaken to do in the following pages, and the method of arrangement and classification which has been employed. There has been no attempt to make such classifications as infectious and non-infectious diseases, because our knowledge of the former is increasing the number of that class so rapidly that for me it no longer constitutes a practical division for teaching. The book begins with a consideration of the infant at birth, and follows it through its various stages of development up to puberty. After dwelling rather more at length on normal development than is usual in works on pediatrics, the abnormal conditions are discussed. Beginning with the diseases which would naturally be met with in the early periods of life, and devoting considerable space to my observations on the blood of infants and of young children, the diseases of the different organs are then considered.

With the exception of a few rare diseases of which it was impossible to get satisfactory types, the illustrations represent actual cases of my own, heretofore unpublished. The colored illustrations have received my closest attention, and the patients were seen personally with the artist, so as to insure accuracy.

The establishment of milk-laboratories during the last three years has marked a new era in preventive medicine, and has made possible the scientific feeding of infants. As I believe that the medical treatment of the various abnormal conditions arising in infants is in the future to be largely dietetic rather than by means of drugs, I have given unusual prominence to the part of the work which is devoted to feeding.

I have also endeavored, in conjunction with my colleagues in the American Pediatric Society, to simplify the nomenclature of the various diseases, in order that physicians in different localities should by using identical names be the better able to aid one another in their investigations. A revision of the nomenclature of gastro-enteric diseases and of those of the mouth was especially called for on account of the changes which have followed our increasing knowledge of the etiology of these diseases.

T. M. ROTCH.

BOSTON, MASS., October, 1895.

CONTENTS.

DIVISION I.

PAGE
INTRODUCTORY.—The infant at term 17
LECTURE I.—Fœtal circulation 19
LECTURE II.—Vernix caseosa 23
  Cord 24
  Spine 26
  Neck 30
  Head 30
  Thorax 38
  Abdomen 44
  Pelvis 48
  Bladder 48
  Uterus 48
  Temperature 48
  Pulse 48
  Respiration 48
  Height 49
  Weight 49
  Vitality 49
  Hands 49
  Feet 49
  Bone marrow 51
  Functions 51
  Blood 52
  Lymphatic system 52
  Urine 53
  Intestinal discharges 53

DIVISION II.

NORMAL DEVELOPMENT.

LECTURE III.—Spine 55
  Neck 59
  Head 60
  Thorax 70
LECTURE IV.—Abdomen 77
  Temperature 94
  Pulse 94
  Respiration 96
  Height 96
  Weight 97
  Feet 105
  Bone marrow 107
  Skin 107

LECTURE IV.—*Continued.*
Cord
Functions
Blood
Lymphatic system
Thyroid body
Urine
Intestinal discharges
Infantile skeletons
Normal infants
Topographical anatomy of the early periods of life

DIVISION III.

HYGIENE OF THE NURSERY.

LECTURE V.—The nursery
Intertrigo
Seborrhœa capitis of infants
Clothing
Feet and shoes
Sleep
Out-door air
Nursery-maids
Mouth
School
Importance of correcting defects of posture
Vaccination

DIVISION IV.

FEEDING.

LECTURE VI.—The general principles underlying all methods of infant feeding
LECTURE VII.—The first nutritive period
1. Maternal feeding
2. Direct substitute feeding
LECTURE VIII.—The first nutritive period (continued)
3. Indirect substitute feeding
LECTURE IX.—Indirect substitute feeding (continued)
General remarks on substitute feeding
Comparison of woman's and cow's milk
Milk-laboratories
LECTURE X.—Home modification
General remarks on artificial foods for infants
LECTURE XI.—The second and third nutritive periods

DIVISION V.

LECTURE XII.—Premature infants

DIVISION VI.

GENERAL PRINCIPLES OF EXAMINATION AND TREATMENT.

LECTURE XIII.—Method of examining a sick child
Drugs

DIVISION VII.

THE BLOOD IN INFANCY AND CHILDHOOD.

PAGE
LECTURE XIV.—Literature 329
  Nomenclature 330
  Blood-key 331
  Methods 332
  Chemistry 334
  Origin 336
  Fœtal blood 337
  The normal conditions of the blood in early life 339
LECTURE XV.—The pathology of the blood in early life 348
  Premature infants 348
  New-born infants 349
  Leucocytosis 350
  Leucæmia 351
  Oligocythæmia 353
  Primary anæmia 355
  Chlorosis 355
  Anæmia progressiva perniciosa 356
  Anæmia infantum pseudo leukæmica (von Jaksch) 359
  Secondary anæmias 365
  Treatment of diseases of the blood 366
  Congenital syphilis 367
  Rhachitis 368
LECTURE XVI.—The blood in individual diseases 370
  Typhoid fever 370
  Scarlet fever 371
  Measles 371
  Variola 371
  Diphtheria 372
  Pneumonia 372
  Broncho-pneumonia 373
  Pneumonia and empyema 373
  Empyema 373
  Miliary tuberculosis 374
  Tubercular meningitis 374
  Hydrocephalus 375
  Chorea 376
  Nephritis 376
  Tubercular peritonitis 377
  Infantile atrophy 377
  Periostitis 378
  Scorbutus 379
  Icterus neonatorum 379
  Sclerema neonatorum 379
LECTURE XVII.—Parasites of the blood 380
  Literature of the blood in early life 398

DIVISION VIII.

DISEASES OF THE NEW-BORN.

LECTURE XVIII.—Maternal impressions 404
  The head 404
  The neck 416
LECTURE XIX.—The trunk 418

LECTURE XX.—The extremities
General diseases

DIVISION IX.

LECTURE XXI.—Diseases of the skin

DIVISION X.

SYPHILIS—ERYSIPELAS—THE EXANTHEMATA.

LECTURE XXII.—Syphilis
LECTURE XXIII.—Erysipelas
LECTURE XXIV.—Variola—Varicella
LECTURE XXV.—Scarlet fever
LECTURE XXVI.—Measles
Rubella

DIVISION XI.

DISEASES OF THE NERVOUS SYSTEM AND THE MYOPATHIES.

LECTURE XXVII.—Introduction
LECTURE XXVIII.—I. Organic nervous diseases
Brain
LECTURE XXIX.—Tubercular meningitis
LECTURE XXX.—Thrombosis of the cerebral sinuses
Hydrocephalus
LECTURE XXXI.—Cerebral abscess
Cerebral paralysis
Athetosis
Intra-cranial tumors
Intra-cranial syphilis
Idiocy
Mirror writing
LECTURE XXXII.—Cord
Myelitis
Poliomyelitis anterior
Paralysis caused by caries of the spine
Hereditary ataxia (Friedreich's disease)
Locomotor ataxia
Syringomyelia
LECTURE XXXIII.—Brain and cord
Multiple cerebro-spinal sclerosis
Cerebro-spinal meningitis
LECTURE XXXIV.—Peripheral nerves
Neuritis
Paralysis of the new-born
Neuralgia
LECTURE XXXV.—II. Nervous diseases presumably organic
Chorea
Epilepsy
Insanity
LECTURE XXXVI.—III. Functional nervous diseases. (1) Probably central
Hysteria
Hypnotism

LECTURE XXXVI.—*Continued.* PAGE

Catelepsy 785
Simulated diseases 785
Insolation 786
Concussion 788
Temporary amnesia 789
Temporary aphasia 789
Arrested psychical development 740
Retarded speech 740
Headaches 741
Vertigo 743
Sensitive spine 744
Tetany 744
Pavor nocturnus (central) 745
LECTURE XXXVII.—IV. Functional nervous diseases. (2) Reflex 746
Pavor nocturnus (peripheral) 746
Dental reflex 746
Reflex nystagmus 747
Reflex of the ear 747
Reflex of the larynx 747
Paroxysmal gasping 749
Reflex of the lung 750
Reflex cough 751
Reflex of the heart 751
Reflex of the stomach 752
Reflex of the bladder 752
Reflex of the vagina 752
Reflex of the rectum 752
LECTURE XXXVIII.—Convulsions 754
Tremor 762
LECTURE XXXIX.—The myopathies 768
Progressive muscular atrophy 768
Pseudo-hypertrophic muscular paralysis 768
Myotonia congenita (Thomsen's disease) 778

DIVISION XII.

DISEASES OF THE MOUTH, NOSE, NASO-PHARYNX, AND PHARYNX.

LECTURE XL.—Stomatitis catarrhalis 776
Stomatitis herpetica 779
Stomatitis ulcerosa 781
Stomatitis mycetogenetica 784
Glossitis 793
Microglossia 794
Macroglossia 794
Difficult dentition 795
LECTURE XLI.—Diseases of the nose, naso-pharynx, and pharynx 801
LECTURE XLII.—Diphtheria 821

DIVISION XIII.

DISEASES OF THE ŒSOPHAGUS, STOMACH, AND INTESTINE.

LECTURE XLIII.—Introduction 834
Œsophagus 834
Stomach and intestine 836

LECTURE XLIV.—Diseases of the stomach
LECTURE XLV.—Diseases of the intestine

DIVISION XIV.

LECTURE XLVI.—Diseases of the liver, pancreas, spleen, and peritoneum

DIVISION XV.

LECTURE XLVII.—Diseases of the kidneys, bladder, and genitals
- Kidneys
- Supra-renal capsules
- Bladder
- Genitals

DIVISION XVI.

DISEASES OF THE LARYNX, TRACHEA, LUNGS, AND PLEURA.

LECTURE XLVIII.—Laryngospasmus
- New growths
- Foreign bodies
- Œdema
- Laryngitis

LECTURE XLIX.—Diseases of the lungs
- Bronchitis
- Broncho-pneumonia
- Atelectasis
- Lobar pneumonia
- Gangrene
- Tuberculosis
- Pertussis
- Asthma 1
- Periodic catarrh 1

LECTURE L.—Diseases of the pleura 1

DIVISION XVII.

DISEASES OF THE HEART AND PERICARDIUM.

LECTURE LI.—Diseases of the heart 1
LECTURE LII.—Diseases of the pericardium 1

DIVISION XVIII.

UNCLASSIFIED DISEASES.

LECTURE LIII.—Rhachitis 1
- Scorbutus 1
- Rheumatism 1
- Purpura 1
- Diabetes 1
- Tuberculosis 1
- Epidemic influenza 1
- Diseases of the thyroid gland 1
- Diseases of the cervical lymph-glands 1
- Parotitis 1
- Diseases of the ear 1

PEDIATRICS.

DIVISION I.

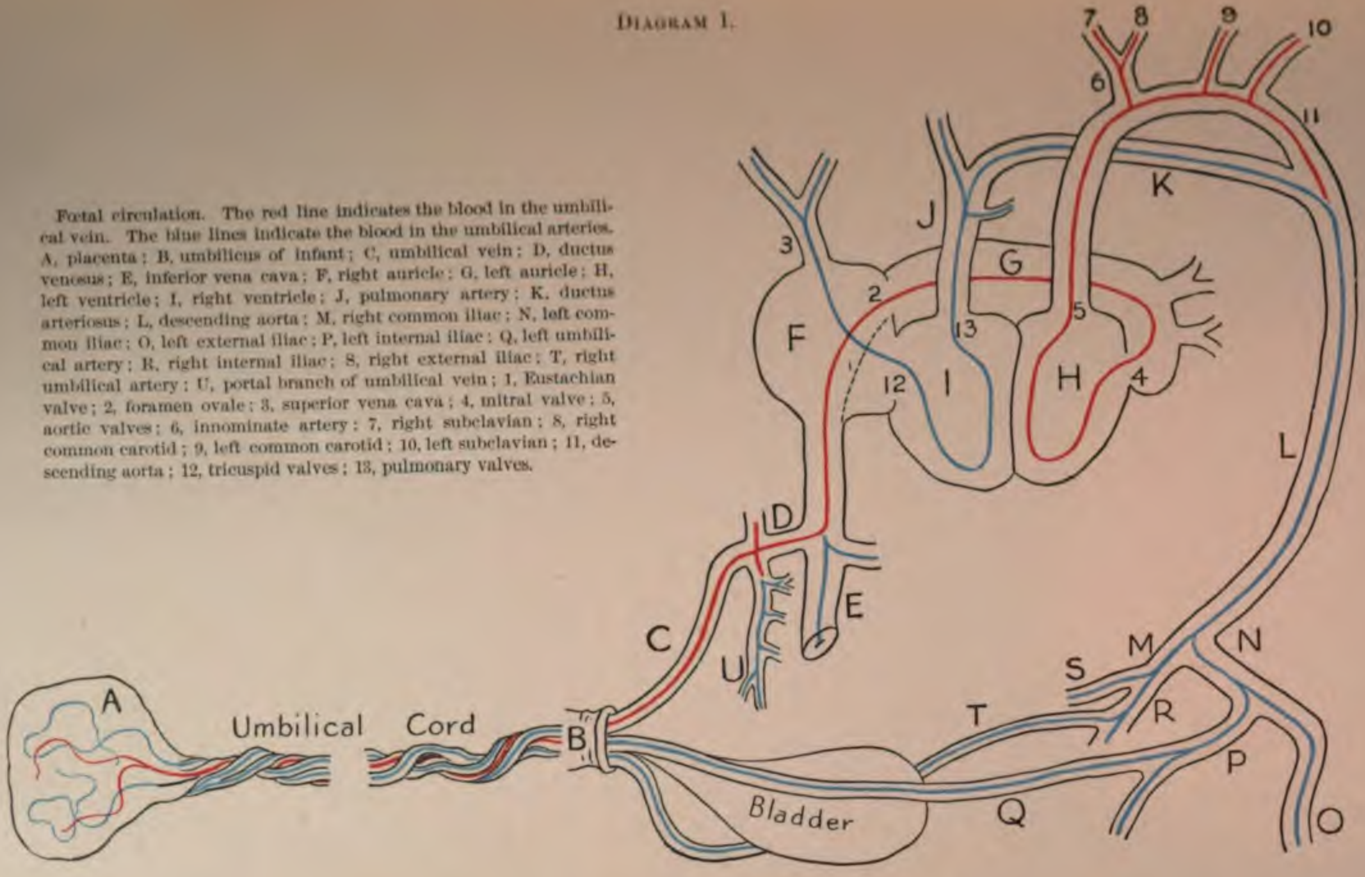
INTRODUCTORY.—THE INFANT AT TERM.

LECTURE I.

INTRODUCTORY.—THE FŒTAL CIRCULATION.

GENTLEMEN,—We are to-day beginning the study of a branch of medicine which will be of the greatest practical importance to you in your future careers. Those of you who enter into general practice will at once be called upon to treat infants and children. The proper appreciation of the sensitive temperaments and needs of this class of patients will be of great aid in successfully establishing your practice among those whose favorable opinion may make or mar your professional success. The difficulties to be surmounted in correctly diagnosticating and treating young children are far greater than those which you encounter in adult life. The reason for this is that for adult cases you have some standard by which you can be guided, being yourselves adults. What standard, however, have you for the feelings and sensitive organization of the child? None within yourselves; it must all come from long and patient observation, with its resulting experience. The mere knowledge that certain diseases exist, and the usual methods of diagnosticating them, prove to be very inadequate when we are brought face to face with a sick and fretful child, or with an infant who is unable to describe its symptoms. Much additional knowledge is needed to enable us to understand the variety of symptoms which may arise in the same disease according to the age and individuality of the patient. It is now well recognized that there is a necessity for making a special study of children beyond what is learned in the general clinical study of adults. As our knowledge advances, we learn to appreciate that the various methods of treatment must be modified to correspond not so much to the special disease as to the special group of symptoms brought abou the age of the individual and the phase of its development. In stud then, the different stages of development in children, we are in re acquiring an alphabet, which when once thoroughly mastered will enabl to read the otherwise obscure language presented to us for translation b various diseases of early life. The proper method of learning to unders sick infants and children is first to notice their peculiarities in health a follow these peculiarities through the different stages of their develop up to puberty. Thus, a pulse which would indicate an abnormal co tion in the adult, or a convulsion which would be of serious import in older subject, may often be but physiological or of slight consequence i child. In fact, there are a large number of physiological and anato truths concerning the young the knowledge of which will simplify great degree otherwise almost insurmountable difficulties in diagnosis. lack of this preliminary training, this alphabet, places the student wh endeavoring to understand diseases in children, in the position of atte ing to read without having first learned his letters. It is our provinc this course of lectures to begin with the human being at birth, to study it is presented to us in the early hours of life, and to follow it in its deve ment during the periods of infancy and childhood up to the age of pub It then approaches so nearly in its development to the adult that its dis assume the type of adolescence, and your studies carry you into the prov of general clinical medicine. For purposes of simplicity, we speak of inf and children, the anatomical and physiological conditions being sufficie apparent to warrant this distinction between them. The period of inf is usually spoken of as covering about the first two years of life. Its distinctive features are presented in the first twelve months, the second y month by month, rapidly approaching the conditions which exist in ch hood. The second year, however, is influenced to such a degree by various growing functions and tissues that its picture both in health in disease resembles more closely the infant than the child. Childhoo empirically reckoned from the end of infancy to puberty, or the begin of adolescence. A distinction must be made between the sexes, the becoming a fully-developed woman some years before the boy beco a man. The age of puberty is usually reckoned as beginning from twelfth to the fourteenth year. Much latitude as to age, however, m be given for the special idiosyncrasy of the individual, and also for climate, as it has been found that children who live in a warm clim arrive at the age of puberty much earlier than those who are exposed the lower ranges of temperature. In taking the period of birth as a st ing-point for our studies we must not overlook the fact that it is simpl stage of development with which we are dealing, and not a perfected bei The better, therefore, you understand the evolution of the embryo to infant, the better will you be prepared to appreciate the evolution of infant to the child and of the child to the adult. It is especially import

DIAGRAM I.



Fœtal circulation. The red line indicates the blood in the umbilical vein. The blue lines indicate the blood in the umbilical arteries. A, placenta; B, umbilicus of infant; C, umbilical vein; D, ductus venosus; E, inferior vena cava; F, right auricle; G, left auricle; H, left ventricle; I, right ventricle; J, pulmonary artery; K, ductus arteriosus; L, descending aorta; M, right common iliac; N, left common iliac; O, left external iliac; P, left internal iliac; Q, left umbilical artery; R, right internal iliac; S, right external iliac; T, right umbilical artery; U, portal branch of umbilical vein; 1, Eustachian valve; 2, foramen ovale; 3, superior vena cava; 4, mitral valve; 5, aortic valves; 6, innominate artery; 7, right subclavian; 8, right common carotid; 9, left common carotid; 10, left subclavian; 11, descending aorta; 12, tricuspid valves; 13, pulmonary valves.

to understand the stage of development which exists just before birth, for on this depends the knowledge whether we have a physiologically and anatomically normal being before us, or one that is abnormal. Remember that disease does not merely mean a pathological change in the tissues, but, as is especially well exemplified in the infant, may simply mean a retardation or arrest of development. Thus, what would be perfectly normal anatomically at the seventh month of intra-uterine life may at birth be abnormal, and hence constitute a disease. In like manner what may be normal at birth may if it persists into the second and third weeks become an abnormal condition. Disease, therefore, is a relative term. We may, however, simplify our classification of diseases by adopting two broad divisions corresponding to the changes which take place during intra- and extra-uterine life. By congenital diseases we mean those resulting from changes occurring during intra-uterine life. These may arise from an arrest of development or from a continuation of normal intra-uterine conditions beyond the usual period of their cessation; also those which are caused by pathological processes such as inflammation. By acquired, we mean a pathological condition of existing tissues occurring after birth, and without regard to the stage of development.

If we thoroughly understand the anatomical conditions existing just before birth, we can intelligently examine the young human being as it emerges from the uterus, and can judge in the early days of its existence whether we have under our care a normal infant or one that is to need special treatment.

FŒTAL CIRCULATION.—The chief anatomical change which takes place at birth is the transition from the intra-uterine circulatory mechanism to a form adapted to extra-uterine life; in other words, from the oxygenation of the blood through the placenta to the same process carried on by the lungs. A general knowledge of the fœtal circulation is, then, evidently of considerable importance for you to acquire, especially when you consider that a large proportion of the cases of congenital heart disease which you will be called upon to diagnosticate is represented by perfectly normal prenatal conditions, such as *absence of the ventricular septum*, an *open ductus arteriosus*, or a *patent foramen ovale*.

This diagram (Diagram 1) represents the course of the (red) oxygenated blood from the placenta to the infant, and that of the darker (blue) deoxidized blood from the infant back to the placenta. We must consider that in the fœtus the lungs are in a collapsed, inert condition, performing no part in the fœtal economy, but remaining quiescent until called upon to perform their special function at birth. The true lung of the fœtus, therefore, is represented by the placenta of the mother. It is here that the blood is oxygenated, and is carried by means of the umbilical vein directly through the umbilicus of the fœtus to the liver, as seen in the diagram. In the liver, the umbilical vein divides into three branches: (1) the smallest, carries the blood directly to the liver tissue, whence it is returned as in the adult to the inferior cava by the hepatic veins; (2) the largest portion meets and mixes with blood from the portal system, and is distributed with it to the liver; the remaining portion is carried, by a vessel called the *ductus veno* directly to the inferior cava, where it meets the deoxidized blood from lower extremities, mixes with it, and is carried to the right auricle: h instead of passing as in the adult into the right ventricle, it is direc by a membrane, called the *Eustachian valve*, through an opening betw the two auricles, called the *foramen ovale*, into the left auricle. It t passes into the left ventricle through the mitral valve, and thence thro the aortic valve into the aorta. The greater part of the blood-curren then carried by the carotid and subclavian arteries to the head and up extremities, where, after doing its work in vitalizing the tissues and tak up their waste (a small portion also passing, as usual, into the descend aorta), it is returned as deoxidized blood through the veins to the supe cava into the right auricle, thence through the tricuspid valves into right ventricle, and up through the pulmonary artery, where a small port is distributed as usual to the lungs, while the remaining portion is carr directly over to the descending aorta by a vessel called the *ductus arterio* It here mixes with the small portion of oxygenated aortic blood mentio above, and passes down the aorta, being distributed on its way, as in adult, until it reaches the internal iliac arteries. From these arteries i carried, by branches called the *umbilical arteries*, through the umbilicus b to the cord and placenta. Thus, by simply referring to this diagram, we c tell at a glance which part of the young infant should be most develop and the reasons for it. A noticeable point of clinical interest, in tracing t course of the fœtal circulation, is that the fresh oxygenated blood is mai carried to the liver, head, and upper extremities, while the devitalized blo is distributed to the thorax and lower extremities. We should theref expect, and we shall find it to be true, when we examine a normal new-b infant, that the head is larger than the thorax, that the abdomen is promin from containing the large liver, and that the legs are insignificant a poorly developed.

When the placental circulation is cut off, an increased amount of blo is carried by the pulmonary artery to the lungs, and by degrees the fœ circulation is replaced by that of extra-uterine life.

The *ductus venosus* and *ductus arteriosus* become fibrous cords.

The *Eustachian valve* disappears.

The *foramen ovale* closes.

The *umbilical vein* and *umbilical arteries* become obliterated, with t exception of the lower parts of the latter.

All these changes, however, do not take place simultaneously, which is point to be remembered in making a differential diagnosis of cardiac disea during the first ten days of infancy. We should therefore endeavor to be in mind at about what time these changes take place. The following tab will, I think, assist you in accomplishing this:

TABLE 1.

POST-NATAL CHANGES OF FŒTAL CONDITIONS.

Ductus Venosus.—The ductus venosus becomes a fibrous cord in the fissure of the ductus venosus in from two to five days.

Eustachian Valve.—The intra-uterine function of the Eustachian valve practically disappears at once at birth, but its remains can be found for an indefinite period, as you see in this heart dissected by Dr. F. Dexter (Fig. 19, facing page 74).

Foramen Ovale.—The foramen ovale usually closes about the tenth day, but the upper part sometimes never closes. The closed foramen ovale is seen in this same heart dissected by Dr. F. Dexter (Fig. 19, facing page 74).

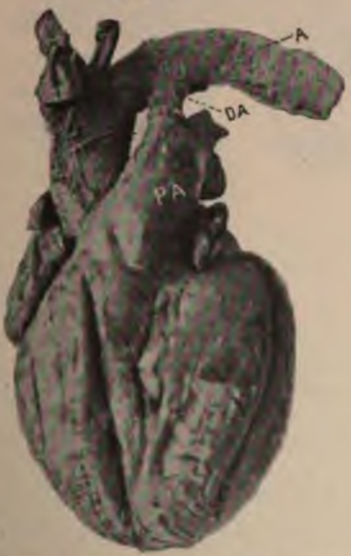
Ductus Arteriosus.—The ductus arteriosus is about 1.5 cm. ($\frac{3}{4}$ inch) long, has a diameter of about .25 cm. ($\frac{1}{8}$ inch), and is usually, so far as being pervious to the blood is concerned, obliterated in from four to ten days. Its remains, forming a fibrous cord connecting the pulmonary artery and the aorta, can be seen in this heart dissected by Dr. F. Dexter (Fig 20, facing page 74).

Umbilical Vein.—The umbilical vein becomes the round ligament of the liver, and is obliterated in from two to five days. As pointed out by Jacobi, it differs from the arteries very much less than is usual with the veins and arteries in other parts of the body. Its muscular layer is very large and strong.

Umbilical Arteries.—The umbilical arteries in their upper parts become obliterated in from two to five days, forming the anterior true ligaments of the bladder, while the lower parts remain pervious and form the superior vesical arteries. The umbilical arteries are usually thick and strong, owing to the great development of their muscular layer.

Thus you will observe that during the first two weeks of infancy we may have conditions existing physiologically which after that time would become pathological, and hence, to be well grounded in the diagnosis of

FIG. 1.



Heart, natural size, at two days. A marks the aorta; PA marks the pulmonary artery; DA marks the ductus arteriosus.

disease in the infant, we must appreciate the importance of these facts retain them for future use.

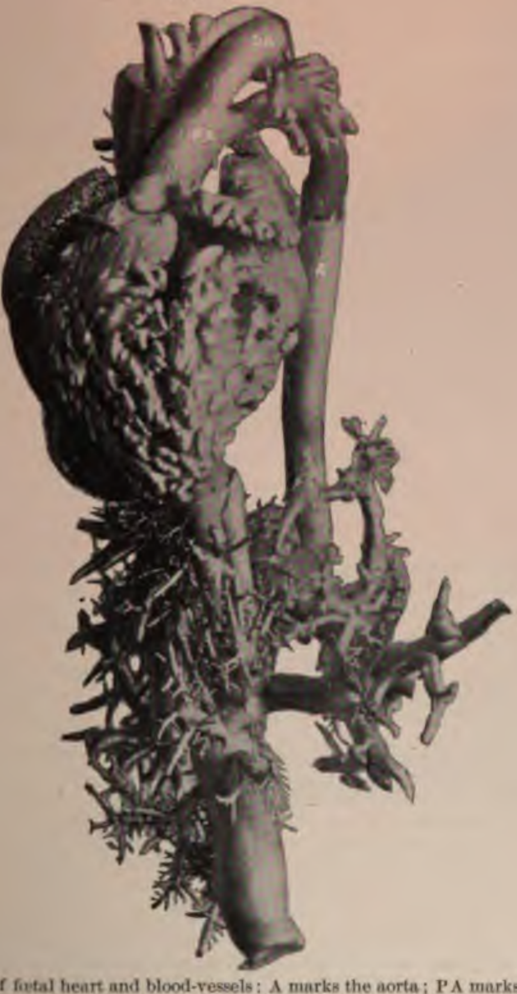
The heart is the organ on which, from the importance of its functio the system in general, our interest is at once centred at birth. It is v therefore, for you to know exactly how it should look normally, and l large it should be.

This heart (Fig. 1, page 21) was taken from an infant two days old is of normal size, and shows the *ductus arteriosus* connecting the *aorta* the *pulmonary artery.*

This metallic injection of the heart and blood-vessels of the fœtus (: 2), made by Dr. S. J. Mixter, shows you very clearly the *ductus arteri* and the ramifications of the various branches of the *pulmonary artery* the *aorta.*

You must, of course, remember that where a cavity existed in the h and vessels of the fœtus, the metal preparation shows a solid mass. T you can learn exactly the appearance of the inner surfaces of the right left auricles and ventricles, the pulmonary artery, the ductus arterio and the aorta.

FIG. 2.



Metallic injection of fœtal heart and blood-vessels: A marks the aorta; P A marks the pulmonary artery; D A marks the ductus arteriosus.

LECTURE II.

THE INFANT AT TERM.

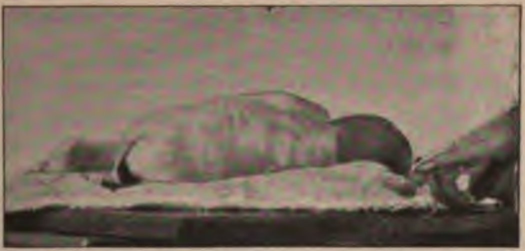
Vernix Caseosa—Cord—Spine—Neck—Head—Thorax—Abdomen—Temperature—Pulse—Respiration—Height—Weight—Vitality—Hands—Feet—Bone Marrow—Functions—Blood—Lymphatic System—Urine—Intestinal Discharges.

By the infant at term we mean one that has been born at the termination of what is considered the usual period of pregnancy, two hundred and eighty days.

I shall by showing you actual cases of normally developed infants in the early days of life endeavor to teach you what conditions are important for you to remember as distinguishing marks from the abnormal cases which I shall present for your inspection later.

This infant (Case 1), one hour old, represents the appearance of a normally developed fœtus when it first emerges from the uterus. The reddened skin, as you see, is covered in many parts thickly by a substance made up of the contents of the amniotic sac, in which the fœtus has been floating, and of the excretion of the sebaceous glands. This substance, which is called the *vernix caseosa*, must be removed in order that we may study the infant as it normally appears in the first stage of its existence. It is evident, however, that the infant is born with highly developed sebaceous glands, which at times produce a secretion so excessive as to be difficult to get rid of. In certain rare cases also this sebaceous matter is so universal and so impenetrable as to constitute a disease of serious import, and at times even to cause death. Infants are also born with the skin almost entirely free from the *vernix caseosa*, so that it is not necessarily present, and in fact I have had to wait for some time before I could get a subject which would present this condition sufficiently marked for illustration. You will also notice the dark fæcal discharge, called meconium, which is coming from the anus, and which is so characteristic of the early hours of life.

Case 1.



Infant immediately after birth, covered almost entirely with the vernix caseosa, and having a discharge of meconium.

This infant was shown to you merely to represent the *vernix caseosa*, while what we are especially called upon to deal with is the new-born infant freed from its amniotic covering and with its entire surface prepared for our inspection.

For the purpose of illustrating this condition I will now show you another infant (Case 2, Frontispiece):

A male, two days old. Its birth-weight was 3800 grammes (8½ pounds); its length is 480 mm. (19 inches); the circumference of its head is 34 cm. (13½ inches); the circumference of its thorax is 33 cm. (13 inches); and the circumference of its abdomen is 35.5 cm. inches).

The infant has just been bathed, and presents the color of a healt skin reacting normally to the temperature of the water, 36.6° C. (98° F and that of the room, 21.1° C. (70° F.). I have chosen this particular c as representing best what a strong healthy infant should look like. I sh presently show you that it is somewhat larger than the average infant two days. In reality, however, so far as my experience goes, the size this infant corresponds very closely to that of most healthy infants that born outside of hospitals in families who live in comfortable homes of th own and in healthy localities. The delicate pink of the skin, the we rounded body and limbs, the vigorous cry, the warm extremities, alrea beginning to move with activity, and the strong grasp of the little han all justify me in showing you what at this age may be looked upon as t picture of health.

The hair at birth is often thick, dark, and quite long, perhaps 2 t cm. (1 or 2 inches); but we also frequently find the hair to be short, fi some shade of light brown, small in amount, and, as you see on examini this infant's head, the temples to be bald and the hair to come down t rounded point on the forehead. The eyes are almost always as you see this case, half open when awake, expressionless, and of a dull grayish bl Notice also what your study of the fœtal circulation explained so well, t large head in comparison with the thorax, the arms more rounded and lar in proportion to the legs, and the prominent abdomen.

CORD.—I have had the dressing removed in order that you should able to study the cord minutely. You see how it is already drying preparatory to falling off on the sixth or seventh day. The cord in heal does not often receive much attention from the physician, and usually it familiar in its appearance to the nurse only. Yet it is quite important you to know how it should look normally up to the day when it separat from the umbilicus, for at times you are called upon to decide whether it diseased, and unless you are familiar with it in health your opinion will n be of much value as to whether you have an abnormal condition before yo You see the slightly reddened areola where it joins and is to part from t abdominal wall. The three vessels are easily picked out, and differ in colo The two dark, almost black, lines twisting in and out around the sing greenish-yellow and broader line are the umbilical arteries. The flat yello line is what remains of the umbilical vein.

Palpation, percussion, and auscultation show that the heart has about t same proportionate position in reference to the lungs as is found in the adul but that the liver occupies much more space, coming fully 1 to 2 cm. (½ 1 inch) below the edge of the ribs in the right hypochondriac and the ep gastric regions, and encroaching on the lung-space in the right back to th extent of fully one rib and interspace. The testicles have descended, an the bladder, which is evidently full of urine, presents an area of dulness o about 2 cm. (1 inch), just above the pubes in the median line. This corroborates the important fact, to which I shall refer later, that the bladder is an abdominal rather than a pelvic organ in the infant and the young child. The dull area of the spleen corresponds in its position to that found in the adult, but is scarcely perceptible.

I should like you to retain carefully in your minds this perfect picture of a human being at term, for it is the central point from which will diverge many interesting conditions of the later and higher development which I have undertaken to elucidate for you in these lectures.

We shall next study more in detail certain anatomical and physiological truths relating to the infant at term, but having reference to what is usually found to exist in the average infant rather than in the individual.

The figures which I shall present to you must necessarily be accepted in a general way, and will often be found lacking in exactness simply because there are so many exceptions to general rules taken from large numbers of cases. In my own experience, however, they have proved to be so near to the truth as to be exceedingly valuable in my clinical work. I have for many years had them verified in a number of large clinics and in my private practice, and they at least form a very fair basis for you to start with.

I shall now call your attention in a general way to a number of new-born infants of various weights and degrees of development, and show you that there are certain characteristics common to them all and corresponding to the period of birth. I am especially indebted to Professor Thomas Dwight for the assistance which he has given me through his own original investigations and for his verification of my clinical and anatomical work, the results of which I shall now lay before you. You must pardon me if, for the purpose of impressing upon you what I consider of absolute importance, I seem to repeat unnecessarily at times.

Remember also that I do not attempt nor deem it wise to give you the complete anatomy and physiology of the period of life we are studying. I shall merely pick out for your use the practical points in these periods which will aid you in clinical diagnosis and treatment. The great importance of thoroughly understanding the normal anatomy and physiology of human beings before attempting to deal with the morbid conditions which arise in them is now so well recognized that no preliminary remarks are needed to show how vital to all advance in clinical medicine is the proper reading of anatomical and physiological truths. There are several points in the anatomy and physiology of the new-born infant which would be better understood if the fact were borne in mind that in many respects the body at this age is more adapted to its intra-uterine life and to its means of exit into the external world than to the conditions which surround it in extra-uterine life.

Notice these infants a few hours old, as they are held up for your inspection by the nurses. By having one with its face (Case 3) and the other with its back (Case 4) towards you, you can easily follow what I about to tell you of the anatomical conditions characteristic of this e period of life.

This infant's (Case 3) face is, as you see, swollen and the features are of shape. This condition is not uncommon at birth : it comes from pres and will soon pass off.

The cord, you see, has already been dressed with cotton.

The anatomical points so evident at birth as belonging to intra-ute life, and the peculiarities of the fœtal circulation, I have already d upon, and I shall now point out to you the characteristics of the new-l trunk. This is egg-shaped, the larger end being below. The pe as a region hardly exists, and the thorax is very small when comp with the large abdomen. The latter is very large, owing to the dispro tionate development of the liver, presumably a great organ of nutri during fœtal life. A striking peculiarity is the almost complete abs of shoulders, which with the arms are relatively insignificant outgro from the sharp end of the egg. I shall later consider the thorax detail, but I may now mention that it is evident that its small size, want of solidity, and the slight development of the pectoral and shou muscles indicate that its action in respiration must be very different f that in adult life.

The greatest breadth of the trunk is in the region of the lower ribs.

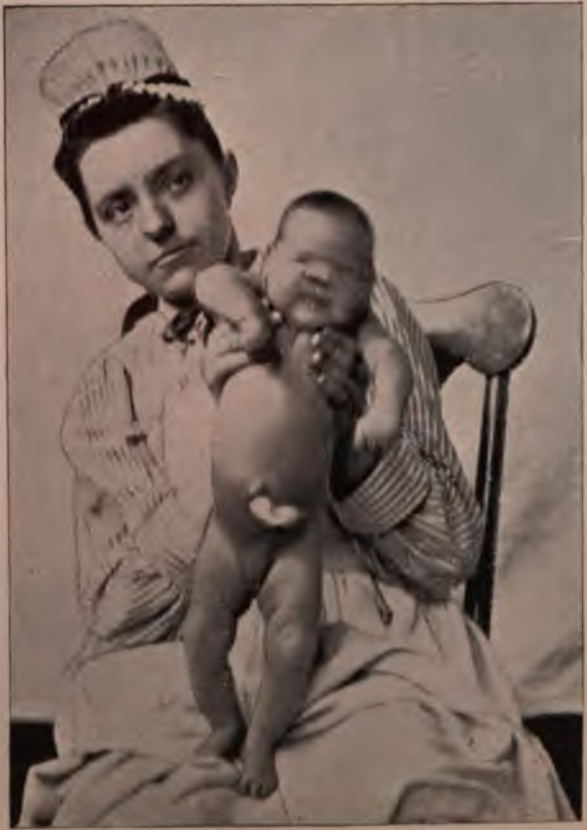
During intra-uterine life, and especially at the time of delivery, g flexibility and compressibility are requisite. Respiration has not yet curred, and the assimilation of nutriment for the growth of the body for preparing the rudiments of future organs has been the function m actively employed. When, therefore, we study the new-born infant must remember that we see it at an essentially transitional stage. Ada tions, the marked utility of which is past, still persist, and new functi are carried on with very imperfect apparatus. These general princi having been stated, I can now discuss more in detail the spine.

SPINE.—One of the most beautiful of anatomical preparations is t cleanly dissected spine of an infant at birth suspended in a jar of alco (Fig. 3).

Owing to the removal of the other parts, its shape (if there be any at t age) is lost, but it is excellent for the study of the component parts. It i wonder of lightness and flexibility. There is little bone and much cartil and fibrous tissue. It can be twisted and bent at will in any directi Looked at critically, it appears relatively broader in proportion to its leng than does the adult spine. The height of the vertebræ is relatively less, a appears even less than it is, from the fact that the broad, narrow, bo nucleus of the vertebral body, which catches the eye, does not represent t whole thickness of the body, as it is embedded in cartilage.

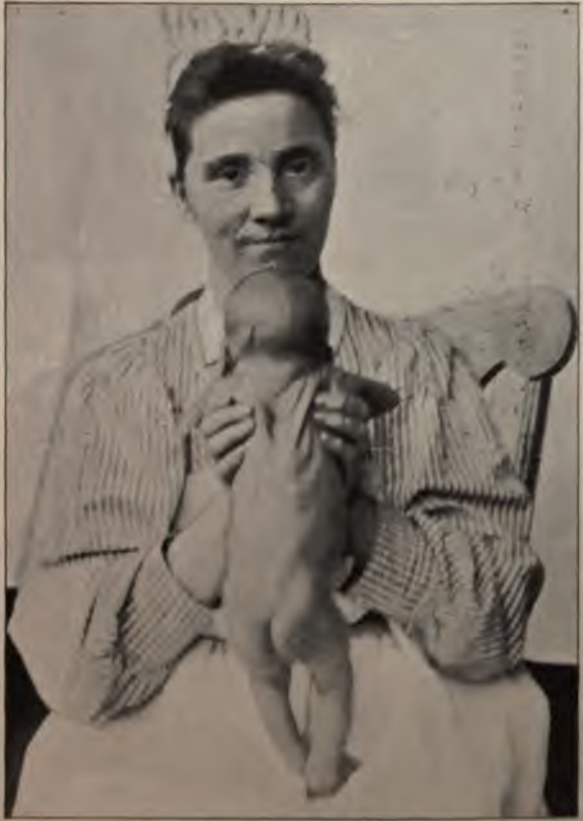
At this early stage of development the whole column is cartilaginou with the exception of the nuclei of the bodies of the vertebræ and those

CASE 3.



Normal new-born infant : front.

CASE 4.



Normal new-born infant : back.

the laminæ on either side, forming a small portion of the body and the beginning of the arch.

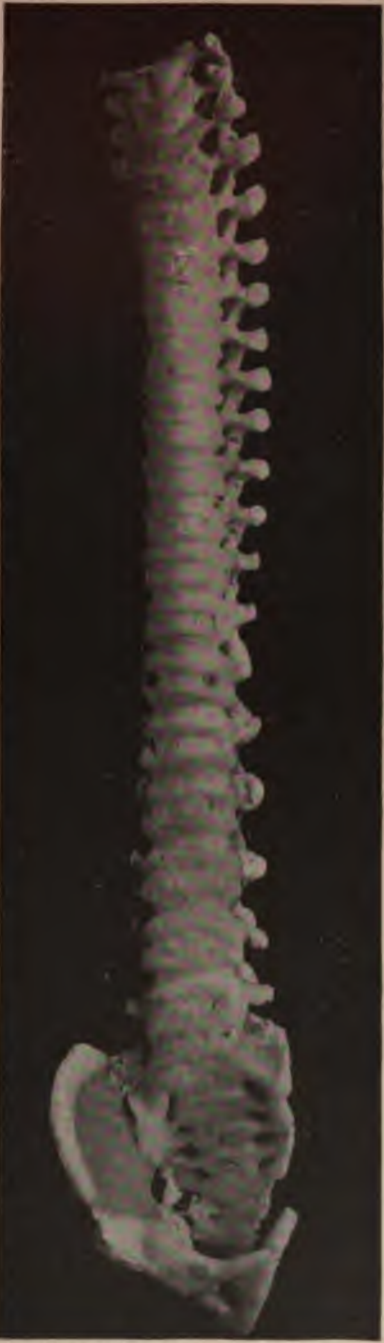
The time of the consolidation of the bodies is not accurately known, but this will be spoken of in the lecture on development.

In the young embryo, the proportion of the neck in the movable part of the spine is greater than that of the loins, a condition which is reversed in the adult, where the neck is less, being a little over one-fifth, and the loins a little less than one-third. In fact, the proportions of the spine change considerably from an early period of intra-uterine life to that of the perfected adult condition. At birth, however, the change has progressed sufficiently to make these two parts very nearly equal. The union of the laminæ to form the spine begins in the upper part of the spine sooner than in the lumbar region. Throughout the greater part they are nearly united, and in some places are quite joined, at birth.

I mention these details not expecting you to remember them, but for future reference in cases where the spine is involved in diagnosis, and perhaps for intelligent orthopedic treatment. What I am about to tell you will also be valuable in directing the care of the normal child in regard to its sitting and standing. You see on examining these infants (Cases 3 and 4) how pliable and easily bent in all directions is the spine, and how their backs can be made to take almost any curve.

You will also understand better what I am about to say if you will examine closely this diagram of three spinal curves, representing (1) the natural curve at birth, (2) the curve which comes especially in the *cervical* region when the infant has learned to sit up and the superincumbent head has to be sup-

FIG. 3.

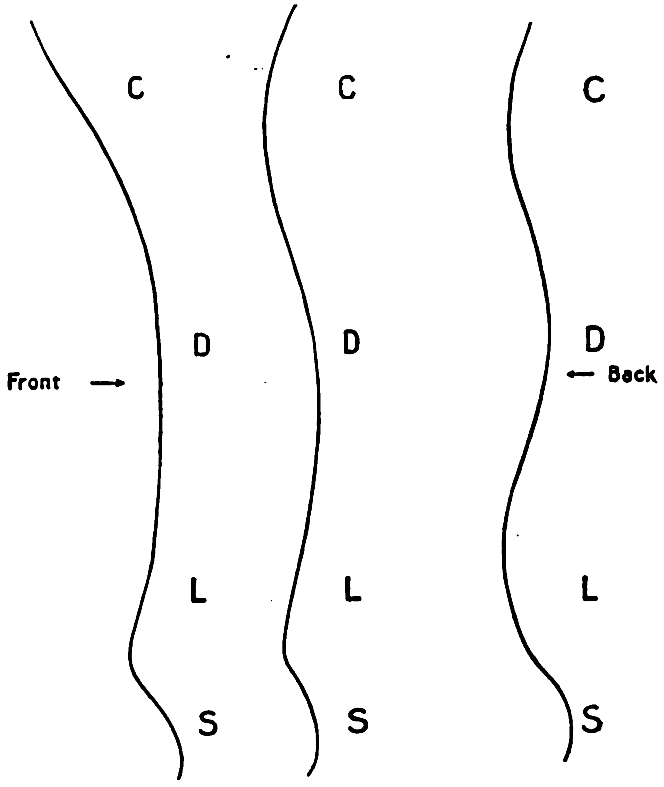


Dissection of the spine and pelvis in a newborn infant. Warren Museum, Harvard University.

ported, and (3) the additional *dorsal* and increased *lumbar* curves appear when the child stands and walks, and which correspond to th the adult condition.

DIAGRAM 2.

SPINAL CURVES.



Infant at birth. Infant sitting. Infant standing.

C represents cervical curve; D represents dorsal curve; L represents lumbar curve; S repre sacral curve.

A great deal has been written about the curves of the spine in new children, and their appearance in the embryo. Much of this literat a monument of wasted ingenuity. The truth is, that at birth, whe child is lying in what may be called its normal position,—that is to s its side, with the head flexed and the thighs drawn up,—the whole column presents one long concavity from the atlas to the coccyx, the of which is subdivided into two curves by the slight projection c promontory of the sacrum. Above this there is a tolerably regula cavity. The head can be thrown back so as to make a slight convex the neck, and by bringing the knees against the table (the infant bei its back) the lumbar region will spring forward; but the former of positions is rather unnatural, and the latter impossible without assis

The concavity of the thoracic curve remains to be discussed, and this is the only one of the curves above the sacrum that can be said to have any real existence at this age. When, however, we analyze more fully the existence of this curve, we begin to doubt whether it is after all so very real, for, though the sternum and ribs have some retaining influence, it is possible by bending the body backward to obliterate this curve also. We can then consider the part of the spine above the sacrum as essentially a fibrous and cartilaginous rod with a number of separate disks of bone embedded in it at different places. The extent of the movements possible at birth, both in the dissected spine and in the whole body, is very remarkable, as is shown by these few experiments. The first was on the body of a female child at birth large and well nourished. The abdominal viscera having been removed, it was very easy to bend the head back so as to touch the buttocks. The head and extremities were then removed, the ribs cut near the junction of the cartilages, and the spine and pelvis roughly cleaned. It was then possible, by some straining, to bend the spine backward so that the atlas and coccyx met. It was, however, easy to bend it backward so as to make an arch, the atlas and coccyx resting on the table. It was noticed that the middle part of the spine was the most flexible, the dorsal concavity of after-life being easily changed into a convexity. The lumbar region appeared to be more pliant than the cervical. The point of greatest motion was apparently between the eleventh and twelfth dorsal vertebræ. The whole spine, with each of the cervical, dorsal, and lumbar regions, bends forward with about the same readiness that it does backward. It may at first appear surprising that it does not bend very much more when, as already said, we look on flexion as the normal position of the infant; but it must be remembered that this effect is largely due to the great head which bends forward on the spine, and that the above statement as applied to the spine after the head has been removed is more remarkable than appears at first sight. Lateral motion is very free, though it is not quite unmixed with torsion. The atlas can without effort be brought to the level of the sacrum either to the left or to the right. The bending is pretty regular through the different regions. In torsion, the sacrum being fixed, the spine could be twisted so that the atlas looked backward, and could even, with some straining, be carried through more than half a circle. From rather crude measurements it appeared that, under the above conditions, the rotation in the cervical region was through an arc of 45°, in the dorsal region 90°, and in the lumbar region 45°. Experiments were then made on the intact body of a girl thirteen years old. The head could easily be made to touch the heels, and it could be bent so as to fit into the middle of the back. Forward flexion appeared little greater than that of the adult, which is to be accounted for by the space taken by the head. When the pelvis was fixed, the head could be rotated through about three-quarters of a circle. The spine, thorax, and pelvis were next made into a ligamentous preparation, and the spine could then be bent backward until the atlas was almost within an inch of the pelvis. (It is to be remembered that, unlike the last preparation, the sternum in this case still in place.)

Under these conditions the spine could be flexed so as to make the a touch the upper end of the sternum and the pelvis the lower. Lat motion was easy until it reached such a degree that the ribs on the fle side came in contact. When the pelvis was fixed, the spine could easil rotated through an arc of 90° without the action of the atlas.

Professor Dwight has pointed out the rather remarkable fact that all ages, from birth upward, the spine of the fourth lumbar vertebra is in the adult) on a level with the highest point of the crest of the ili Under certain circumstances this might advantageously be used as a starti point from which to count. At birth the spinal cord descends only the sp of about one vertebra lower than in the adult. The third lumbar sp which should mark its termination, cannot be easily recognized under tl years, but the correspondence of the top of the ilium with the fourth tebral spine allows its position to be estimated. It might be desirabl know how far the cavity of the spinal dura mater descends inside sacrum. Recent investigations by Dr. R. Wagner show that in child under a year old it ends usually near the top of the third sacral vertel which makes it a little lower than its usual termination in the adult. point on the surface corresponding to this could be approximately e mated without any definite landmarks.

NECK.—Now notice the large heads and short necks of these infa (Cases 3 and 4, facing page 26).

It is customary to say that young babies have no necks; and yet wl speaking of the spine I stated that the cervical region of the vertet column of the infant and young child is relatively longer than in the ad From this point of view the shortness of the infant's neck must be seem rather than real, but from a clinical stand-point it is real enough. causes of the short neck are first the large head, which naturally falls ward, covering the upper portion, and next the high position of the stern encroaching on it from below. The large proportion of subcutaneous tends to make the neck appear still shorter.

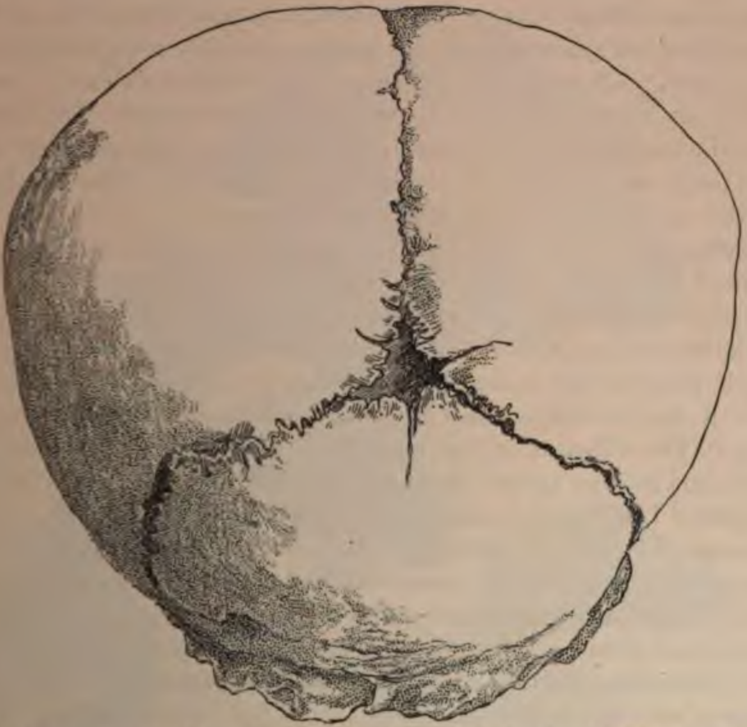
Symington, referring to the soft parts, says, "The peculiarity of t part of the child's neck is not that it is relatively short, but that it is higl in relation to the vertical column than in the adult." He has shown b series of observations that the larynx is at first placed much higher th later. In the adult the lower border of the cricoid is about on a level w the top of the seventh vertebra. In the infant it usually seems to be n the lower border of the fourth vertebra.

HEAD.—As a rule, if you take the measurements of the head over t middle of the forehead and around to the occipital protuberance, you w find that at birth the circumference is about 33 cm. (13 inches).

FONTANELLES.—The opening between the frontal bones and the ant rior borders of the parietal bones is called the *anterior fontanelle*, and, thou somewhat depressed below the level of the bones at first, should soon be about on a level with them. Its size is variable, but is usually about 1 to 2 cm. (½ to ⅞ inch) long, and about 1 cm. (½ inch) wide. In the early days and even weeks of infancy the frontal suture is usually open in its upper part. The anterior fontanelle at term is well represented in this skeleton of the infant at term. (Lecture IV., Fig. 33, page 118.)

The opening between the occipital bone and the posterior edges of the parietal bones is much smaller, is of less significance than the anterior opening, is often temporarily obliterated by the overlapping of the bones, and is called the *posterior fontanelle.* You see it here beautifully shown in the skull of a new-born infant.

FIG. 4.



Skull of infant at term, natural size. Posterior view, showing parietal and occipital bones and posterior fontanelle. Warren Museum, Harvard University.

FACE AND CRANIUM.—You will notice that the proportion of the face to the cranium in these infants (Cases 3 and 4, facing page 26) is strikingly different from what we meet in adults, where it is as one to two, while according to Froriep the face in the infant is to the skull as one to eight.

If we contrast the front view of the face and cranium of the infant and of the adult by counting as face all below a line at the tops of the orbital arches, and as skull all that is seen above that line, considering it projected on a vertical plane as in a photograph, we find that in the infant the skull forms about one-half, and in the adult much less.

It is found that the height of the orbit bears pretty nearly the sam portion to the skull at all ages, but that it equals barely a third of the face, while it makes nearly a half of it at birth. While the top of the opening retains pretty nearly the same relation to the orbit at all ag lower border is but very little below the lowest point of the orbit at while it is much below it in the adult. In the latter, a line connectin lowest points of the malar bones crosses the nasal cavity, or at least to its lower border, while in the infant it runs almost half-way betwee lower border and the edge of the alveolar process. The breadth of the in its greatest diameter in the infant equals, or even exceeds, the total h of the skull and the face, while in the adult it is but about three-quart it. Still more striking is the difference between the length and the br of the face at different stages. The breadth, measured between the mos tant points of the zygomata, is to the height of the face in the adult abo nine to eight, while at birth it is perhaps as much as ten to four.

The side view is equally or even more characteristic. The aud meatus is situated about midway between the front and the back i infant, but in the adult it is decidedly behind the middle. The face ap to be but an insignificant part of the whole structure.

Jaws.—The lower jaw is almost on the same plane as the ma process of the temporal bone, and the upper border of the zygoma is a on a level with the floor of the nasal cavity, while in the adult it is near the level of the floor of the orbit. It is evident that a very impo factor in the adult face is the development of the jaws and of the teeth, that it is due to their rudimentary condition that the face is so sma infancy. The difference in the comparative development of the lower at birth and at three years is well exemplified by these two skulls (Div II., Lecture III., Fig. 16, page 67).

Gums.—The gums do not meet in the new-born (McClellan). They composed of a dense fibrous tissue covered by vascular mucous memb of very slight sensibility, and are protective to the growing teeth.

All these points you will find of practical importance when you asked to determine whether the face and skull at various ages are nor or present some vice of formation.

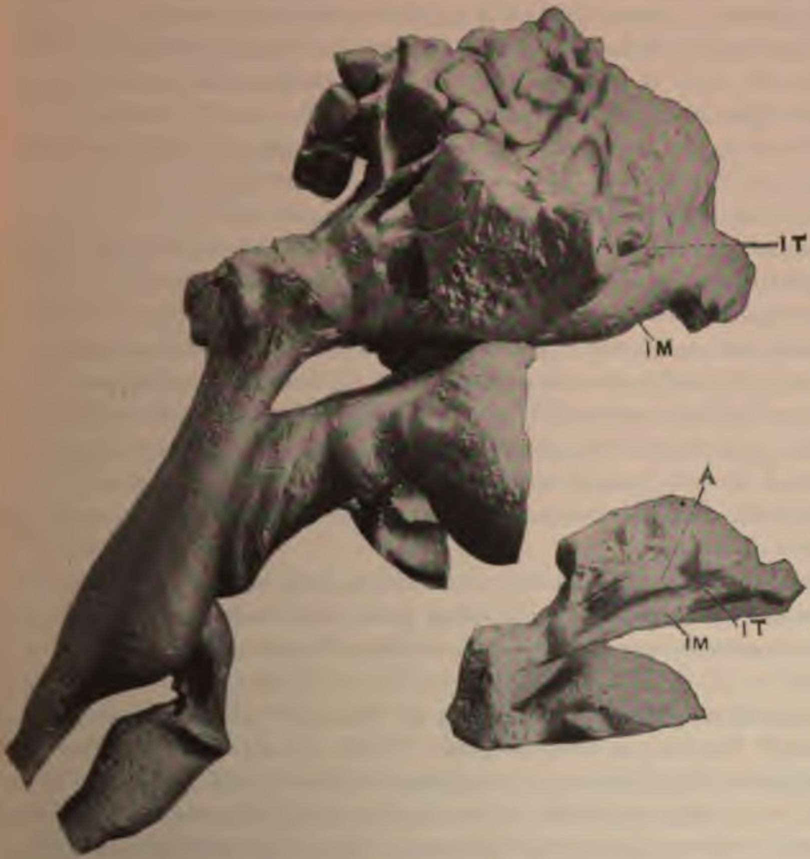
Young infants frequently have at birth quite a startling shape to t heads, produced by pressure. One side of the skull may be flattened w the other bulges, or the natural diameters of the head may be altered, senting a long narrow head instead of the round, well-formed crani These different shapes give at times an idiotic expression to the infant w causes much distress to the parents. In almost every case in my exp ence these abnormal appearances pass away as the skull and brain gr and do not, as a rule, indicate disease unless very extreme, so that it is v to state this fact to the parents at once and thus to relieve their minds.

NASO-PHARYNX.—A knowledge of the change in size and shape of nasal cavities and *naso-pharynx* in the course of growth is very importa

Valuable work has been done by Professor Disse on this subject. He divides the nasal cavity into the vestibule in front, the exit behind, and the intermediate portion, which consists of an upper olfactory region, occupying the ethmoidal portion of the cavity, and a lower respiratory region, occupying the maxillary part. In the infant the nasal cavity is relatively long and shallow, and the respiratory portion is very narrow.

These casts in fusible metal taken from the Warren Museum of the Harvard Medical School were made by Dr. S. J. Mixter, and, as you see, show a striking difference between the infant and the adult in the proportions of the inferior meatus.

FIG. 5.



I M marks inferior meatus; I T marks inferior turbinate bone; A marks antrum.
Warren Museum, Harvard University.

Observe that in the adult preparation the metal runs deep under the inferior turbinate bone in the form of a long cylinder, while in the infant, though the inferior turbinate projects slightly into the nasal cavity, there is but a very minute expansion below it, and none passing up behind it.

According to Professor Dwight, the height of the posterior nares at is 6 to 7 mm., and the breadth between the pterygoid processes at the palate is 9 mm.

In infancy, the posterior border of the vomer is very oblique. Sit just behind the nasal cavity is the upper or nasal portion of the pha which shares in its changes. I may perhaps be permitted to doubt wh many practitioners who have not had the advantage of modern anato teaching appreciate how small a cavity the naso-pharynx is even in the a Its height is twenty millimetres, and its antero-posterior diameter, fro hard palate back, is twenty to twenty-two millimetres. In the infant very much smaller. It is less of a vestibule and more of a narrow pa running obliquely backward and downward from the constricted op of the posterior nares. The soft palate of the child seems to be p more horizontally than in the adult, and bounds its anterior portion b Kostanecki gives the height at birth as ten millimetres, and its an posterior diameter as fourteen or fifteen millimetres.

I do not give you Professor Dwight's observations on this point, seems to me that its shape is so peculiar that measurements are deceptiv at least inadequate to give the proper idea. Imagine the posterior nares the inferior meatus alone, but the whole opening on either side) large en to admit the end of a medium-sized male catheter, and that this leads the passage just mentioned, and you can conceive how a congestion o nasal mucous membrane in infancy, with the addition of the mucous s tion, may effectually close the opening from the nose to the pharynx.

It is, perhaps, not sufficiently recognized clinically how importa function is performed by the nasal passages in early infancy,—far important, indeed, than at any other age. I can, in fact, say that the of the infant is in inverse ratio to the dangers which may arise from struction of the nares.

These dangers, consequent on obstruction, congestion, and the resul mechanical disturbance of neighboring parts, thus leading to actual dis of those parts, become in the new-born infant of most serious and even import.

In my own practice I have seen an infant die of simple acute n catarrh in the first two or three days of life. In this case the infant indeed, puny and ill cared for. Nothing abnormal could be detected in throat, or, in fact, anywhere, except in the nares, which were comple occluded by the congestion and tumefaction resulting from an acute infl mation of the nasal mucous membrane. Occurring, as the case did, in earlier years of my practice, I did not appreciate as I do now the extr importance of the naso-pharyngeal function of the young subject. Th fore, after prescribing the usual remedies for such cases, on the second of the infant's life, I was surprised to learn that it had died suddenly the third day.

This unfortunate experience, however, served to draw my attentio the proper treatment of this class of cases, and there is no doubt that, with due appreciation of the value of the nasal function and the danger of allowing it to be interfered with, we can, as a rule, even in extremely weak infants, prevent a fatal result.

I speak of this case in connection with the anatomical conditions of the naso-pharynx in order that you should appreciate the fact that these details, which are somewhat difficult to remember, are not merely of theoretical interest, but have a practical bearing on disease. I shall also refer to this case and its proper treatment in a later lecture when we are considering diseases of the throat and nose.

Lymph-Vessels of the Pharynx.—An anatomical condition of great importance, which I shall especially dwell on when I speak of pharyngeal diphtheria, is that in comparison with the faucial tonsils, which are relatively poor in absorbents, we find an exceedingly rich plexus of absorbents in the posterior wall of the naso-pharynx.

Eustachian Tubes.—The Eustachian tube in its clinical aspect is so closely associated with the naso-pharynx that it can best be spoken of in connection with it. In the fœtus the nasal opening is below the level of the hard palate, which it reaches at birth. While in the adult the cartilaginous portion slants downward, nevertheless the opening of the tube is opposite a higher part of the nose than in the child. At birth the tube is horizontal or nearly so.

Professor Dwight has shown me the opening of the Eustachian tube at birth just above the level of the hard palate, and, in a child a year or more old, a little below the line of the palate. This statement may perhaps be misleading. It must be borne in mind that even if the opening of the tube be below the level of the hard palate, the soft palate none the less runs beneath it, shutting it off from the cavity of the mouth and the passage from it to the fauces.

In the infant and the young child there is but a slight development of the end of the cartilage which makes in the adult so prominent a fold at the back of the pharyngeal opening of the tube, and by its prominence does much to determine the depth of the fossa of Rosenmüller, the recess behind it at the lateral posterior angles of the pharynx. At birth this prominence hardly exists. The opening of the tube is at first very small. That the catheterization of the tube at this age presents great difficulties of its own, apart from the intractability of the patient, is sufficiently obvious.

The tube in infancy, while of course shorter than in the adult, is stated to be not only relatively, but absolutely, wider at its narrowest point, which may explain the ease with which catarrhal processes travel at that age to the middle ear.

Faucial Tonsils—Pharyngeal Tonsil—Lymphoid Tissue.—The faucial tonsils, the pharyngeal tonsil, the lymphoid masses under the mucous membrane of the posterior third of the tongue, the lymphoid tissue about the orifices of the Eustachian tubes, to say nothing of irregular aggregations of the same tissue in the neighborhood, form a lymphoid ring the pharynx which is most important. It is to be noticed that the from the nose, as well as that from the mouth, is guarded by thi ratus. That its function is in part protective seems very probable, of the fact that when hypertrophied it gives rise to serious troubl fore birth this system is but slightly developed. Indeed, the foll the back of the tongue are not always to be found at that time. I reg Professor Dwight has not had material enough to aid me in addin to the little that is known as to the progressive development of the Killian states that the pharyngeal tonsil is at birth a raised bunch con adenoid tissue with ridges running in various directions, often more converging to a point, and rarely running directly forward and back

A pocket in the pharyngeal tonsil is the famous *bursa pharyngea*, clinically important merely as a recess in which inflammation may and secretions be retained.

As for the physiology of the tonsils, in which I include all the tissue of this region, I will mention that Stöhr showed that leucocyte their way from them through the mucous membrane to escape i throat. This process begins with life. He found the infiltration surface of the tonsil of a child of three months much greater than case of new-born infants.

The supposition that this system is protective receives suppor Killian's observation that the pharyngeal tonsil is much developed in mals that live in the dust of houses. Metschnikoff's theory, that leu devour bacteria, does not seem to be supported: nevertheless, it is possible that this lymphatic ring forms a bulwark against septic inva

Stöhr's observations of the escape of white corpuscles do not nece conflict with the view that the tonsils absorb the secretions of the in front. If these secretions are irritating, inflammation of the tonsil result. The effects of enlargement of the faucial tonsils are well k those of hypertrophy of the pharyngeal tonsil have been recognized within a few years. Indeed, I imagine that it has not been more th or twelve years since the general practitioner became aware of the exi of such a structure. The small size of the naso-pharynx in the infan the young child must not be forgotten, for it explains its obliteration enlargement of the pharyngeal tonsil.

MOUTH.—I wish you now to get a general idea of the mouths of young infants as I open them for your inspection. You see the wh comparatively dry tongue, which, with the lips, cheeks, and roof o mouth, immediately closes around the inserted finger and produces the sation of sucking. The mouth, then, as a whole, is pre-eminently an intended for the reception of a liquid food, its mechanism being that o tion. It is a natural and necessary passage-way to the organs of dige but is not at first, as I shall explain to you later, intended to aid the d tion by a salivary secretion.

Gums.—The gums have already been described on page 32.

Teeth.—At birth there are twenty embryo teeth, ten in each jaw, enveloped in their respective tooth-sacs, protected above by the submucous tissue and mucous membrane, on either side by alveolar bone-substance, and below by the groove in the maxillary bone from which the alveoli have developed.

I do not propose to undertake a general description of the cavity of the mouth, but shall merely call attention to some especial points in connection with the discussion of the relations of the pharynx. A median section of the infant's head shows very strikingly the want of height of the naso-pharynx and the great obliquity (approaching the horizontal) of the posterior edge of the vomer. The naso-pharynx is relatively very long from before backward. Strange as it may seem, the distance from the back of the hard palate to the soft parts of the back of the pharynx (excluding the tonsil) is about as great at birth as in the adult.

This statement appears incredible, but is easily proved by measurement. The tongue of the infant is greatly wanting in vertical thickness, and is shown on such a section to be long and low. The soft palate rests, therefore, on the tongue, and, the mouth being closed, runs in the main backward, descending very much less than in the adult. The uvula is rudimentary (Merkel). It seems to me that, owing to the depth of the pharynx (from before backward), the soft palate is unable to shut off the passage to the naso-pharynx as completely in early infancy as subsequently.

It is very curious that, in spite of these peculiarities, the distance from the tip of the uvula to the top of the epiglottis is relatively as slight in the infant as later.

Hard Palate.—I shall now call your attention to the level of the hard palate, and to what vertebræ are behind the mouth at different ages. This may be studied in connection with the position of the larynx already mentioned. We find by examining anatomical specimens that at birth and in the early months of life the line of the hard palate, continued backward, would strike near the top of the basi-occipital, that is, near its junction with the sphenoid, or perhaps even strike the latter. Accordingly, at this age, if the finger be introduced directly backward through the mouth, pushing the soft palate upward, it will strike the occipital bone, and, being carried a little downward, will pass over the arch of the atlas, the base of the odontoid, and the body proper of the axis. Going still lower, the top of the third cervical veretebra might be felt, but the larynx would hardly permit the finger to go lower, and the parts are so small that I doubt if much could be recognized below the axis.

Brain.—The brain of the new-born infant is proportionately very much larger than in the adult, bearing a relation of about 15 to 1. (Vierordt.)

Eye.—The eye is anatomically perfectly developed in the new-born. (McClellan.)

Ear.—The development of the ear, as stated by McClellan, is in its several parts very unequal. The structures of the internal ear, the tympanic cavity, and the auditory ossicles are fully formed at birth, while t ternal auditory meatus, the Eustachian tube, and the mastoid portion temporal bone undergo many modifications before their full developı puberty. At birth the meatus passes inward and inclines downwa the membrana tympani is almost horizontal, conditions to be rememb necessitating a little different management of the ear speculum fro you are taught in the examination of the adult ear. (Vide Lectur page 65.)

The mastoid antrum exists at birth, but the cells do not develo later.

PETRO-SQUAMOSAL SUTURE.—An important anatomical conditi isting at birth is, that the *petro-squamosal suture* is open, allowing connection between the blood-vessels of the brain and the middle ea its resulting clinical significance.

THORAX.—The thorax of the infant forms the upper and smal of the egg-shaped body which I have already described the trunk senting. As I have pointed out, the small shoulders of the infant m chest very different from that of the adult. Besides this, the whole of the thorax is very peculiar. The proportion of the dorsal region spinal column is pretty nearly the same throughout life, but the itself varies greatly. At birth the thorax is very insignificant. I the breast-bone is relatively much smaller than that of the adult ma not very different from some very small breast-bones which are occas met with in women. I shall consider this in detail later, and I now mention that the lower part is but slightly developed. The borders ribs diverge relatively rapidly. This is perhaps due to the great b of the abdomen.

TOP OF THE STERNUM.—The sides of the thorax are not relati long as in the adult, which is probably partly due to the lesser develo of the lower ribs and partly to the very important characteristic infant's thorax,—namely, that the top of the sternum is placed highe in the adult. The top of the sternum in the latter is about on a leve the disk between the second and third dorsal vertebræ. The top sternum, according to Symington, is opposite about the middle of th dorsal vertebra in the new-born infant, and a frozen section by Rü shows it to be rather below the middle of the first.

DIAMETERS.—Another most important peculiarity of the infantil child's thorax is its want of breadth. In the adult throughout the t from about the level of the second costal cartilage, or even a little h to the top of the diaphragm, the antero-posterior diameter of the in of the thorax is to the transverse as one to two and a half or one to there being, of course, a certain amount of variation. At birth, o other hand, it is as two to three.

It is well known that in the infant the ribs are more nearly horiz than in adult life. A striking feature of the young infant's chest is th ribs form the sides of the chest, and the sternum and cartilages the front. I will now give a more detailed description of the latter parts, which are of great importance for two reasons: first, on account of their influence on the type of respiration, and, secondly, because the costal cartilages are used as landmarks for the organs beneath them.

OSSIFICATION.—At birth the sternum is practically a strip of cartilage in which a varying number of bone-centres have been deposited. There is one for the manubrium and usually one or two for the second and third pieces, those for the latter being very frequently double. These, however, are mere thickenings of the cartilaginous strip, which is flexible and pliable in all directions. The divisions of the sternum in infancy are plainly seen in these skeletons, especially in the larger one, which is nineteen months old. (Lecture IV., Figs. 33 and 34, page 118.)

MOVEMENT OF RIBS.—A word as to the movements of the ribs will be of interest before we discuss the mechanism of respiration as a whole. The movements of the adult ribs are very imperfectly explained in many of the treatises on anatomy, and in others the explanation is labored and complicated. A ligamentous preparation of the spine, with a small piece of each rib *in situ*, shows the following state of affairs. The first rib moves up and down on a single axis running through the head of the rib resting against the body of the vertebra and its tubercle on the transverse process. This movement is a perfectly simple one, the front of the rib moving up and down, and no other movement is possible. In the second rib the conditions are practically the same; but in the third there appears a new feature, which is more developed farther down. It is that the tubercle of the rib no longer remains in place on the transverse process, but slides up and down on it, so that while the inner end of the axis remains stationary the outer end is raised (in respiration), and consequently we have, in addition to the raising of the forward end of the rib, a swinging upward of its outward convexity, which may be referred to a rotation on an imaginary antero-posterior axis. Skipping now to the last rib, which has no tubercle and rests on no transverse process, we find that we can raise or depress it, move it forward or backward, and circumduct it, by carrying it from one of these positions to another. This is true in a less degree of the eleventh rib, and perhaps to some extent of the tenth. The raising of the front of the ribs not only increases the antero-posterior diameter of the chest, but, by bringing the lateral convexity of each rib to a higher level, also increases the transverse diameter; this is further increased by the rotation of the longer ribs on an antero-posterior axis. The freedom of the lowest ribs allows them to be pulled backward and downward by the muscles of the back, thereby giving a firmer attachment to the diaphragm, and thus favoring its contraction, or they may be drawn inward by it or upward, following the outer ribs. It is to be remembered that in such a ligamentous preparation the movements are far more extensive than they can be in life, owing to the restraint exercised by the sternum and costal cartilages as well as by the soft parts. The influence of the sternum is especially important, as in adult the body is in one piece, and the amount of motion between it and manubrium is probably not often great.

RESPIRATION.—An important feature in the mechanism of thor respiration is the rigidity of the thorax. In the infant at birth this rigic is almost wholly absent, as it is found only in the ribs.

The sternum, as has already been said, is at this age practically a fectly flexible strip of cartilage, for the small points of ossification in it o modify the softness of certain separate parts. The dorsal region of spine is not fixed as a concavity, but can be bent freely backward. motions of the ribs are, as Professor Dwight has satisfied me from our servations on the dissected spine, practically the same as in the adult, the effect of these motions is different. In the first place, as has b shown, the ribs are more nearly horizontal, and the thorax, even after de is in what is called the inspiratory condition. The nearly horizontal f rib can hardly rise any higher unless the whole spine is bent backwa The ribs, being straighter than in the adult, do not when raised incre the breadth of the chest to the same degree. The nature of the infan respiratory movements is far from easy to analyze. Sometimes it see abdominal and sometimes thoracic. The fact is, that at first it is of a v indefinite type. The thorax seems to expand as it can. It is common see its lower part drawn inward by the contraction of the diaphragm.

An examination of the living subject during the different periods infancy has been made by me with considerable interest, and my resu coincide closely with what I had already been led to expect from my a tomical and physiological studies. At birth no especial part of the respi tory apparatus has attained a sufficient development to insure its continu equable action, and I have therefore found, as would be expected, irregu respiratory movements and no decided type of respiration.

A sufficient number of observations, however, have not yet been ma to warrant our stating any especial age at which the type of respiration the two sexes separates and the female infant assumes the thoracic type respiration. But if the breathing of the infant is essentially irregular type, it is admirably adapted to the wants of its age. The elastic thor can give way under pressure and expand in almost any direction. T flexible sternum submits to liberties which no adult breast-bone wou endure. One-half of the chest may be compressed and yet the other go acting independently.

The facts concerning the shape of the infant's thorax, which I ha already pointed out,—namely, that the top of the sternum is highe reckoning from the spine, that the ribs are more nearly horizontal, and th (probably) the lower part of the sternum is relatively less developed than the adult,—necessarily imply certain peculiarities in the relations of t internal parts. There is, however, a difficulty in understanding and statin these peculiarities, which, though sufficiently evident, is often overlooke and which may occasion both obscurity and confusion. This is the want of a generally accepted standard by which to judge of the position of these parts. Is this standard to be the spine or the front of the chest? We cannot use both indiscriminately, for their relations differ with the age. It is clear that the spine is the more fixed point of the two, and therefore the better scientifically; but for most clinical purposes it is desirable to refer to the front of the body.

DIAPHRAGM.—I shall now speak of the position of the diaphragm. This, as is well known, rises highest on the right over the summit of the liver, is a little lower on the left, and lower still at its tendinous centre in the median line. It is generally stated that the diaphragm is higher in the child than in the adult. Dwight's observations, partly original, partly on the frozen sections of other writers, give the following result. In the infant the diaphragm appears to be opposite the disk between the eighth and ninth dorsal vertebræ.

We now come to the insertion of the front of the diaphragm. In the infant it appears as if there were a lower insertion of the diaphragm to the sternum and the seventh costal cartilages than in the adult. Usually the line runs from one costal arch to the other, somewhat above the apex of the ensiform cartilage, leaving, therefore, a space on either side of the latter, where the interior of the thorax is against the abdominal walls. It is remarkable how vague and various are the statements in anatomies on this point in the adult. The sternal origin of the diaphragm is said in some instances to arise from the ensiform near its base, and in others near its apex. Undoubtedly there is ground for both assertions. In the two well-known median frozen sections of the body by Braune, it arises in the male at the apex of the ensiform, and in the female near its base. I hesitate, therefore, to assert that there is any difference in the points of attachment in the infant, but the effect is different none the less. Owing, perhaps, to the greater flexibility of the body and to the less firm attachment of the internal parts one to another, it certainly seems that at least after death the thoracic cavity is more accessible at the sides of the ensiform than it is in the adult.

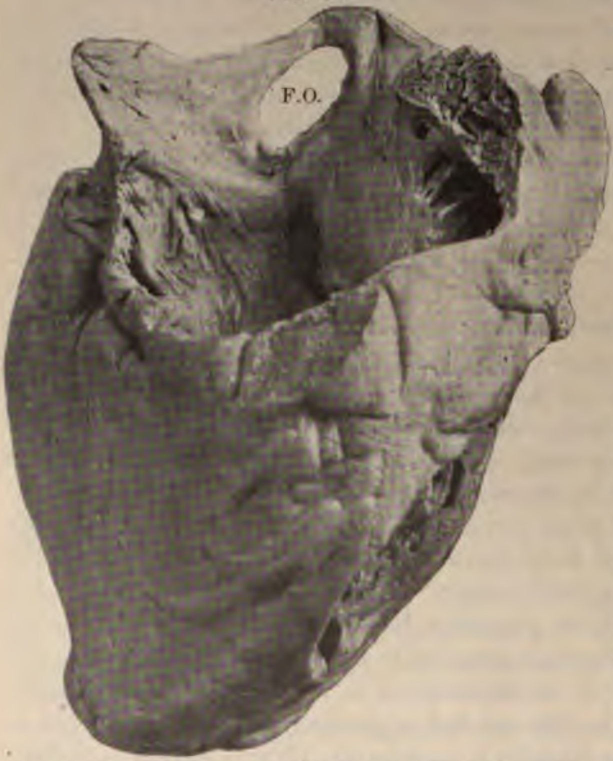
In the adult it may be as low as the middle of the tenth vertebra, but more often probably will be at the disk above it or the lower part of the ninth vertebra and occasionally higher. In Rüdinger's median section of a woman in the last months of pregnancy, it is as high as the lower border of the eighth. We may conclude that, while there is some variation, on the whole, the central point of the diaphragm is in the infant higher in relation to the spine than later in life, and that it gradually becomes lower. How high the diaphragm rises laterally is hard to say, for it is a point very difficult to observe. According to Kölliker, in the fœtus at term, on the right, it reaches the level of the anterior end of the fourth cartilage, and on the left that of the fourth intercostal space. Henke adds to this quotation that certainly after respiration has begun it will never be so high again.

There is another point concerning the attachment of the diaphragm to the front of the chest which will most conveniently be considered a l later: so, keeping this in reserve, I shall pass on to a consideration of thoracic organs.

THYMUS GLAND.—The thymus gland exists at birth, and lies above to some extent before the heart. It will be referred to later in the lec on Development. (Fig. 18, page 73.)

HEART.—The most striking peculiarity of the infant's heart is that less covered by the lungs than in adult life. Together with the thy gland it forms a solid mass from the posterior mediastinum to the stern pushing the lungs far apart. It is to be noticed, however, that the ple cavities extend as far forward as in the adult. The relations of the h to the chest-walls are curious in the infant, for these anterior walls are already stated, high in relation to the spine, yet the heart itself is high relation to the walls. At least the upper half of it is so. With regar the apex and the lower borders the relations are less certain. We usu find the impulse the heart rather hig and nearer to the m mary line in the inf than in the adult. weight of the heart birth is 20.6 gram (about $\frac{2}{3}$ ounce), cording to Boyd, its proportion to rest of the body largest at about time of birth.

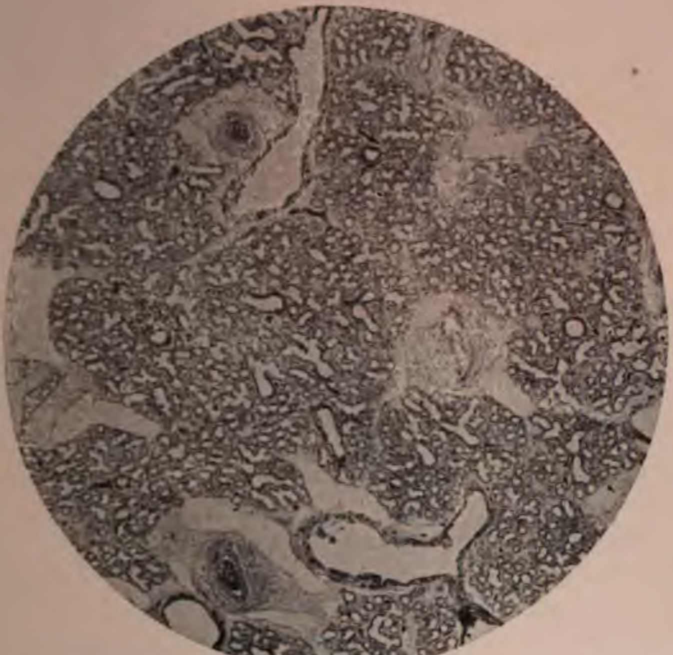
FIG. 6.



Right auricle and ventricle. Infant's heart. Open foramen ovale, marked F.O. Warren Museum, Harvard University.

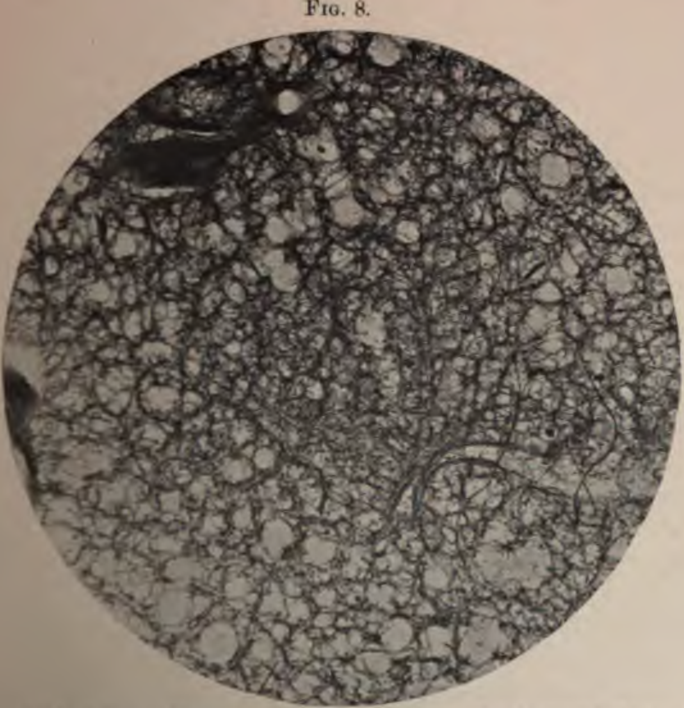
It will be well you in this conn tion to examine aga carefully this hea of the new-born i fant which I ha already shown yo (Lecture I., Fig. page 21.) As t foramen ovale is often open at birth, should also like you familiarize yourselv with what a pate foramen ovale looks like, as seen in this specimen (Fig. 6) of an old infant's heart, where you see there is a free and permanent connectio between the right and left auricles. The heart is slightly hypertrophied.

FIG. 7.



Section of fœtal lung at 5 months, showing development of bronchi; no alveoli.

FIG. 8.



Section of infant's lung at 10 months, showing increased proportionate amount of parenchyma in comparison with the fœtal condition; distended alveoli.

COMMON CAROTID ARTERY.—The common carotid artery has in the new-born half the length of the descending aorta, but this proportion is much lessened at a more advanced age, when the vertebral column increases in length.

VEINS.—According to Jacobi, there are one hundred valves in the veins of the lower extremities of the new-born.

PULMONARY ARTERY.—The pulmonary artery also, as stated by Jacobi, is from two to four centimetres (three-fourths to one and five-eighths inches) larger than the descending aorta.

LUNG.—I have already referred to the fact that the liver encroaches so much upon the space which on the right side of the thorax is occupied later by the lung that an important difference is found between the percussion of the right and the left lung. On the right side the eleventh rib behind marks the lower border of the lung, while it descends as low as the twelfth rib on the left side. In front the lung extends to about the fourth or fifth rib on the right side and the sixth rib on the left side. The lung at birth is characterized by its embryonic type. The infant's lung represents an intermediate condition of growth, which illustrates the gradual change from the fœtal to the adult condition. These photo-micrographs (Figs. 7, 8), made by Northrup, of sections of a fœtal lung at five months and of an infant's lung at ten months, explain fairly well the anatomical conditions of the lung at birth.

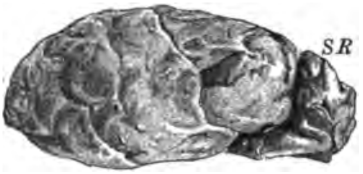
These conditions have been carefully studied by Northrup, who deserves great credit for the work which he has done on this subject, and which will be referred to later in the lecture on Development, and also in that on the Lungs. This author in speaking of the characteristics of the lung in infantile life says that if we examine the lung of a five months' fœtus it is found that the bronchi constitute the entire respiratory tract thus far developed. At the terminal end of the bronchi there are bud-like dilatations, which are the rudimentary air-spaces. Between these dilatations, and separating them from each other, is loose, delicate connective tissue, which makes up the remaining bulk of the lung, so that what subsequently becomes the alveoli is about equal in extent to the previous bronchial development. This rudimentary air-space is destined to enlarge, subdivide, and finally, in early adult life, to occupy all the available room among the bronchial branches. The loose connective tissue becomes finally thin, dense bands constituting the stroma. This serves to distribute the vascular net-work, and upon this are laid the close-fitting epithelial linings of the air-spaces. In fœtal life the mucous membrane lining the bronchial tubes is loosely attached to the muscular walls, and is commonly seen lying in wavy folds within the contractile ring, where the same delicate connective tissue loosely holds the growing tissues together. As has been said, the aerating portions of the lungs develop as bud-like dilatations at the tips of the smallest bronchi. These dilatations in the course of their development extend into the stroma. The epithelium, changing from the columnar type characteristic of the smaller bronchi, covers the newly-made walls with flat respiratory epithelium. At birth the loose connective-tissue stroma of the fœtal lung of five months has been condensed into rather thick alveolar walls. Another feature of tl child's lungs as contrasted with those of adults is the behavior of the bloo vessels. Being loosely restrained in the walls, they easily become distend and tortuous and encroach upon the cavity of the alveoli. With sma alveoli, thick walls, and abundant distribution of vessels, it is easy to unde stand how, in hypostasis, distention of the vessels may be an important fact in displacing the air in feeble subjects with weakened respiratory vigor a partially obstructed bronchi. Finally, the lung of the infant differs fro that of the adult mainly in the following respects. Proportionately tl extent of the bronchial tubes is greater than that of the air-spaces. Tl connective-tissue stroma is likewise in greater abundance and tends to cell lar proliferation. The submucous connective tissue of the bronchi is loo and more abundantly supplied with nuclei, and its vessels are held mo loosely. The cells lining the air-spaces form a continuous layer. Tl alveoli are small, their epithelium proliferates abundantly, and the absorl ents accomplish their work slowly, the blood-vessels playing a more in portant rôle. These facts are to be borne in mind in connection with tl bronchial lesion which forms so important a part of broncho-pneumonia.

ABDOMEN.—The essential differences between the abdomen of th infant and that of the adult are, *first*, the great size of the liver in the forme

LIVER.—This organ, especially on the right side of the abdomen, encroaches on the space which is later occupied by other organs. Its relativ weight to that of the whole body at birth is about 1 to 18. (McClellan.)

KIDNEY.—*Second*, but of less importance, is the relatively large size o the kidney and the supra-renal capsules. On the left side of the abdome these conditions are not of much importance, but on the right, occurring a they do in connection with the great size of the liver, the large kidney occupies a lower position, and thus still further curtails the free space in th right flank. Viewed from the stand-point of the adult condition the relations are, as has been pointed out by Henke much more peculiar on the right than o the left. The kidney as a whole is lobulated, as you see in this specimen take from an infant three days old. (Fig. 9.)

FIG. 9.



Lobulated kidney, natural size. Infant three days old. *SR* marks the supra-renal capsule. Warren Museum, Harvard University.

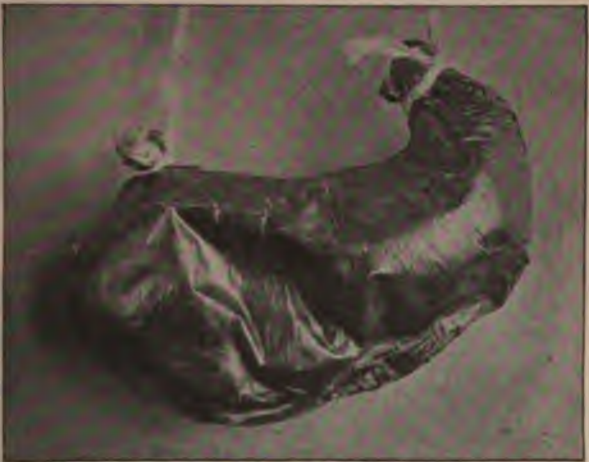
Uric Acid Infarction.—At birth a prenatal condition, represented by an orange or a light-red colored deposit nea the pyramids in the straight tubules of the kidney, exists normally. This condition is called the *uric acid infarction*, and the deposit consists of urate of ammonium, amorphous urates mixed with uric acid crystals, and some epithelial cells. (Plate III., 5, facing page 112.)

Supra-Renal Capsules.—The supra-renal capsules at birth quite cover and surmount the kidneys, as you will notice in this same lobulated kidney. (Fig. 9; the supra-renal capsule is indicated by *S R*.)

STOMACH.—Although it has long been known that in the adult stomach the greater part of the lesser curvature is vertical, and the long axis of the organ more nearly vertical than transverse, yet these facts have been slow in getting into the text-books and winning general recognition. It is probable that it has so long been taught that the stomach is placed transversely because when the abdomen is opened a triangular piece of the stomach comes into view, bounded on the left by the costal cartilages, on the right by the edge of the liver, and below by a part of its own greater curvature, which runs in a gentle curve from left to right. If this alone is seen it is very natural to assume that the stomach is placed transversely. The stomach at birth is remarkably small, and more tubular than in the adult, the fundus being but slightly developed. It is consequently even more vertical than in the adult, for it is the enlargement of the greater *cul-de-sac* that makes the obliquity of the axis pronounced.

This stomach (Fig. 10), taken from an infant three hours old, represents very well the organ at birth. Its capacity is 25 c.c. ($\frac{5}{6}$ ounce). The weight of the infant was 2500 grammes ($5\frac{1}{2}$ pounds). Although the weight was below that of the average infant at birth, the stomach was of about the average size, as was shown by its gastric capacity.

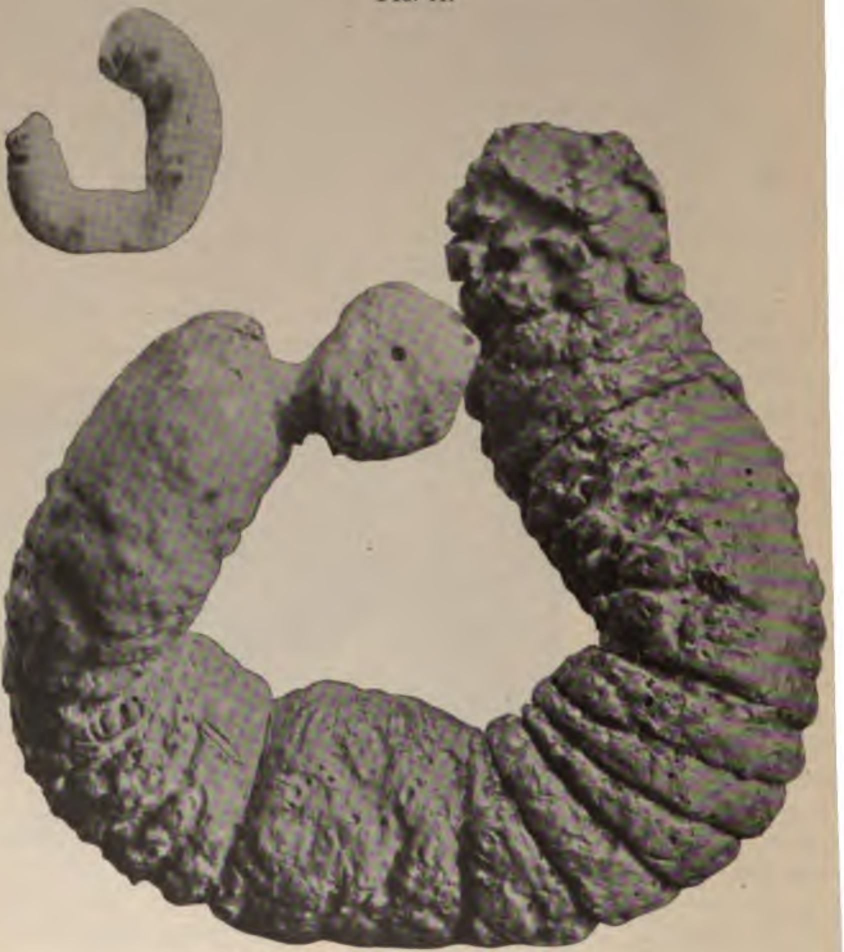
FIG. 10.



Stomach, natural size. Infant three hours old.
Warren Museum, Harvard University.

DUODENUM.—The duodenum, in the adult, has of late usually been described as ring-shaped, but it generally presents pretty well marked angles, which divide it into a horizontal part running backward, a descending one along the right side of the spine, a transverse one crossing usually the third lumbar vertebra, and, finally, an ascending part along the left of the spinal column, which brings the end to about the same level as the beginning. Sometimes the last two parts are represented by a single one running obliquely upward to the left, in which case the duodenum is called V-shaped. The first horizontal portion is often somewhat dilated, and its walls are smooth, the valves beginning usually with the descending portion. The walls of the duodenum just beyond the pylorus are lined by a continuous layer of Brunner's glands, which extends through the first part, becoming more or less broken up towards the end. In the infant the shape of the duodenum, as shown by plaster casts, is more nearly that of a ring, the two lower angles be rounded off. A constriction is often (perhaps usually) seen at the junct of the first and second parts, but Dwight's casts of the infant's duoden do not show the folds, which are very striking in the casts taken fr adults. That is to say, those of the infant show a few deep cuts i the cast instead of a great many near together. I have seen the fol

Fig. 11.



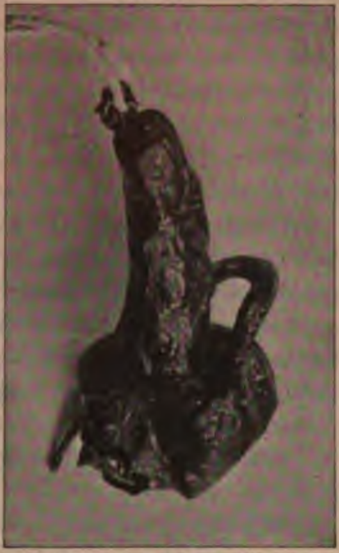
Casts of duodenum taken from infant and adult, natural size.
Warren Museum, Harvard University.

however, very richly developed in an infant of three weeks. In on case, that of a female six weeks old, Dwight found the duodenum of th V-shaped pattern, and, what is more remarkable, after it had passed th gall-bladder it was surrounded by peritoneum so as to swing freely as a loo suspended from the posterior abdominal wall. As to Brunner's glands, a few observations on young children have suggested that they were rather less developed relatively than in the adult, but I am by no means sure that this is always the case. The duodenum has been compared to a trap, its ends being always higher than its middle, which is thus fitted to retain the fluid poured into it from the liver, the pancreas, and its own glands, besides that which it receives from the stomach.

The different points concerning the duodenum which I have just described are well shown in these casts taken from the adult and from the infant (Fig. 11), and must be borne in mind when we are considering the digestive functions of this important part of the intestinal tract.

The number and size of the folds and the shape of the duodenum in the adult would tend to delay the passage of its contents through it, and thus it also prevents the passage of gases from the small intestine upward into the stomach. If it be true, as I am inclined to think it is, that in the infant the system of folds is less developed, its passage would be relatively easy, which with a fluid diet seems desirable.

FIG. 12.



Normal cæcum and appendix, natural size. Infant five days old. Warren Museum, Harvard University.

CÆCUM.—I should now like you to examine this specimen taken from an infant five days old. It represents the cæcum and appendix, and will aid you in understanding an important disease which we shall consider later,—*appendicitis.*

The cæcum is an interesting portion of the intestine at any age, and especially in the child. As is well known, the cæcum descends in the course of development from under the liver in the middle of the abdomen to the right iliac fossa, apparently passing first to the right and then descending; thus leaving behind it in its course the right half of the transverse colon and the whole of the ascending colon. It is needless to say that if it is possible for the cæcum to accomplish this journey it cannot be tightly bound by the peritoneum. On the contrary, the cæcum has a complete peritoneal coat and is perfectly free. At birth, and very possibly for a year or two afterwards, the cæcum has not, as a rule, reached its permanent position in the right iliac fossa. I have found it to measure three inches in length in an infant eleven weeks old.

INTESTINES.—From what we know of the development of the intestinal tract, which was at first merely a loop loosely attached to the posterior abdominal wall, it is natural to expect that in the infant and young child it should be less fixed than in adult life; and this is in fact the case. The difference is most striking in the large intestine, and is shown particularly in the cæcum, ascending colon, and sigmoid flexure. That this condition gives rise to dangers is evident, and I should say that there is a strong probability that the cases of infantile intussusception which occur unusual frequency during the middle of the first year may arise this anatomical peculiarity, and this makes a thorough knowledge anatomy of the cæcum important. The growth of the different pa the intestine has been studied by Treves. He points out that in not only does the length of the intestine vary greatly, but also there constant relation between the small and large intestines. A long sm testine may be followed by a short large intestine, and *vice versa*, or both may exceed or fall short of the average. In the fœtus at full ter length of the intestine, and especially of the colon, is singularly const

Small Intestine.—The average measurement of the small intest 287 cm. (9 feet 5 inches). The greatest variation that I have met amounted to 61 cm. (about 2 feet).

Large Intestine.—The large intestine at birth, according to T measures 56 cm. (about 1 foot 10 inches). So regular are these mea ments that the greatest variation that I have met with in the colon w little as 12.7 cm. (about 5 inches).

Sigmoid Flexure.—But little of the sigmoid flexure is found in the at birth.

PELVIS.—The small size of the infant's pelvis is to be noted al the cause which, to a greater or less extent, forces the pelvic organs of life into the abdomen during infancy. This condition is quite evide this spine (Fig. 3, page 27) which I have already shown you.

BLADDER.—In the infant the bladder is practically wholly an dominal organ. (This fact is well illustrated in Division II., Lecture Case 18, page 78.)

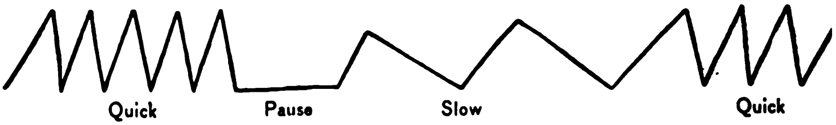
UTERUS.—At birth, part of the uterus is above the brim of the pe

TEMPERATURE.—The temperature at birth is slightly higher in the adult. It is about 37.2° C. (99° F.).

PULSE.—The pulse varies from 120 to 140 to the minute at birth, it is at times irregular, especially during the first few hours.

RESPIRATION.—The respiration is about 45 to the minute, but of a very irregular type, and if you will closely watch the rise and fal the thoracic walls in this infant (Case 3, page 26) you will see that rhythm changes continually. The breathing is superficial, sometimes qu and again dying away so as to be almost imperceptible. This condit if occurring in an older child, would be a symptom of grave disease, may be said to be normal at birth. The rate may be much quicker t 45, and I have frequently observed it as high as 60 or 70.

CHART 1.



Respiration at birth for one-fourth minute. Awake, but quiet.

HEIGHT.—The new-born infant's average height is in the male about 49.5 cm. (19¾ inches); in the female 48.5 cm. (19¼ inches).

WEIGHT.—The weight of the male infant is usually rather greater than that of the female. The average weight in a large number of cases showed that of the male to be 3250 grammes (7⅐ pounds), while that of the female is 3150 grammes (7 pounds). Parker, in a careful examination of 170 infants at birth, of whom 89 were males and 81 females, found that the average weight of the males was 3520 grammes (7¾ pounds), while that of the females was 3290 grammes (7¼ pounds). There is, then, as I have said when speaking of Case 2, a certain amount of latitude to be accepted in this question of weights. The weight, however, has so close a connection with the vitality of the infant, that although we often see infants of light weight vigorous and thriving, and those of considerable weight failing to gain, yet as a general index of vitality the weight is a valuable starting-point and guide for our treatment. I would impress upon you that all rules and averages of this kind are not to be depended upon absolutely, but simply represent conditions which with other important factors aid us in solving the problem of vitality.

VITALITY.—In the early hours and days of existence it is the disturbance of the equilibrium of the infant's vitality which is especially to be feared and combated rather than the specific morbid processes of later childhood. We should therefore in each infant carefully determine the degree of inanition which we are called upon to deal with at this period of life, and I have personally found it useful to divide the weak and strong infants into groups according to their weights, allowing, as I have already explained to you, a somewhat lighter weight for girls than for boys.

This table (Table 2) will explain to you the meaning of what I have just said:

TABLE 2.

Relation of Weight to Vitality.

Groups.	Weight.	Vitality.
1	2000 grammes (about 4½ pounds)	Very low.
2	2500 " " 5½ "	Low.
3	3000 " " 6½ "	Fair.
4	3500 " " 7½ "	Normal.
5	4000 " " 8 "	High.
6	4500 " " 9 "	Very high.

HANDS.—At birth it is quite remarkable to find with what manifest strength the infant can grasp your finger. The nails are well formed.

FEET.—An important part of the infant's anatomy is the foot, and I take great pleasure in introducing for your study some original work which has been done by Dr. John Dane.

Here is an infant (Case 5) four days old. Dr. Dane has taken an impression of its feet, which shows very beautifully certain points about the instep at birth which are entirely different from, and in fact controvert, what has heretofore been taught on this subject. The practical importance of this truly scientific and laborious work I shall refer to in a later lecture.

LANE LIBRARY

Where flat foot really exists, the internal border of the impression shows an undulating appearance, and there is evidence of equal pressure over the whole of the tracing, as seen in the tracing taken from the foot of this infant also four days old. (Case 6.)

It is interesting and instructive to compare the different appearances which are presented in Fig. 13, showing the well-developed arch, and those in Fig. 14, representing the true flat foot.

You should also examine carefully these babies' feet which have produced these appearances.

BONE MARROW.—At birth, and in the early months of life, the marrow of the bones is red, as you see in a section of this bone taken from an infant seven months old. (Plate II., facing page 107.)

You will notice that the red color caused by the numerous injected blood-vessels is more intense at the central portion of the section of this bone than at the periphery or towards the ends. I merely show it to you as a normal and characteristic condition of early life, and one which may appear again at a later period in certain diseased conditions.

FUNCTIONS.—It is important for you to have a general idea of which of the functions are absent, partially developed, or developed at birth. The endeavor to call into use an undeveloped function, to tax a partly-developed function, or to overtax a developed one, is productive of great harm, and it has in my experience been the source of many conditions which, looked upon as diseases, are in reality but proofs that our anatomical and physiological knowledge has been deficient.

VOICE.—The normal infant at birth should present a developed voice, and should cry vigorously, thus assisting the lungs to expand and the new circulatory mechanism to be well started.

SIGHT.—Although the eye is, as I have already stated, anatomically developed and is sensitive to light, and although the visual perception is also possibly developed, yet there is still a lack of power to interpret the images perceived.

HEARING.—The auditory sensations appear to be rather dull during the first few days of life. This is possibly due to the absence of air from the tympanum and a tumid condition of the tympanic mucous membrane.

TOUCH.—The sense of touch is well developed.

TASTE.—The sense of taste is well developed.

SMELL.—The sense of smell is probably well developed; but this is still a matter of dispute.

SEBACEOUS GLANDS.—The function of the sebaceous glands is fully developed at birth, as I have already described to you (page 23, Case 1).

LACHRYMAL GLANDS.—The secretion of the lachrymal glands is not developed at birth. The new-born infant does not shed tears, a fact of some clinical consequence in connection with the prognosis as to the convalescence of disease in the early days of life.

SWEAT-GLANDS.—The function of the sweat-glands is not developed at birth as a rule, but according to my observations perspiration in cert dividuals certainly occurs at a much earlier period than is usually su I have seen an infant, premature at the seventh month, perspire fre week after it was born, and in a number of individuals this functio exist in the early days of life.

SALIVARY GLANDS.—The salivary secretion, as has so clearl pointed out by Forchheimer, is not fully established at birth, and quently the mucous membrane of the mouth is comparatively dry, you see, these infants' tongues (Cases 3 and 4) have a peculiar whitish This appearance is caused by the epithelium not being washed away saliva to the extent that it is after the later development of the funct the salivary glands. The amylolytic function of the saliva is very s present at birth, as has been shown by Zweifel and Korownin, who mented with infusions of the salivary glands taken from young i The amylolytic action is indeed so insignificant that it merely shows the function of the salivary glands in the early months of existence i partially developed and certainly should not be called into use.

PANCREAS.—The amylolytic action of the pancreatic secretion at is probably not all developed. The fat digestion is fairly developed at The albuminoid digestion is fairly developed, but not fully.

BILE.—According to Foster, "the excretory functions of the liv developed early, and at about the third month of intra-uterine life bil ment and bile-salts find their way into the intestine. A quantity o secreted during intra-uterine life accumulates in the intestine, especia the rectum, and forms, together with the smaller secretion of the rest canal and some desquamated epithelium, the meconium. The distinc mation of bile is an indication that the products of fœtal metabolism longer wholly carried off by the maternal circulation, and that to the tory function of the liver are now added those of the skin and kidney.

BLOOD.—It is impossible by the methods at present known to dete exactly the total amount of blood in either infant or adult, but, whil adult's blood is approximately about one-thirteenth of the entire weig the body, the infant's is represented by only one-fifteenth. The blo rather more dense than in the adult, and contains a large amount of h globin. It is not rich in fibrin, and does not coagulate perfectly, a fact remembered when we are considering the hemorrhagic disease and h philia of the new-born. Soon after birth some of the globules are found to have nuclei, but these soon disappear.

RED CORPUSCLES.—The proportion of the red globules at birth is 5,900,000 to the cubic centimetre.

WHITE CORPUSCLES.—The number of white corpuscles is about times as numerous as in the adult's blood, and about 21,000 to the centimetre.

LYMPHATIC SYSTEM.—The lymphatic system is very activ birth.

URINE.—The amount of urine secreted during the first two days of life is very small, and its specific gravity is about 1010. The kidney shows the condition of the uric acid infarction, and it is not infrequent to find the napkins stained with a uric acid deposit, such as you see represented on this napkin (Plate III., 1, facing page 112).

INTESTINAL DISCHARGES.—Unless a discharge of the contents of the intestine has taken place during the delivery, as is so often seen in breech presentations, it occurs immediately or very soon after birth, as you have already seen in the first case which I presented to your inspection this morning.

MECONIUM.—This discharge which first comes from the intestine is called the *meconium.* It is inodorous, viscid, slightly acid, and of a brownish-black color, such as you see on this napkin taken from an infant a few hours old (Plate III., 2, facing page 112). The meconium contains mucus, epithelium from the intestinal mucous membrane, epidermal cells, hairs, and fat-drops from the vernix caseosa which have been swallowed with the amniotic fluid from time to time. It also, according to Vierordt, contains the constituents of the bile, and its total amount is from sixty to ninety grammes (two to three ounces), of which the solid part forms about twenty per cent. The intestinal contents at birth are sterile.

DIVISION II.

NORMAL DEVELOPMENT.

LECTURE III.

SPINE.—NECK.—HEAD.—THORAX.

WE have considered in a general and practical way the conditions exist in the infant at term. The data which we have acquired in thi sideration constitute only a part of the alphabet which we are endea to master.

In order to differentiate normal from abnormal conditions in the gr infant and child, we must now examine the different stages of develo which correspond to the various ages, and thus complete our anatomic physiological alphabet.

You remember the condition of the cord in Case 2 (Frontispiece), I showed you at the previous lecture. You see that in this infant (Ca nine days old, the has fallen off. Th curred twenty-four ago.

CASE 7.

Infant nine days old. Natural condition of umbilicus after recent separation of cord.

By a process o integration the co about the sevent eighth day separates the living tissues ar the umbilicus. A tain amount of ble may take place at point of separation, this is usually very slight: it may, however, be the beginning of one o most serious forms of disease in the new-born, *umbilical hemorrhage.*

You will notice how the umbilical depression is well marked even the infant cries, and you will thus distinguish this normal anatomical dition following the separation of the cord, from the umbilical promin which I shall show you later as representing cases of umbilical hernia.

SPINE.—The time of consolidation of the bodies of the vertebræ is not accurately known, but it may be roughly stated to begin in the third year, and, probably, to end in the seventh. A large number of observations must still be made before the march of ossification can be determined. The statements regarding this point are copied from one book to another, and are often quite imaginary.

The union of these chief centres to form the bodies of the vertebræ begins in the lumbar region, and is first completed there. This union, however, had not taken place in the dorsal and cervical region of the child said to be three years old, used for "The Frozen Sections of a Child" (Dwight). On the other hand, in a girl of five or six years, figured by Symington, the process was found to be hardly finished in the lumbar region, and higher up it seemed about the same as in the younger child.

The process of union of the laminæ is probably completed in the first few months of life.

LENGTH.—Aeby gives the following table for the adult spine, showing both the absolute and the relative length of the cervical, dorsal, and lumbar regions (the measurements are in millimetres):

TABLE 3.

Absolute.

	Cervical.		Dorsal.		Lumbar.
Female	122.9	+	265.8	+	190.3 = 579
Male	129.9	+	273.4	+	184.1 = 587.4

Relative.

	Cervical.	Dorsal.	Lumbar.
Female	21.12	45.7	32.8
Male	22.1	46.6	31.3

Cunningham obtained strikingly similar proportions in an average of the measurements of six males and five females:

TABLE 4.

Relative.

	Cervical.	Dorsal.	Lumbar.
Female	21.6	45.8	32.8
Male	21.8	46.5	31.7

Aeby gives the following table of the average of five infants, and Cunningham a table of three:

TABLE 5.

Relative.

	Cervical.	Dorsal.	Lumbar.
Aeby	25.6	47.5	26.8
Cunningham	25.1	48.5	26.4

The following table shows the results of the measurements of the spines of children by various authorities, as well as by Professor Dwight. The table requires no elucidation, but I shall call attention to the remarkable uniformity of observations by different men in spite of the errors incident to the personal equation of the measurements and the individual variat which doubtless exists. The relative length of the dorsal (more prop the thoracic) region throughout the table is somewhat greater than that the adult; still it appears that after the age of five or six the proporti are not far from those of after-life.

TABLE 6.

Length of Spine to Sacrum.

Absolute Length, in Millimetres.						Relative Length. Total = 100.		
Age.	Observer.	Cervical.	Dorsal.	Lumbar.	Total.	Cervical.	Dorsal.	Lum
8 months . .	Rasenel . .	50	100	58	208	24	48.1	27
6 months . .	Aeby . . .	52.5	103	60	215.5	24.8	47.5	27
6 months . .	Aeby . . .	53.5	107	61	221.5	24.1	48.6	27
10 months . .	Dwight . .	61	125	77	263	23.2	47.5	29
2 years, boy .	Rasenel . .	70	140	90	300	23.3	46.7	30
2 years, boy .	Aeby . . .	79.5	153.5	98	331	24	46.4	29
3 years, girl .	Dwight . .	78	162	101	341	22.9	47.5	29
4 years, girl .	Aeby . .	79.9	162	103.3	345.2	23.1	46.9	29
5 years, boy .	Symington.	80	170	104	354	22.5	48	29
5 years, boy .	Rasenel . .	80	180	135	395	20.3	45.6	34
6 years, boy .	Symington.	80	175	106	361	22.2	48.5	29
9 years, girl .	Rasenel . .	85	195	150	430	19.8	45.4	34
11 years, boy .	Aeby . . .	91	218.7	153.5	463.2	19.7	47.2	33
13 years, girl .	Symington.	95	220	136	451	21.5	48.7	29
16 years, girl .	Aeby . . .	100	221.9	151	472.8	21.1	46.9	31
16 years, girl .	Aeby . . .	107.5	229.5	152.5	489.5	21.9	46.9	31
17 years, girl .	Dwight . .	113	250	161	524	21.5	47.7	30

The figures to the left of the double line in the table express the al lute length of the different portions of the spine, in millimetres.

Those to the right are the same figures reduced to terms of 100, wit a fraction.

FLEXIBILITY.—I have already shown you how very flexible the s is at birth. This flexibility becomes less as the infant grows older.

In the cadaver of a female child of ten months it was found that ext sion was no longer so free as in the earlier months, and it required a str pull to make the head touch the nates. The dorsal region, however, co still be made concave behind. Flexion was free, especially in the lower of the lumbar region, where the pelvis and legs could be swung forwa On rotation the head could be turned through an arc of 90° without us the joint between the atlas and the axis. In a male child of the same a extension of the spine was found to be still more restricted.

CURVES.—In the last lecture I explained to you that at birth there w no natural curves in the infant's spine.

An important factor in the production of the curves in the cervi and dorsal regions is probably the pull of the muscles, as will be presen described. The dorsal curve seems to be a permanent condition of a p of the general curve of the body. As soon as the muscles of the back the neck contract so as to raise the head from the chest, the front of the neck will be convex, and finally this becomes the habitual position. As Symington has pointed out, however, this cervical curve is never, properly speaking, consolidated, for it can always be obliterated by a change of the position of the head. The production of the lumbar curve is more complicated. If an infant be laid on its back on a table, the knees are raised and fall apart; if they are brought together and forcibly pressed down, the lumbar region will spring up from the table and the beginning of a lumbar curve will appear. It is supposed that this is caused by the shortness of the ilio-femoral ligaments, which, when the thighs are brought down, flex the pelvis, throwing the promontory of the sacrum forward. As the child begins to stand, the body is inclined forward, and when this is straightened by the muscles of the back the same thing occurs, for of course it is unimportant whether the legs are extended on the trunk or the trunk on the legs. The credit of this explanation has generally been given to Ballandin, but it appears to belong to Cleland.

This curve, therefore, is first observed when the child is one or two years old, but it is not until some time later that it is habitually present, and I am not prepared to say when it actually occurs. It can be obliterated up to adult life, and I rather suspect in many cases even later. The influence of the muscular system is important not only in forming two of the spinal curves, but in maintaining them afterwards. I am convinced that the greater rigidity of the body that is found after puberty is largely dependent on the muscles. The tonicity of the muscles has a great deal to do with retaining the curves of the spine and with limiting its movements. Many of the feats of contortionists are due to this power of relaxing antagonistic muscles, and, as a rule, we find in children a greater proportion of muscle to tendon than in adults. It is, therefore, due more to the want of power to relax the muscles than to the lack of a peculiar formation of the bones and joints that children cannot perform many of these feats. The importance of the muscles in distortions is very great. The spine of the child is flexible in many ways, and the unruly pull of a muscle may easily produce a lasting effect. Not only should the muscles have strength enough to maintain the figure without conscious effort, but their action should be symmetrical on both sides, and should also have a proper relative force before and behind (Case 47, Lecture V., page 145). The importance of light gymnastic exercises is now so generally understood that I need do no more than allude to it. What, however, is of great practical clinical interest in connection with the anatomical and physiological facts concerning the spine, spoken of above, is the way in which they distinctly emphasize the value of this preliminary knowledge in the study of preventive medicine. This point will be spoken of in a later lecture.

What I have just told you regarding the curves of the spine at different ages will, I think, be better understood and remembered if you will for a moment again look at these lines (Diagram 2, Lecture II., page 28), representing the curves of the infant's spine at birth and also at different age to the period of standing.

These lines were made at my suggestion by Professor Dwight.

SURFACE ANATOMY.—The surface anatomy of the spine is of m importance in the adult, and must not be overlooked in the child, wher presents striking differences. In the first place, a prominent feature in adult, especially in a muscular male, is that a depression is found wher the skeleton shows a prominence, owing to the development of the mus Thus, the skeleton shows a ridge of spines in the middle line of the b with a valley on either side; but during life normally we have a median row between two swellings formed by muscular masses. In the infant th not the case (except perhaps in the neck), but the back is rounded; later i more flattened, and the line of the spinous processes, far from being i depression, is rather prominent. This is the more remarkable as when examine the dissected spine from behind we find it very different from of the adult. In the infant the laminæ look more directly backward, their presence in the median line is marked by knobs and ridges v different from the spine of the adult. Up to a year, or perhaps eight months, the proportions are not very different, but the spine at three sh that a great change has occurred, for the spinal processes now stand ou a prominent row, and present very nearly adult proportions. The grea difference is in the dorsal spines, which are relatively broader at their poi and less gracefully drawn out than in the adult. The bodies of the verte still remain less deep, and therefore the relative positions of the spines bodies show less difference than might be expected. For example, the of the spinous process of the seventh dorsal vertebra in the adult reac down to the lower border of the body of the eighth vertebra, or the head the ninth rib. At three it goes very nearly as far, though its shape is the same. At six or seven the spine has made still further progress towa the adult proportions. By the end of the second year the back of living child is not only flatter and broader (the results of continu changes), but there is the appearance of the median furrow, and at five six the differences in this respect from the adult are not marked. It barely possible to count the spines in the infant and young child, and three and four years it is not very easy, though less difficult than in the adu

PROMINENT SPINOUS PROCESSES.—A source of error is the adjecti "prominens" applied to the seventh cervical vertebra, which naturally s gests that its spine is the most prominent in the back of the neck. This not usually the case. The first dorsal spine is the most prominent in t region. The atlas has no spine at all; the spinous process of the axis thick and prominent, perhaps relatively less marked in the child than in t adult; the third and fourth spines are very small; the fifth is not mu larger; but the sixth projects more, and the seventh is said to be usual the first prominent one. He who trusts, however, to this rule is very lia to error, for the relative size of the lower cervical spines varies considerab

The sixth may be the first to assume prominence, and the seventh cervical and first dorsal may exceed it but little. It is easier to examine a child of three years and upward than an adult, on account of the greater softness of the tissues, which allows us to feel more deeply in through the furrow of the neck, and, having recognized the axis by alternately flexing and extending the head, to count the cervical vertebræ in order. If it should be in any case absolutely impossible to feel the third and the fourth, it is better to allow a certain space for them and to call the next one the fifth than to assume arbitrarily that a certain one is the seventh. Confirmatory evidence may be gained from the height of the sternum, to which point I shall return later.

NECK.—I have already referred to the peculiarities of the infant's neck at birth. (Lecture II., page 30.)

CRICOID CARTILAGE.—Symington states that in two children respectively five and six years old the lower border of the cricoid cartilage was found to be at the lower border of the fifth or at the top of the sixth vertebra. I do not quote his observations at intermediate ages, as the position of the head in these measurements varied a good deal. In a girl of thirteen he found that it had reached the adult position; that is, about on a level with the top of the seventh vertebra.

EPIGLOTTIS.—Symington found also that the top of the epiglottis descends during growth from about the level of the lower border of the atlas to the middle of the third cervical vertebra, or even lower.

LARYNX.—This high position of the larynx would imply a greater part of the trachea relatively above the sternum, but this is neutralized by the high position of the latter. The amount of fat in the neck makes the trachea less accessible. The greater distance of the trachea from the surface, as it descends, and the greater danger of meeting the large arteries and veins above the sternum in the child, are points of anatomy so well known in connection with tracheotomy that it seems hardly worth while to insist on them.

Tillaux made a series of measurements of the distances from the sternum to the hyoid, the thyroid, and the cricoid, in men, women, and children of both sexes.

DISTANCE FROM STERNUM TO CRICOID.—I give a condensation of his statements of the distance from the sternum to the cricoid, as the most practical. In twelve women it ranged from five and a half to seven and a half centimetres, the average being six and a half centimetres. In men the variation was greater, ranging from four and a half to eight and a half, but the average was precisely the same. Among the men was a boy of fifteen and a half years, in whom the distance was seven and a half centimetres. Tillaux measured thirty-one children, nineteen girls and twelve boys, ranging from two years up to ten and a half. There seems no reason for keeping the sexes distinct, and I further condense the table by giving the average in the cases of several of the same age, with the following result:

TABLE 7.

Relation of Cricoid to Sternum.

Years.	Distance from Cricoid to
2½	3.5 centimetres.
3	4 "
3½	4 "
4	3.8 "
4½	4 "
5	4.5 "
6	4.9 "
6½	5.5 "
7	5.1 "
7½	4.5 "
8	5 "
8½	5.25 "
9	5.25 "
9½	6.5 "
10	6.5 "
10½	6.5 "

It seems rather remarkable that at ten years the distance shoul great as in the adult, but this may be accounted for by the sub descent of the larynx, and also, probably, by its proportionate enlar (at least in the male) about puberty.

The peculiarities of the relations of the top of the larynx and p to the spine in the young child are points of much practical import which I shall return. The changes which occur during growth largely on changes in the base of the skull, and on the downward of the jaws, which will be considered presently.

HEAD.—CIRCUMFERENCE.—The measurement of the circumfer the head increases very rapidly, and in early childhood almost attai of the average adult's head. We must therefore be careful about gi opinion that the head is relatively large for the age of the child. myself measured over one hundred children of different ages in both tal and private practice in order to get a general idea of the circum of the head and its proportion to that of the thorax. The numbe course, too small to make any precise average deductions from, bu general way I have found these measurements useful.

CIRCUMFERENCE RELATIVE TO THORAX.—Thus, I have fou while at birth the head usually has a circumference of 33 cm. (13 the thorax, measuring over the nipples and just under the angles scapulæ, has a circumference of 1 or 2 cm. (½ to ¾ inch) less. A ch these measurements and proportions soon takes place. In the fourth week, for instance, and extending into the seventh and eighth weeks, (15 inches) for the head and 35 to 36 cm. (14 to 14½ inches) for the I have found to be not uncommon figures. In like manner at five months 42 to 45 cm. (16½ to 18 inches) for the head and 41 to 42 cr to 16½ inches) for the thorax are figures occurring in my measuremen nine months it is not uncommon to find 45.5 cm. (18 inches) for tl and 43 cm. (17 inches) for the thorax. At the end of the first year in a number of cases I found that the circumference of the thorax had reached and even surpassed that of the head, as seen in this infant (Case 8), where the head is 45.5 cm. (18 inches) and the thorax 47.5 cm. (18¾ inches). In exceptional cases the thorax surpasses the head at a much earlier period; and I have even seen it to be a trifle larger at birth, but this is unusual. I have recently measured a healthy infant whose weight at birth was 3800 grammes (8½ pounds), whose head measured 34 cm. (13½ inches) and whose thorax also measured 34 cm. (13½ inches). In the second year I find very varying figures, and the head often still remains larger than the thorax. Thus, in these two infants which I shall now show you, one (Case 9), who is eighteen months old, has a head measuring 49 cm. (19½ inches) in circumference, and a thorax 46 cm. (18¼ inches), while the other (Case 10), also eighteen months old, has a head measuring 47 cm. (18½ inches), and a thorax 45 cm. (17¾ inches). Here is another infant (Case 11), twenty-one months old, who has a head 51 cm. (20¼ inches) and a thorax 50 cm. (19¾ inches) in circumference. My measurements have been taken mostly from boys. The girls that I have measured seem proportionately for the same age to show smaller measurements of the thorax, and to have the thorax overtaking in its circumference the head at a rather later date than is the case with boys. By the second year the thorax has almost always overtaken and surpassed the head. I will now show you some measurements of the head and thorax from two to thirteen years which I happen to find in my notes. They were all males, and it must be remembered that they are not exact averages for a large number of cases, but merely measurements which I found corresponded in a number of children of these different ages. I present them as showing especially how it becomes noticeable when the circumference of the head is taken at random in your general practice, that after the second year the measurements of the head correspond pretty closely, and depend upon the individual rather than upon the age. The thorax, on the contrary, seems to increase year by year.

TABLE 8.

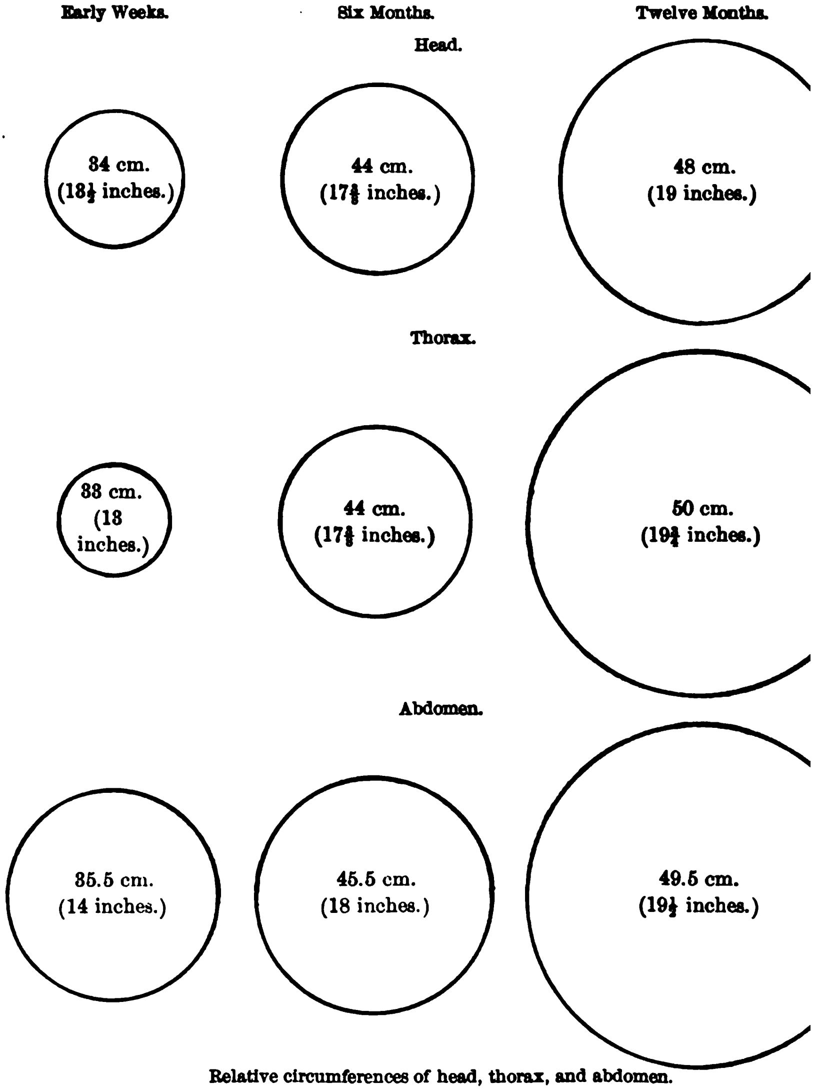
Circumferences of Head and Thorax from Two to Thirteen Years.

Males.

Years.	Head.	Thorax.
2	48 cm. (19 inches)	51 cm. (20⅛ inches).
3	51 cm. (20⅛ inches)	55 cm. (21¾ inches).
4	53 cm. (21 inches)	54 cm. (21¼ inches).
5	53 cm. (21 inches)	54 cm. (21¾ inches).
6	52 cm. (20½ inches)	55 cm. (21¾ inches).
7	54 cm. (21¼ inches)	54 cm. (21¼ inches).
8	53 cm. (21 inches)	59 cm. (23⅛ inches).
9	54 cm. (21¼ inches)	61 cm. (24 inches).
10	53 cm. (21 inches)	62 cm. (24½ inches).
11	56 cm. (22⅛ inches)	63 cm. (24¾ inches).
12	53.5 cm. (21⅛ inches)	63 cm. (24¾ inches).
13	54 cm. (21¼ inches)	66 cm. (26 inches).

This series of circles, representing the circumferences of the head, tl and abdomen, will, I think, show you at a glance what you may exp to the relations of these parts of the child in the first year. They rep the average of a number of actual cases which I have had an opportun closely watching in their nurseries from birth to one year.

DIAGRAM 3.

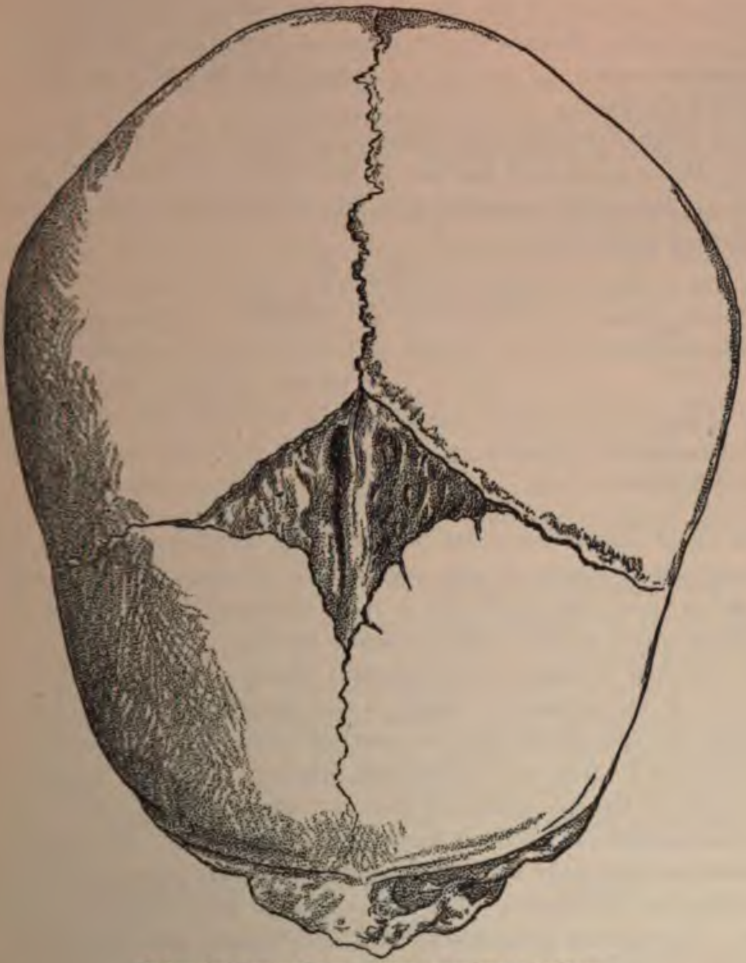


Relative circumferences of head, thorax, and abdomen.

The general idea which you can get from these diagrams will, I help you in your nursery practice, where you have to determine in minutes whether an infant is fairly developed. No especial significanc be given to the circumference of the abdomen in this connection b what I have previously said concerning the liver, as its measuremen course, vary very much normally according to the degree of distention present.

The fact that I have represented the head and thorax equal in the middle of the year, and the thorax larger than the head at the end of the year, does not establish any rule for these periods, as you see from what I have previously said. The diagram merely in a very simple way enunciates that, although there is great activity shown in the growth of the head, this activity is still greater in regard to the thorax.

FIG. 15.



Infant skull, natural size. Anterior fontanelle 4 × 3 cm.
Warren Museum, Harvard University.

FONTANELLES.—The *posterior fontanelle*, although ordinarily quite perceptible at birth, soon disappears, either from overlapping of the bone or from a permanent closure, and is usually imperceptible by the sixth week.

The *anterior fontanelle*, so far as my observation is concerned, seems to grow larger as the infant grows older, up to about the ninth month; this point is, however, disputed, and the increase may be apparent rather real. It also seems to remain stationary, or almost so, from the nin the twelfth month, and then decreases slowly. It should be closed b nineteenth to the twentieth month.

When we study the disease rhachitis you will understand how impo is a knowledge of the closure of the fontanelles.

This skull (Fig. 15, page 63) of an infant in the early weeks of life s very well the increase of the diameter of the anterior fontanelle. W as you see, in this skeleton of the infant at term (Fig. 33, page 118 measurements were 3 × 2 cm. (1¼ × ⅞ inches), this fontanelle measures 4 cm. (1¾ × 1¼ inches).

Face and Cranium.—As I stated in a previous lecture (Lecture page 31), the proportion of the face to the cranium in infancy is as 1 Froriep has also made observations on this point in older children, and the following proportions:

TABLE 9.

Proportions of Face to Cranium.

Age.	Face.		Cranium.
Early infancy	1	to	8
2 years	1	to	6
5 years	1	to	4
10 years	1	to	3
Adult female	1	to	2¼
Adult male	1	to	2

The small size of the facial portion of the skull in infancy and e childhood is well shown in these skulls (Fig. 16, page 67) of the infa birth and at three years, and also in these skeletons (Figs. 33 and 34, 118) of the infant at birth and at nineteen months.

As the child develops, very important changes occur in the base of skull, one of the greatest of which is the downward growth of the Originally the base of the skull is practically flat. The sudden rise of basilar process in front of the foramen magnum, the angle formed wit by the body of the sphenoid, and then the sharp descent of the vomer, adult characteristics of which at birth there is little trace. The nasal ca is shallow and relatively long, the posterior nares are small, and the vo approaches the horizontal. The naso-pharynx has, therefore, very li height. The alveolar processes are still undeveloped, and the ramus of lower jaw is very oblique, so that the cavity of the mouth is small. A consequence, the larynx is, as we have seen, placed very high up. (of the chief causes of its descent is the downward growth of the face.

Brain.—Much credit is due to Dr. George McClellan, of Philadelp for his careful and laborious work, extending over many years, on anatomy of the different periods of life. His careful dissection of infant's brain is very valuable for reference, and I wish to acknowledge use which I have made of it. I desire also to express my appreciation the anatomical work on infants done by Dr. J. W. Ballantyne, of Edinbur

Dura Mater.—An important anatomical condition in connection with the brains of young subjects is that the dura mater is adherent to the skull, and thus prevents the collection of extravasations between it and the bone.

Subarachnoid Space.—The subarachnoid space usually contains a larger amount of fluid in childhood than in later life.

Growth.—I have already mentioned the large proportionate size of the brain at birth (Lecture II., page 37).

Up to the seventh year the brain shows an active growth, and after that year increases slowly in weight. The convolutions are not fully developed at birth, and are gradually perfected as the child grows older. The various centres of the brain which gradually become so highly developed in later childhood have but little action, so far as we can judge, at birth and in the early weeks.

Ear.—The osseous meatus is not developed until about the fourth year. In introducing the aural speculum under four years of age, you should therefore draw the ear forward and downward instead of upward and backward as in older children and adults, or the canal will be bent on itself.

Petro-Squamosal Suture.—The time at which the *petro-squamosal suture* closes is not at present known.

Naso-Pharynx.—Now, if you will again examine these fusible metal casts (Fig. 5, page 33), you will see, as I have already pointed out to you, in this one taken from an infant, that although the inferior turbinate projects slightly into the cavity of the nose, yet there is but a very minute expansion below it and none passing up behind it.

According to Disse, it is this part which shows the greatest growth. It begins to increase in height directly after birth, and goes on pretty rapidly till the beginning of dentition, when it is slow till the second year is completed. After the first set of teeth are cut, the growth is rapid till the end of the seventh year. The increase in breadth occurs in the last-mentioned period, which also is the time in which the growth of the olfactory portion is most marked. Disse states that the posterior opening doubles its size in six months, remains stationary till the end of the second year, and then increases again.

Professor Dwight's measurements on bones are as follows:

TABLE 10.

Age.	Height of Posterior Nares.	Breadth between Pterygoid Processes at Hard Palate.
About birth	6 to 7 millimetres.	9 millimetres.
From 12 to 16 months	13 “	16 “
“ 12 to 18 “	15 “	16 “
“ 14 to 20 “	14 “	17 “
“ 18 months to 3 years	15 “	21 “
“ 2 to 4 years	15 “	20 “
About 6 years	16 “	20 “
7 or 8 years	20 “	22 “
About 11 years	18 “	22 “
17 years, female	22 “	20 “

We may compare with the above, ten measurements which P Dwight has made on adult skulls. I give both the average and the e of variation.

TABLE 11.

Ten Adults.	Height of Posterior Nares.	Breadth between P goid Processes at Palate.
Average	28.4 millimetres.	27.7 millimetr
Extreme	28 and 31 "	24 and 31 "

These figures show that the height does not gain the predominan adult age. At the end of the seventh month the nasal cavity approa adult shape, though it seems broad in proportion, and has not, of attained its full size. Merkol has shown that in later adolesce growth of the respiratory portion takes place chiefly in the middle In infancy the posterior border of the vomer is very oblique. W growth downward of the jaw this obliquity is much diminished at of seven or eight years.

Eustachian Tubes.—The course of the Eustachian tube and th tion of its opening undergo changes corresponding to the developn the nasal cavity. As I have already told you, at birth the tube is h tal, or nearly so. In the adult the cartilaginous portion slants dow Nevertheless, the opening of the tube is opposite a higher part of t in the adult than in the child. In the fœtus the opening is below th of the hard palate, which it reaches at birth. Up to the ninth mont birth, according to Disse, there is but little change. After that tim ever, the opening is distinctly higher than the floor of the nasal c At four years, Kunkel found it to be three or four millimetres highe the adult it is opposite the end of the inferior turbinate bone.

Pharyngeal Tonsil.—The pharyngeal tonsil increases after birt by the end of the first year has a length of eighteen millimetres.

Professor Dwight tells me that he failed to satisfy himself of the pr of anything that could be called a pharyngeal tonsil in the head of nourished child of four weeks which he recently divided in the media There is probably much variation. Dr. Dwight has a beautiful spe of one in a similar section of the head of a child of three years or les has a length of about twenty millimetres, and narrows most striking passage from the nose to the lower part of the pharynx.

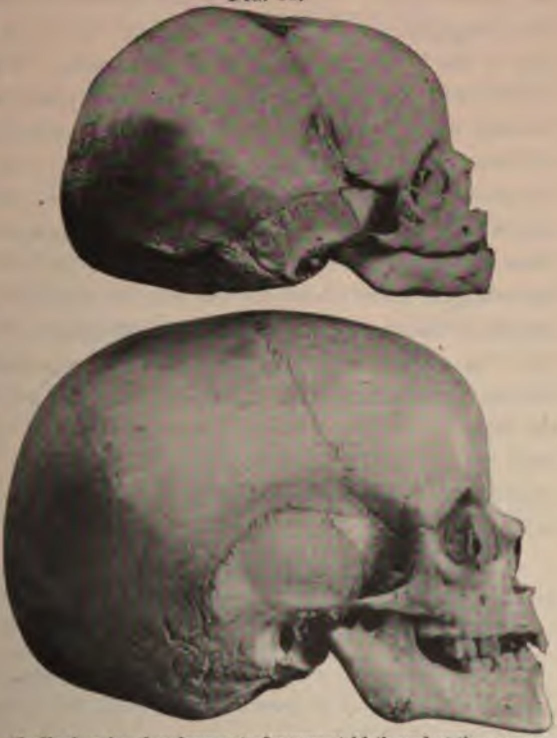
From the tip of the uvula to the top of the epiglottis Braune fou distance to be twelve millimetres in the median section of an adult f In Symington's section of a boy of about six years it is five millimetre a section of a head of three years or less it is not over two millimetres, another of four weeks we find that had the mouth been closed wh head was frozen, the parts would probably have been in contact. The p progress of the changes from the infantile condition is still to be obs I may say, however, from the sections at the Harvard Medical School, Symington's plates of children of six and thirteen years, and from measurements of children, that the change in the first two or three years is very great, and that the pharynx of older children resembles more that of the adult than that of the infant. Indeed, at four weeks we find the tip of the epiglottis on a level with the lower part of the odontoid process, but, of course, by opening the mouth and depressing the soft parts space may be gained.

HARD PALATE.—In a child of three years or less the line of the hard palate strikes about the middle of the basi-occipital bone. It would hardly be possible, without passing the finger round the soft palate, to feel much higher than the arch of the atlas. The base of the odontoid process would be under the mucous membrane seen at the back of the throat through the open mouth. The tip of the epiglottis is at the junction of the odontoid with the body of the axis. I doubt if more than the very top of the third vertebra could be satisfactorily explored. At six and at thirteen (Symington's plates) I find that the line of the hard palate has about the adult direction, —that is, it strikes about the top of the atlas or the basilar process near its beginning. In both the finger could probably examine the vertebræ from the first to the fourth inclusive. The atlas, however, would be reached with much more difficulty in the older than in the younger subject, as the relations of the soft palate are more nearly those of the adult.

MOUTH.—As the infant grows older the mouth becomes an organ more adapted for certain uses beyond that of a mere means of entry for the food to the stomach.

MAXILLARY BONES.—The ossification of the maxillary bones begins early, progresses slowly, and, together with the final formation of the jaw, is completed at puberty. These skulls (Fig. 16), one of an infant born at term, the other of a child three years old, represent the characteristic incomplete development of the ramus of the inferior maxillary bone in the early weeks and months of life, and its almost complete development at three years.

FIG. 16.



Skulls showing development of ramus at birth and at three years. Warren Museum, Harvard University.

The chief characteristic, as you see, is the oblique angle which th makes with the body of the bone at birth, and this becomes more when you compare it with the jaw at three years. You will obse much greater proportion of the ramus to the body of the bone at thre and the nearer approach to a right angle where they join.

Teeth.—Fleischmann's work on this subject is worthy of especia tion, as it will elucidate many points of interest when we come t in a later lecture of diseases of the mouth and difficult dentition description of the development of the teeth, and McClellan's desc which can be found in the first volume of Keating's "Cyclopa the Diseases of Children," need hardly be mentioned in detail, b provide us with facts which will in a measure explain certain sy of clinical interest during the period of dentition. The developm the first set of teeth begins at about the seventh week of intra-uter and, progressing slowly, is completed about the end of infancy. A the twenty embryo teeth, ten in each jaw, are so enclosed in the a processes that nothing but the smooth mucous membrane is appa the gums above. Below, they are connected with the branches of t rior dental nerve (an important clinical fact to be remembered) throug ings at the bottom of the alveolar processes. When calcification of t of the tooth begins, elongation also takes place, and, as the tooth is e in bony walls below and on the sides, it gradually grows through th of least resistance, namely, the gum, which covers the top of the a processes. The continued pressure gradually causes atrophy of the membrane, and the crown of the tooth appears on the edge of the The various teeth come through the gum at times which are re according to their development, that is, at times corresponding to the cation of their roots and consequent elongation. This process usuall place in groups and with considerable regularity in the average infant. Variations both as to the order in which the teeth appear the time of their appearance are so common that it seems hardly cable to have set rules designating these times. The experience of di physicians seems to differ, but all practically are guided by very g rules.

An infant may be born with one or more teeth, as you see in this infant f days old, which has just been brought to the clinic to be operated on by Dr. A Thorndike, who kindly presents the case for your inspection. see that it has an everted intermaxillary bone, on the out of which is the left middle incisor, which evidently came t the gum before the infant was born.

FIG. 17.

Upper incisor tooth in infant at birth, natural size.

The first tooth may appear at any time durin first year of life, or may be delayed until the s year without any other apparent vice of development. In like ma every kind of variation may be met with in the order in which the teet pear, without the slightest evidence of any pathological condition, men otherwise, being found either at the time or later. It is therefore unnecessary to alarm the parents by stating that their child is abnormal because it has not cut a tooth in the first year. We should, however, carefully watch these children and be sure that their food contains the proper nutritive elements not only for their age, but also for their individual digestion.

The appearance of the teeth in groups suggests certain practical divisions which I shall make use of in later lectures to determine various questions, such as the best time for weaning, or for vaccination. These divisions constitute the *dental* and *interdental periods.* In my individual experience, the first tooth appears at about the sixth or seventh month, though at times I find it much earlier, as at the fourth month, and later, as at the ninth, tenth, eleventh, or twelfth month. The first tooth which develops sufficiently to come through the gum is in most cases one of the middle lower incisors. The groups and the dental periods, allowing always for many variations, are, as I have noted them, as follows:

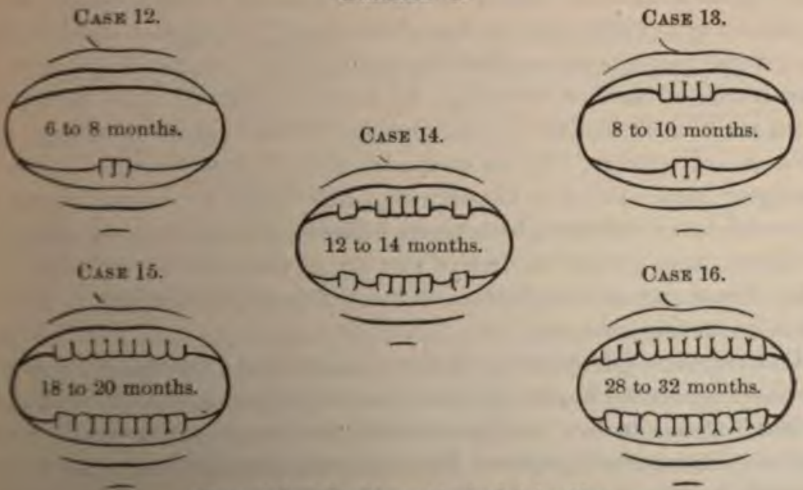
TABLE 12.

Temporary Teeth. First Dentition. Twenty in Number.

	Dental Periods.	Groups of Teeth.
I.	6 to 8 months	2 middle lower incisors.
II.	8 to 10 months	4 upper incisors.
III.	12 to 14 months	2 lateral lower incisors and 4 first molars.
IV.	18 to 20 months	4 canines.
V.	28 to 32 months	4 second molars.
		20

I shall now pick out a number of infants in these various periods who happen to have their teeth corresponding to them, and I should like you to examine their mouths. Here are five typical cases which will illustrate what I have just said.

DIAGRAM 4.


CASE 12. CASE 13. CASE 14. CASE 15. CASE 16.

Five periods of development in the first dentition.

The second set of teeth begins to replace the first at about the six and this table will aid you in remembering their order :

TABLE 18.

Permanent Teeth. Second Dentition. Thirty-two in Number.

Years.	Groups.
6	4 first molars.
7	4 middle incisors.
8	4 lateral incisors.
9	4 first bicuspids.
10	4 second bicuspids.
11	4 canines.
12	4 second molars.
17 to 25	4 third molars (wisd
	32

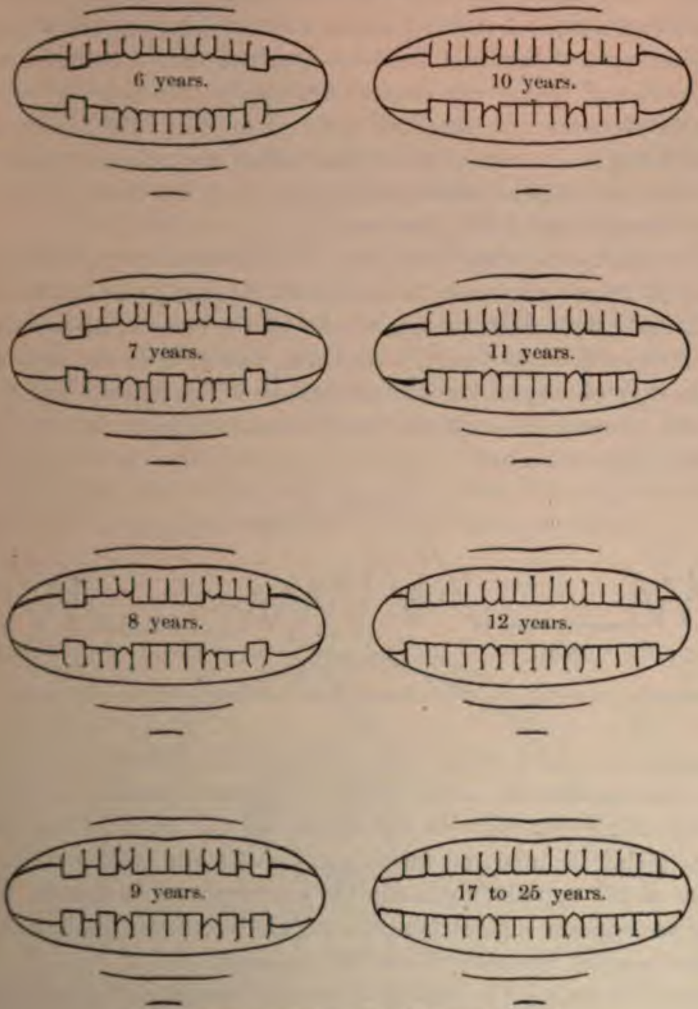
The first four teeth of the second dentition are usually called t year molars. They do not replace any of the permanent teeth, but, having grown so as to provide space back of the temporary teeth, pear back of and next to the second molars. This usually occurs the sixth year.

In the seventh and eighth years the permanent incisors replace the temporary set. In the ninth and tenth years the bicuspids rep temporary molars. In the eleventh year the permanent canines re temporary, and in the twelfth year the four second molars appea really completes the second dentition of childhood, twenty-eight teet remaining four molars belong to a period of adult growth. Dia (page 71) shows very clearly the manner in which the permanent teet the temporary set between the ages of six and twenty-five years.

THORAX.—Top of Sternum.—Professor Dwight found on ex two children each ten months old that the top of the sternum wa nearly opposite the disk between the first and second dorsal vertebra the other a little lower, near the top of the second. In a black child years, whose proportions were strikingly infantile for her age, it was lower border of the first vertebra. In the three-year-old child the for the work on Dwight's "Frozen Sections" it was opposite the lo of the second vertebra. In the median section of a boy about six y Symington found the top of the sternum a little below the level of th the second dorsal vertebra; he believes, however, that this was an in peculiarity, as in several children of that age he found nearly the ad tions. From several observations on the living subject Dwight is to agree with this statement.

Diameters.—The antero-posterior diameter of the interior of th is to the transverse diameter at three years, according to Dwight's " Sections," as one to two, and in a child of from five to six (Syming depth is even relatively greater. The ribs bend much less backward the adult, and the back, as has been said, first becomes rounder a flatter. At four or five years great progress in growth has been made, and the infantile form of the thorax has wholly disappeared. Slight changes, however, probably go on for some years.

DIAGRAM 5.

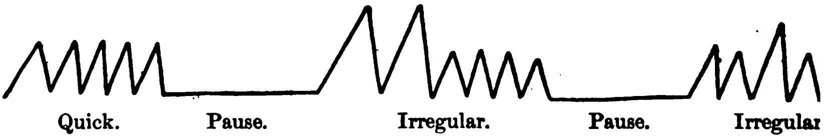


Eight periods of development in the second dentition.

OSSIFICATION.—Towards the end of the first year the bone-centres of the sternum have grown, and the sternum has gained a good deal in stability. New points of ossification have probably appeared, but still the sternum is essentially cartilaginous, the bone merely consisting of islands in a sea of cartilage. At two years of age the manubrium and the second and third pieces are nearly ossified, but their shape is made by their cartilaginous borders. At three years I have twice seen the manubrium and the second piece of the sternum presenting in bone their real shape, while the t piece was still framed in cartilage. Sometimes, however, the process of fication is more backward. The ossification of the lower part of the ster is less advanced than the upper part. As to its relative size opinions d While it seems to me that it is usually small, I must admit that statisti not confirm this view. Probably the individual variation is very g The ribs being comparatively horizontal, the cartilages rise very little, at the lower part of the chest in front they are nearly together, ma narrow intercostal spaces, the seventh cartilages often meeting below body of the sternum. In the dead body of a young child, especial it be emaciated, it is striking to see how, after the cadaveric rigidit passed away, the sternum and cartilages, forming the front of the c fall in at the point where they join the ribs.

RESPIRATION.—At birth there is no decided type of respiration fo two sexes, as I have proved by a number of observations. As the inf both male and female, however, grew older and a more equable respir mechanism became established, I found that, as a rule, in the early m of life the type of respiration was abdominal. This infant (Case 17), months old, presents the irregular respiration of infancy, but you se type is distinctly abdominal.

CHART 2.



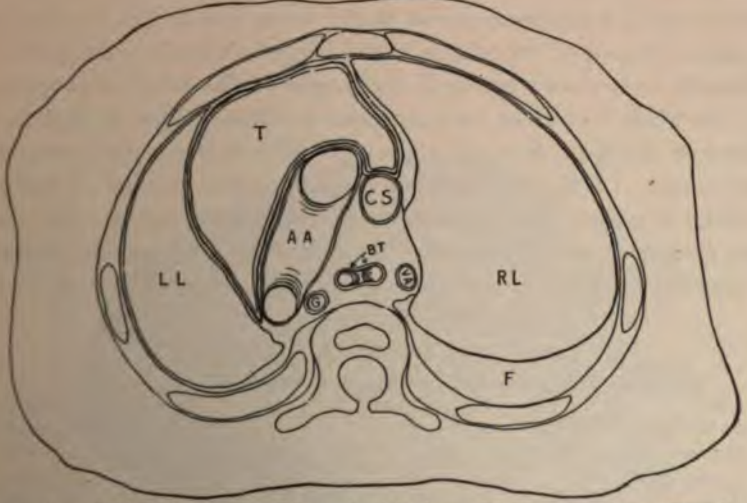
Respiration for one-half minute in a healthy infant nine months old: awake, but quiet.

DIAPHRAGM.—In a child three years old Dr. Dwight found the phragm to be opposite the lower part of the eighth vertebra, and in an child it was at the disk between the eighth and the ninth. Both the dren were girls. In a boy of five it was opposite the middle of the n and in one of six opposite a point in the lower half of the ninth; in a of six it was opposite a point between the ninth and the tenth, and i of thirteen opposite the lower border of the ninth.

THYMUS GLAND.—The *thymus* is most developed in the first two of life, but it persists longer than was formerly taught. During its gr development it is found in the neck as well as in the thorax, exten perhaps 2 cm. ($\frac{4}{5}$ inch) above the sternum, which, you must remember, small part of the surface of a child's neck. The thymus extends dow anterior mediastinum, lying on the pericardium in two long lobes on e side of the median line. The extent of these lobes is very variable, an two are not usually symmetrical. I have seen them, even in an infa developed that the longer nearly reached the lower end of the sternum; it is very uncommon for it to reach the diaphragm. These prolonga become thinner as they descend. The thymus is a thick mass behind the first piece of the sternum, where it rests on the top of the heart against the great vessels concealing the innominate veins, more or less of the superior vena cava and the arch of the aorta, and extending back to the trachea. Lower down it extends on either side into the angle between the pericardium and the lungs, or rather pleuræ. Except for the front view, obtained by removing the sternum, the size and relation of the thymus are best shown by frozen sections. One, made by Dwight, of a child of three years or less, at the Harvard Medical School, gives a most remarkable view of it. The section in question runs nearly horizontally from the top of the fourth dorsal vertebra to just above the junction of the second costal cartilage with the sternum.

The cavity of the thorax seems to be divided into three parts, one on either side of the lungs and a median one occupied by the thymus, the transverse part of the arch of the aorta, with the superior vena cava on its right, and the trachea and œsophagus behind. The area occupied by the thymus is very nearly equal to that of the left lung. The thymus reaches backward on the left of the aorta behind the lēvel of the front of the spinal column. There is also what seems to be a piece of it between the vena cava and the trachea. On the upper surface of the same section, at about the level of the sterno-clavicular articulations, it is in front of both innominate veins and behind the right one. The lungs are prevented from approaching each other so nearly behind the manubrium as they do in the adult.

FIG. 18.



Frozen section, child of three years: RL marks right lung; LL marks left lung; T marks thymus gland; G marks gullet; CS marks superior vena cava; AA marks aortic arch; VA marks vena azygos; F marks some fluid which happened to be in the right chest; BT marks bifurcation of trachea.

The section of the child just described shows that behind the manubrium there is much more of the thymus to the left than to the right of the median line, and its dulness on percussion must have been evident at the left of the sternum. Below it merges into the cardiac dulness, no distinction between them is possible.

The theoretical results of enlargement of the thymus are very seri Resting on the anterior and weaker ventricle, which is prolonged upw into the pulmonary artery, it may interfere with the pulmonary supply blood, and by compressing the innominate veins and the superior cav may interrupt the return of venous blood to the lungs. Whether or it may compress the thoracic duct is doubtful, but it certainly may pres the trachea.

The thymus is said often to persist for several years after puberty, observations are not numerous on this point. It seems to disappear fi the neck and from the front of the heart and to remain longest behind first piece of the sternum.

For further information on this subject, I shall refer you to Dr. Jacobi's excellent monograph on the Anatomy and Pathology of Thymus Gland.

HEART.—It is generally held that in the first year of life the long a of the heart is more nearly horizontal than later. The apex is thought many to be higher. I am inclined to think that this is true in the f few years, but somewhat later it may be found in the adult position, or, cases where the lower part of the sternum is backward in development the cartilages crowded together, it may be in a lower space than normal. is not unlikely that a subsequent change in these portions of the walls wou correct this. Thus, if in the early condition the apex were at the si intercostal space, a lengthening out of the lower end of the sternum mig cause such a descent of the ribs as would bring it into the fifth space.

Weight.—As shown by Boyd, the weight of the heart in proportion that of the whole body does not vary much at different ages, so that the re tive labor of the heart does not materially differ between the young subj and the adult. In the first few years, however, the increase of the weig of the heart is greater than at about the fourth or fifth year, and this increa is again greater at about puberty. These are facts of practical importan to be remembered when we are studying the diseased conditions of the hea

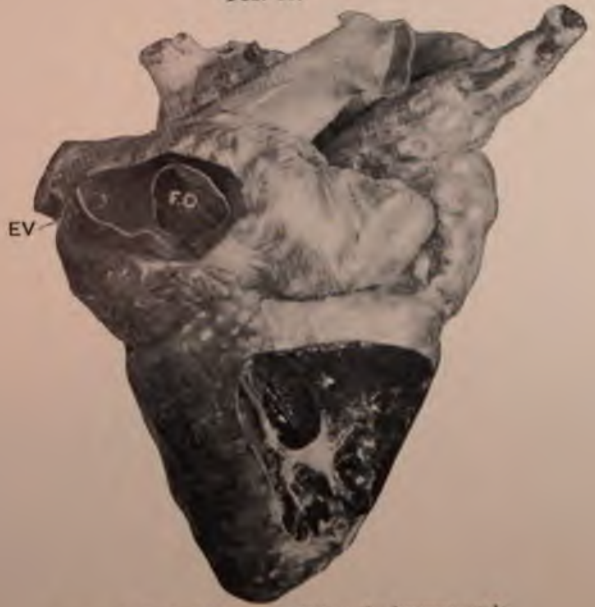
TABLE 14.

Weights of the Heart during its Development. (*Boyd.*)

Age.	Grammes.
At birth	20.6
1½ years	44.5
3 years	60.2
5½ years	72.8
10½ years	122.6
17 years	283.7

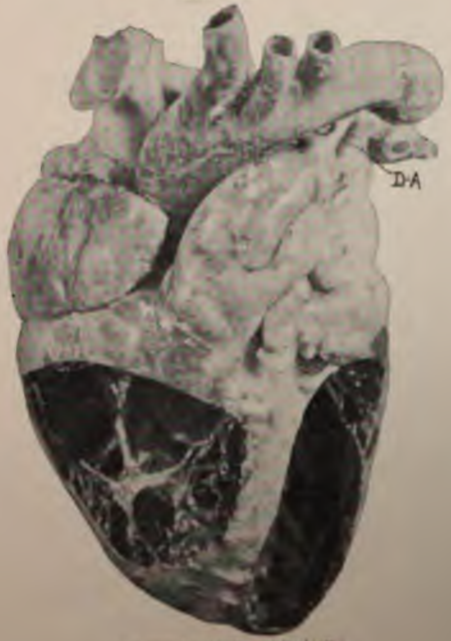
I should now like you to examine carefully this heart of an infant in th early weeks of life (Figs. 19 and 20), at a period when the heart and bloo vessels have completely changed from the fœtal type to that of the adu

Fig. 19.



EV marks Eustachian valve ; FO marks foramen ovale.

Fig. 20.



D A marks ductus arteriosus.

It has been prepared by Dr. Franklin Dexter to show the different cavities and also the remains of the fœtal conditions. First, looking into the right auricle (Fig. 19), you see the remains of the *Eustachian valve,* and the distinctly outlined but closed *foramen ovale.* Next, on turning the heart around (Fig. 20), you see this small tense cord connecting the aorta and the pulmonary artery. This is the obliterated *ductus arteriosus*, and, as you see, it pulls the aorta somewhat out of line, a condition which you will find to be of considerable significance when we are studying diseases of the heart.

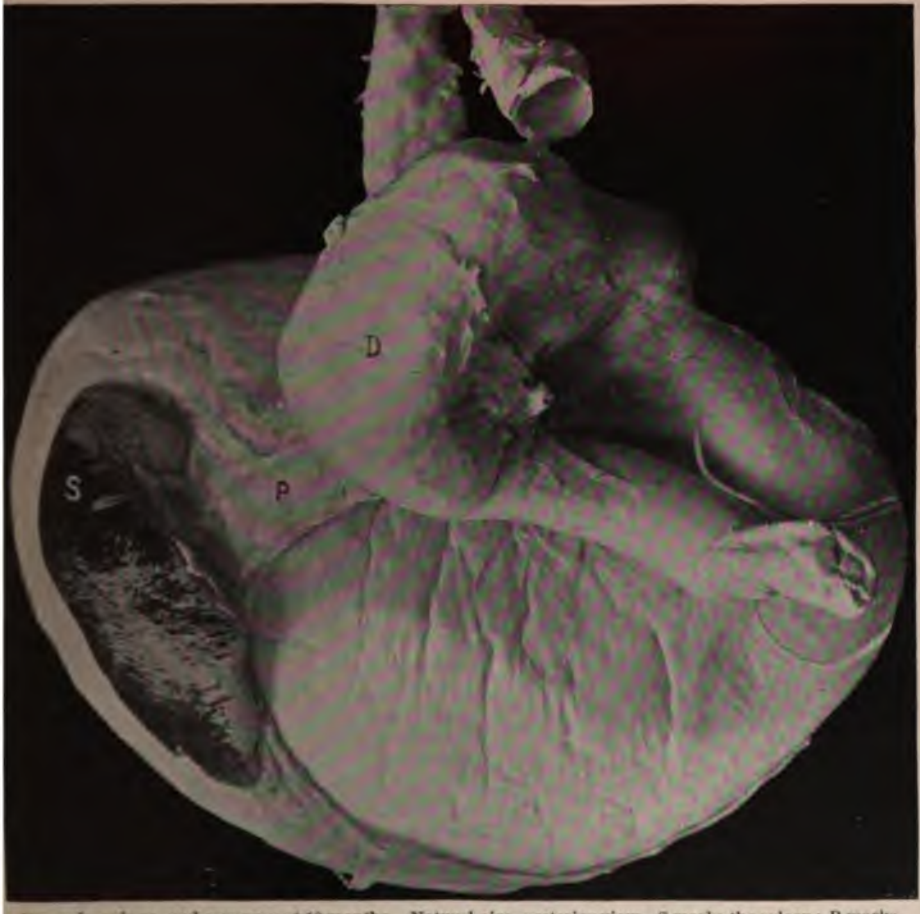
BLOOD-VESSELS.—Jacobi, in speaking of the extensive work of Thoma on this subject, writes as follows:

"According to a number of actual observations made by R. Thoma, the post-fœtal growth is relatively smallest in the common carotid, and largest in the renal and femoral arteries. Between these two extremes there are found the subclavian, aorta, and pulmonary arteries. These are differences which correspond with the differences in the growth of the several parts of the body supplied by those blood-vessels. In regard to the renal artery and the kidney, it has been found that the transverse section of the former grows more rapidly than the volume and weight of the latter. Thus, it ought to be expected that congestive and inflammatory processes in the renal tissue were almost predestined by this disproportion between the size of the artery and the condition of the tissue. Moreover, the resistance to the arterial current offered by the kidney substance depends also upon the readiness with which the current is permitted to pass the capillaries. It has been found experimentally that within a given time more water proportionately can be squeezed through them in the adult than in the child. These anatomical differences may therefore be the reason why renal diseases are so much more frequent in infancy and childhood from all causes, with the exception of that one which is reserved for the last decades of natural life, atheromatous degeneration."

Pulmonary Artery.—Professor Dwight has found the origin of the pulmonary artery at ten months to be near the top of the first intercostal space and at the same age at the level of the second costal cartilage. At three years he found it near the lower border of the first space, also near the lower edge of the second cartilage, and again at about the lower part of the second space. On the whole, considering the great variations which occur in the adult, as recorded by Gibson and others, it is doubtful if there is any essential difference at different ages. If we say that in the infant it is rather higher than later we have stated about all that is justifiable.

LUNGS.—At what age the lungs reach their full expansion forward has not been determined. It would appear that it is not before five or six years, and it is probably still later. As the chest expands laterally the lungs of course increase, and the relatively greater size of the heart to the lung in the infant depends essentially on the size of the lungs. During the first year of life (according to Northrup) the alveolar walls are thick and their blood-vessels are held loosely. It is not until the fourth or fifth year that the proportionate adult development between the alveoli and the b is attained, and the stroma has become dense and binding, restr the capillaries as in adult life. In infant life the underlying loose lining the bronchial tubes gradually binds the mucous membrane fibro-muscular wall. From this time it keeps pace in its growth wi other compact tissues, until in adult life it appears as dense fibrous During the first two years the air-cells have not attained the propor capacity which exists in adult life, and the bronchial tree is still proportion to the dilating and multiplying alveoli. Again the developed from the terminal bronchi have covered themselv continuous layer of flat nucleated epithelium. In its subseque in adult life it is believed that the expanding alveolus does not number of epithelial cells to cover the more extended wall, but enlarges their size, and, still further, that some of the flattened loses its nuclei and expands to form large, very thin plates, calle tory epithelium.

FIG. 21.



Stomach, spleen, and pancreas at 10 months. Natural size, posterior view. S marks the spleen; P marks the pancreas; D, the duodenum. Warren Museum, Harvard University.

LECTURE IV.

ABDOMEN. — TEMPERATURE. — PULSE. — RESPIRATION. — HEIGHT. — WEIGHT.—FEET.—BONE MARROW.—SKIN.—CORD.—FUNCTIONS. —BLOOD. — LYMPHATIC SYSTEM. — URINE. — INTESTINAL DISCHARGES.—INFANTILE SKELETONS.—NORMAL INFANTS.—TOPOGRAPHICAL ANATOMY OF THE EARLY PERIODS OF LIFE.

ABDOMEN.—Liver.—The liver is, as I have told you, proportionately large at birth and in early childhood, and, as I shall presently show you on the living subject, can be felt below the edge of the ribs in the right hypochondrium, its border being about 1 or 2 cm. ($\frac{3}{8}$ to $\frac{6}{8}$ inch) below the lower rib.

Gall-Bladder.—The fundus of the *gall-bladder*, according to McClellan, is in relation to the surface of the body about that of the ninth costal cartilage near the border of the right rectus muscle.

Spleen.—There is nothing especially to be noted in the spleen in childhood, as it corresponds in its position to that of the adult. According to Foster, the spleen grows rapidly in early infancy, but in proportion to that of the adult is both absolutely and relatively smaller. It is said that the spleen when enlarged encroaches more upon the thoracic cavity than in the adult, owing to the greater resistance offered by the costo-colic fold of the peritoneum upon which it rests. My clinical experience, however, does not especially support this view, as in many cases of enlarged spleen from varied causes which I have met in infants it has always seemed to me that the abdomen was encroached upon to a greater extent than in adults, and that both the physical and the rational signs of the enlarged spleen in the thorax were relatively insignificant and often difficult to detect.

Pancreas.—The function and the anatomy of the pancreas correspond very closely to those of the salivary glands. It is situated in front of the first lumbar vertebra, behind the stomach, and, according to the variations produced by age and the growth of other parts, lies somewhere between the umbilicus and the ensiform cartilage.

The relative position of the spleen and pancreas to the stomach and duodenum is very beautifully shown in these organs obtained at the autopsy of an infant ten months old, which lately died in my wards. You see that the spleen is behind the cardiac end of the stomach, and very near its extremity (Fig. 21, organs seen from behind). You will also notice how the pancreas extends from the spleen (its tail being in close apposition to the latter organ) along the posterior surface of the stomach and somewhat upward to the smaller curvature, passing behind the duodenum and its head resting in the concavity of the duodenum. The curve of the duodenum also clearly shown in this specimen.

KIDNEYS.—The kidneys are lobulated at birth, as I showed you in specimen taken from an infant three days old. (Division I., Lecture Fig. 9, page 44.) This condition continues for a long time and then appears, the lobulation being represented by the pyramids of Malpighi. few years after birth the position and relations of the kidney approxim those of the adult (McClellan).

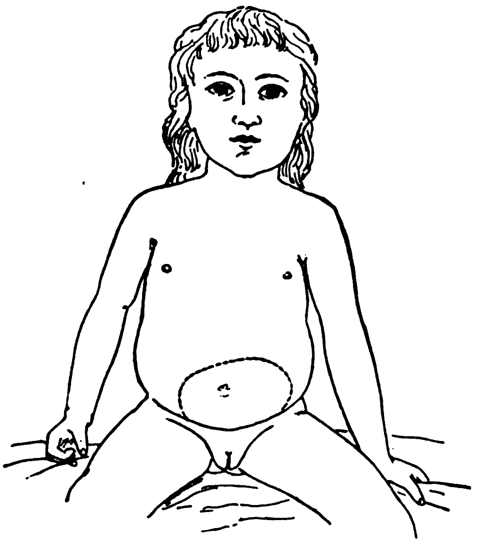
Supra-renal Capsules.—The supra-renal capsules are, as I have t you in Lecture II., relatively large in size, and gradually approach adult proportions as the child grows older.

BLADDER.—Although small at birth, the bladder soon becomes capa of great distention.

Symington, from a frozen section which he made in the median pl through the body of a child seven months old, shows the position of bladder, which happened to be distended. It takes up, practically, the wh of the lower portion of the abdomen, an observation which at once prese to our minds the difficulty of making a correct physical examination of infantile abdomen during life, unless we are sure that the bladder is empt

The above fact was strikingly exemplified in this little girl, three years old (Case who entered my wards at the Childr Hospital yesterday. She was sent to hospital for an examination in refere to the advisability of an operation to move an abdominal tumor. On ins tion, a rounded prominence extend from the pubes to 8 cm. (1½ inch above the umbilicus could be plai seen. By palpation the tumor could felt extending from the right ingui region over to the crest of the left iliu The tumor was soft, elastic, and fl tuating. It was evidently not in abdominal walls, but intra-abdomin The child was said to have been aili for over a week, and to have grown th She passed her urine frequently, but small quantities. Nothing abnormal h been found on an analysis of the uri made before she entered the hospit You see I have marked in black t outline of the tumor as it appeared entrance.

CASE 18.



Girl 3 years old. Distended bladder.

Suspecting a distended bladder, had a catheter introduced, and removed 270 grammes (9 ounces) of urine. The tum immediately disappeared, and, as you see, the abdomen is now soft and resonant.

A practical lesson to be drawn from this case is, that the bladder shoul invariably be carefully examined and emptied before diagnosticating operating for abdominal disease. I have seen a distinguished laparotomist neglect this precaution in a young child while operating for appendicitis, and on opening the abdominal cavity cut directly through the walls of the bladder. The urine flowing out through the wound was the first indication to him that he had failed to appreciate that in early life the bladder is essentially an abdominal organ.

You can see that many peculiarities of the digestive tract may arise from the causes which I have already spoken of in Lecture II. Especially to be noticed, however, are those which are due to the different proportionate stages of development of the parts of the gastro-enteric tract at different ages, and to differences in their peritoneal attachments.

STOMACH.—The stomach grows very rapidly, and peculiarities of shape appear at an early age. I have seen a stomach of four and one-half months which, although small, was relatively broader than in the adult. The adult shape, however, is soon acquired. How permanent this may be is as yet unsettled. There is no doubt that great dilatation may be induced, and it is highly probable that where too small quantities of food are given the normal stomach will contract. It is also very likely that certain shapes are acquired at a very early period. I have seen in a young child a well-marked *antrum pylori*,—that is, a pouch above the pylorus, which, in extreme cases, forms almost a separate chamber. It is evident that the clinical significance of our anatomical knowledge of the growth of the stomach in the first year is very great. This question of growth is, in fact, one of the most important factors in the problem of the substitute feeding of infants, and a lack of its thorough comprehension often leads to most unfortunate results.

Capacity.—There has been much dispute as to the proper method of determining the gastric capacity during infancy. All methods of which I know are open to criticism, but I have found that by combining all the methods and making general deductions I have arrived at very practical conclusions concerning the size of the stomach at different ages. I have also found that my results correspond closely to those of others who have made careful studies of this subject, notably Fleischmann of Vienna, and Holt of New York. One of the methods which I have employed has been a clinical one, which I shall show you, as I happen to have a wet-nurse with a healthy baby four months old here in the ward.

[CASE 19.] The mother is healthy, and has plenty of milk in her breasts. Her milk is evidently in equilibrium, and agrees with her baby, who is digesting it well and gaining about 30 c.c. (1 ounce) a day. Now, if we wish to determine the gastric capacity of an infant's stomach at four months we can experiment with this infant. The weight of 30 c.c. (1 fluidounce) of human milk is very nearly 30 grammes (1 ounce). If then we introduce 30 c.c. (1 ounce) of milk into an infant and weigh it immediately before and immediately after the introduction, the infant should increase 30 grammes (1 ounce) in weight. This method I have proved a number of times to be fairly correct, as you see it is in this especial case (Case 19) within four or five grammes. It is well known among those who deal in cattle that when fat cattle are transported long distances, as from Chicago to New York, they are found to

have lost materially in weight, perhaps thirty or forty pounds. Now, if these cattl allowed to fill their stomachs with water, an increase in their weight will be found spunding exactly to the weight of the water which they have drunk. I find that this i (Case 19) weighs before nursing 7080 grammes (15½ pounds). We will now let it until it evidently is satisfied, that is, practically until it feels that its stomach is full. I immediately weigh it again, and find that it has increased to 7145 grammes (15¾ poun gain of 115 grammes (about 4 ounces). This would, in a general way, denote that the g capacity of this especial infant was 120 c.c. (4 ounces).

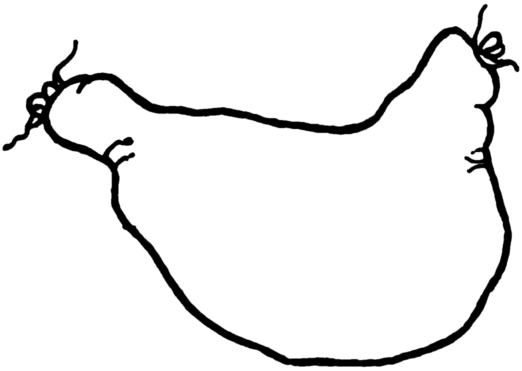
Now, if a number of healthy infants of different ages and of ave weights are fed and weighed in this way, we can approximately by paring the gains in weight which correspond to the same ages deter the gastric capacity for each age. I should not, however, consider thi itself a reliable method for determining the gastric capacity, as it is to many objections, which need not be discussed at present. One sourc error, for instance, is the variation of the infant's appetite, which may c either too great distention or underfilling of its stomach. Another me which I have used is the actual measurement of the gastric capacity at autopsy, with suitable precautions to avoid over-distention. Combining t methods, I have arrived at certain general conclusions, which I shall you in figures. I have in this way also determined that the stomach gr very rapidly in the first three months after birth, grows slowly in the fo month, and is then almost quiescent for about two months. It then be to grow again until it has reached its adult size. Frolowsky's rules fo termining the gastric capacity of young infants approximate in their re so closely my own investigations that I have prepared from them this t of infants' stomachs at different ages and at different periods of gro The tracings of the stomachs are life-size. Frolowsky shows that the acti of the stomach's growth is very great in the first quarter of the first y that it is very slight in the second quarter, and that it again shows a mode activity in the last part of the year. He represents this activity of stomach's growth by the ratio of 1 for the first week to 2½ for the fo week and 3⅕ for the eighth week, while it is only 3⅓ for the twelfth w 3 4/7 for the sixteenth week, and 3⅗ for the twentieth week. As a start point from which to calculate the above proportions I have taken the inf stomach which I presented to you at my lecture on the Infant at Ter representing a fair average capacity for this age, 25 to 30 c.c. (⅚ to 1 ou (page 45, Fig. 10).

This, of course, is also intended to represent an infant with the ave birth weight. This table will with its six tracings explain what I have said about the rapid increase in size which the stomach shows at the pe I have mentioned (Table 15).

TABLE 15.

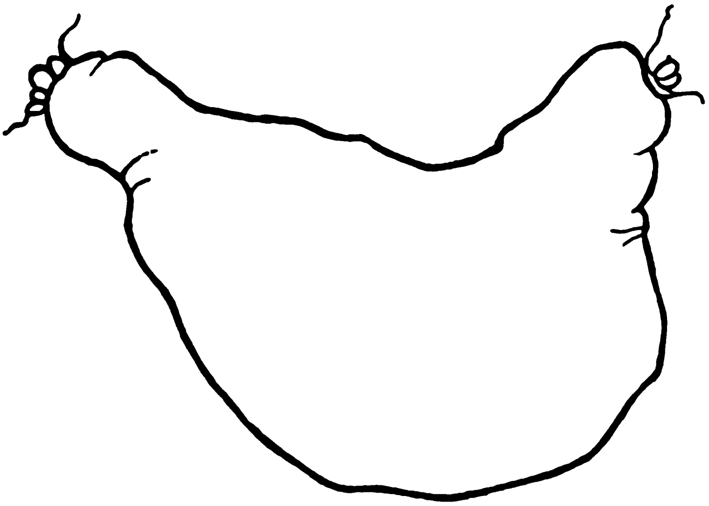
Gastric Capacity in the First Five Months of Life.

I.



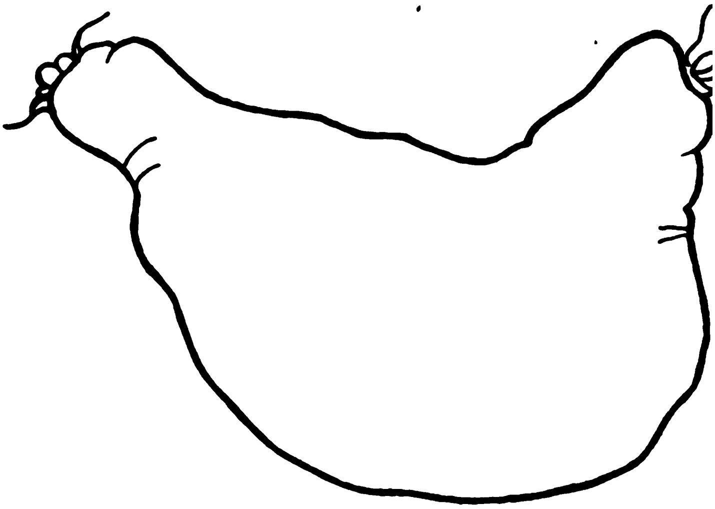
Infant 3 hours old. Capacity of stomach 25 to 30 c.c. (⅚ to 1 ounce).

II.



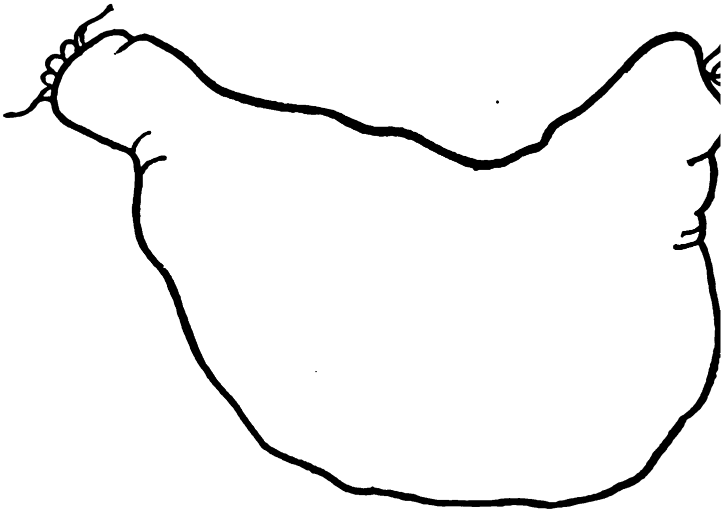
Infant 4 weeks old. Stomach 2½ times larger than I. Capacity 75 c.c. (2½ ounces).

III.



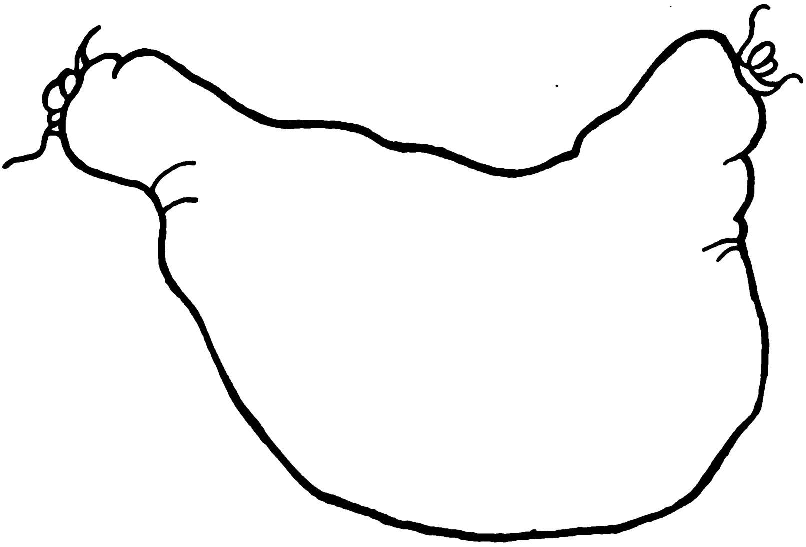
Infant 8 weeks old. Stomach 3¼ times larger than I. Capacity, 96 c.c. (3¼ ounces)

IV.



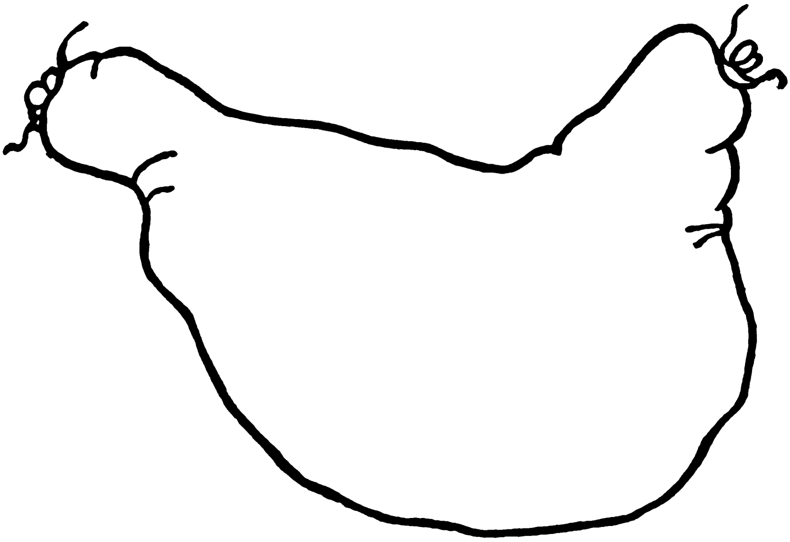
Infant 12 weeks old. Stomach 3⅓ times larger than I. Capacity, 100 c.c. (3⅓ ounces

V.



Infant 16 weeks old. Stomach 3¾ times larger than I. Capacity, 107 c.c. (3.56 ounces).

VI.



Infant 20 weeks old. Stomach 3⅘ times larger than I. Capacity, 108 c.c. (3.6 ounces).

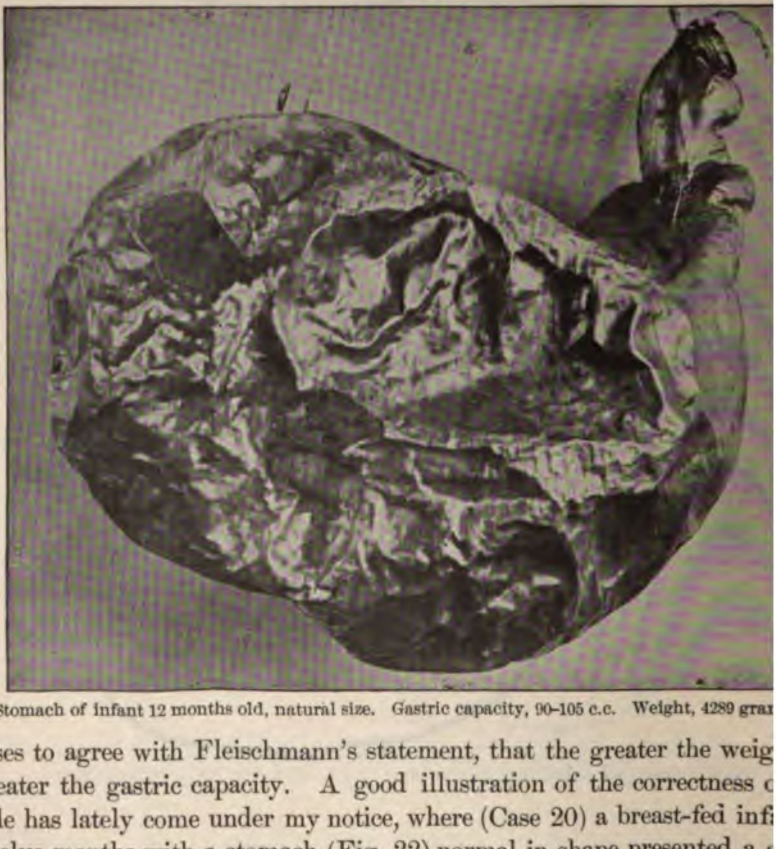
In comparing these measurements with actual stomachs, the
capacity as given for sixteen and twenty weeks is somewhat sma
consider, however, that they are extremely valuable to begin with,
is always better to err on the side of giving too little food than too m

As has been admirably pointed out by Fleischmann, the gastric ca
is greater at the same age in the artificially-fed than in the breast-fed
This observation, however, in all probability only emphasizes the i
tance of bearing in mind the normal gastric capacity of the differen
and of using this knowledge to prevent the overfeeding which has pr
so noticeable a difference between the sizes of the stomach in breast-f
in artificially-fed infants.

Since I have developed my methods of substitute-feeding in con
with milk modification, I have no reason to suppose that when the i
stomach has been properly managed, as I can now accomplish in subs
feeding, it is any larger than in breast-fed infants.

The cause, however, which produces the most uniform individual
ence in the gastric capacity at the same age is the weight of the infant.
inclined from the results of my own observations in a considerable num

Fig. 22.



Stomach of infant 12 months old, natural size. Gastric capacity, 90–105 c.c. Weight, 4289 gra

cases to agree with Fleischmann's statement, that the greater the weig
greater the gastric capacity. A good illustration of the correctness
rule has lately come under my notice, where (Case 20) a breast-fed inf
twelve months with a stomach (Fig. 22) normal in shape presented a

capacity of only 90 to 105 c.c. (3 to 3½ ounces). This capacity corresponded to its weight, 4289 grammes (9½ pounds), about the average normal weight of an infant at eight or ten weeks, rather than to its age, which in the average infant would present a gastric capacity of 240 cubic centimetres (8 ounces).

I have also had under my care an infant of six weeks whose general development and weight corresponded so closely to those of the normal average infant of twelve weeks that it was self-evident that the two ounces of food which would ordinarily have been the proper allowance, so far as the age was concerned, was not sufficient, and that its weight indicated a gastric capacity for an allowance of four ounces, which, in fact, it took and digested with the greatest ease, while with any amount less than the four ounces it was never satisfied.

It will now, I think, be instructive for you to examine these stomachs which from time to time I have obtained at autopsies, and see how they compare with the measurements which I have just given you. Dr. Townsend, who prepared most of these specimens, drew my attention to the important fact that in measuring the gastric capacity it should be done before the stomach is separated from its mesenteric attachment, as otherwise it is easily stretched by the fluid introduced, and will then show a greater capacity than would be within the normal limits during life. I have had some of the stomachs distended beyond their natural size in order to show you how misleading it would be to depend for exact results on this method of investigation. You will therefore find quite a variety of figures representing the gastric capacities, but on the whole they correspond so closely to the rule already stated that they corroborate rather than disagree with the other methods. It is interesting also to note the different shapes which these stomachs represent, as some of them are very different from the usual classical figures represented in books. So far as I could ascertain, these shapes did not occur from any especial disease, such as would influence the outline of the stomach, as has happened in this specimen taken from a case of rhachitis. (Page 849, Fig. 108.)

(The illustrations represent the actual sizes of the stomachs.)

Beginning with the youngest subject, an infant three hours old, you see, as I have already shown you at a previous lecture (Division I., Lecture II., Fig. 10, page 45), that the stomach has the average capacity of the newborn, 25 c.c. (⅝ ounce), although the infant's weight was only 2500 grammes (5½ pounds).

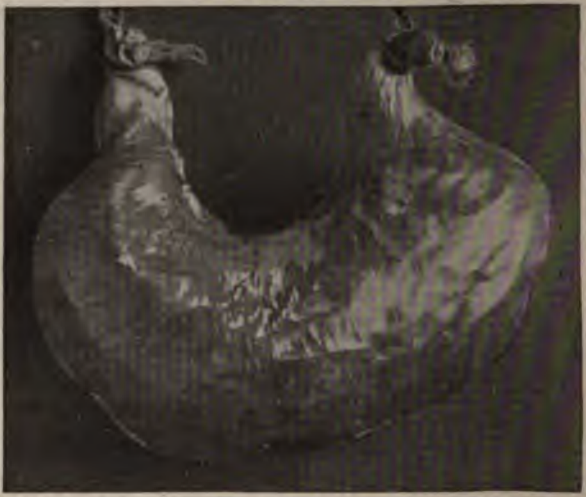
The next stomach (Fig. 23, page 86) was taken from an infant two and one-half days old, and weighing 4000 grammes (8⅘ pounds). The gastric capacity is 25 c.c. (⅝ ounce).

This next stomach (Fig. 24, page 86) was taken from an infant five days old and weighing 3000 grammes (6⅗ pounds). The gastric capacity is 25 c.c. (⅝ ounce).

This stomach (Fig. 25, page 87) was taken from an infant seven days old and weighing 2700 grammes (6⅛ pounds). Its gastric capacit (1⅓ ounces).

These four stomachs (Fig. 10, page 45, Fig. 23, Fig. 24, Fi taken from infants within the first week of life, show us at onc

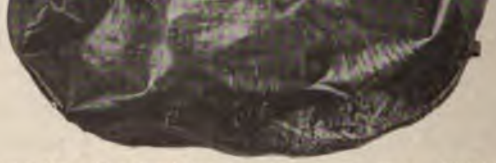
FIG. 23.



Stomach of infant 2⅚ days old, natural size. Gastric capacity, 25 c.c. Weight, 4000 g

cannot always depend on an infant's weight for determining i capacity in the early days of life. Thus, the weights of 2500 3000 grammes, and 4000 grammes all had the same gastric c 25 c.c. Again, the weight of 2700 grammes had a greater capacit

FIG. 24.

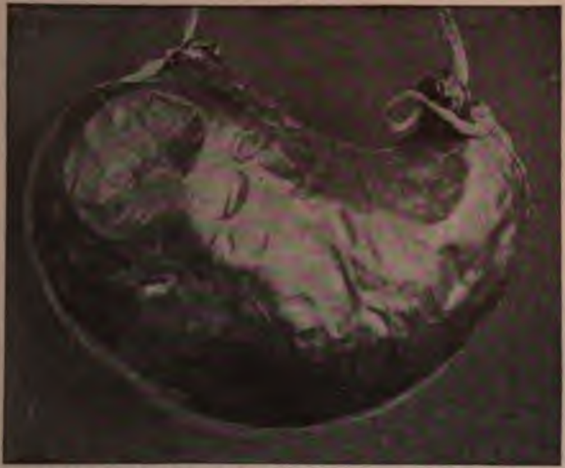


Stomach of infant 5 days old, natural size. Gastric capacity, 25 c.c. Weight, 3000 gra

than the weights of 3000 and 4000 grammes. We must, how allow that there might be an error in measuring the gastric capacit

Do not for a moment think that I am deducing any rules for gr

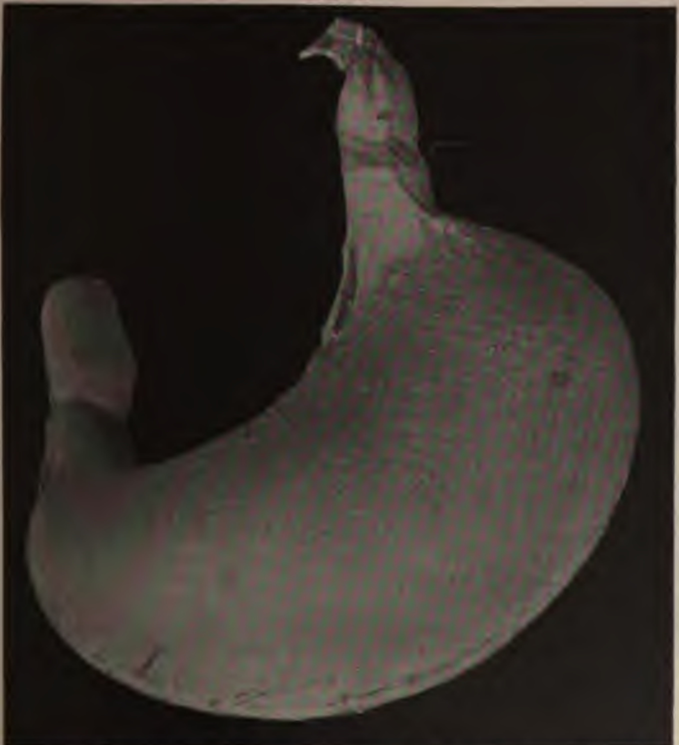
FIG. 25.



Stomach of infant 7 days old, natural size (posterior view.) Gastric capacity, 40 c.c. Weight, 2700 grammes.

this very limited number of cases. You will, however, understand the significance of these figures a little later when we are discussing the feeding

FIG. 26.



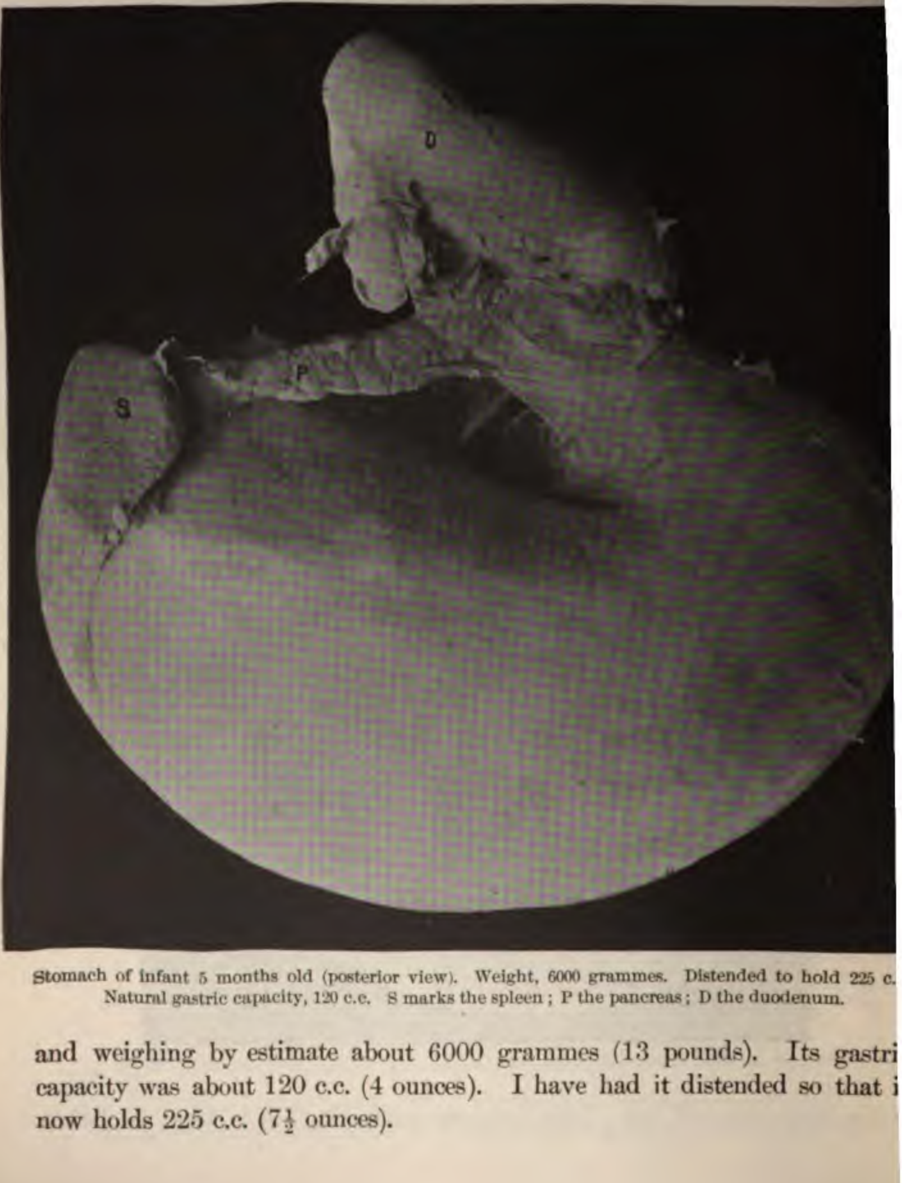
Stomach of infant 12 days old, distended to hold 80 c.c. Natural gastric capacity, 40 c.c.

of the early days of life. This next stomach (Fig. 26) is from an infant

twelve days old. I have, unfortunately, lost the record of its weig
It represents very well, however, the usual shape and position of
stomach in early life, and I have had it distended beyond the li
of its normal capacity, so as to show you the great elasticity of the v
tricular walls to which I have already referred. The gastric capac
was about 40 c.c. ($1\frac{1}{3}$ ounces). As you see it now distended, it holds
c.c. ($2\frac{2}{3}$ ounces).

This next specimen (Fig. 27) shows the stomach, duodenum, spleen, a
pancreas of a well-developed infant five months old, 66 cm. (26 inches) lo

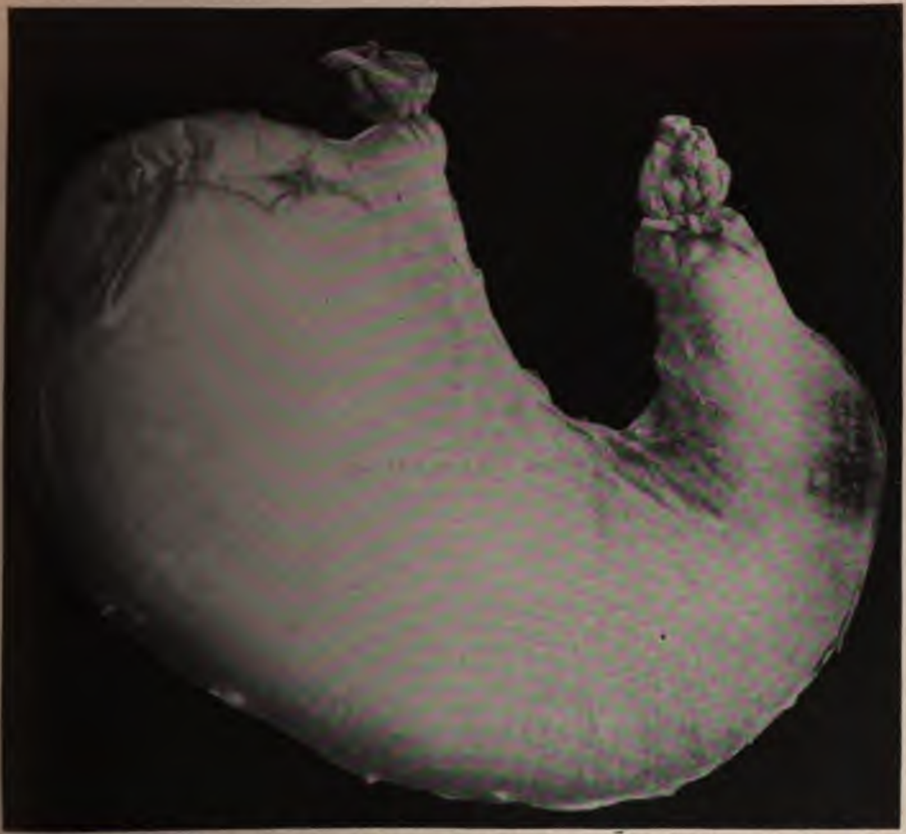
FIG. 27.



Stomach of infant 5 months old (posterior view). Weight, 6000 grammes. Distended to hold 225 c.
Natural gastric capacity, 120 c.c. S marks the spleen; P the pancreas; D the duodenum.

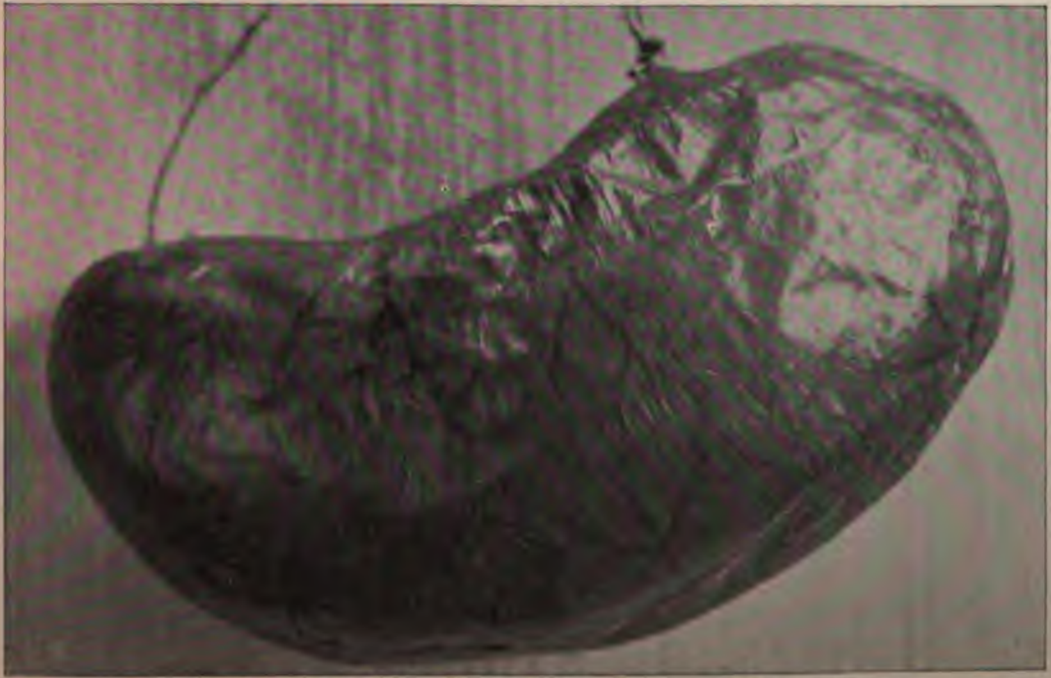
and weighing by estimate about 6000 grammes (13 pounds). Its gastri
capacity was about 120 c.c. (4 ounces). I have had it distended so that i
now holds 225 c.c. ($7\frac{1}{2}$ ounces).

FIG. 28.



Stomach of infant 7 months old (posterior view). Weight, 5230 grammes (11¾ pounds). **Its gastric** capacity was about 150 c.c. (5 ounces), but it has been distended to just twice its size and **now holds 300** c.c. (10 ounces).

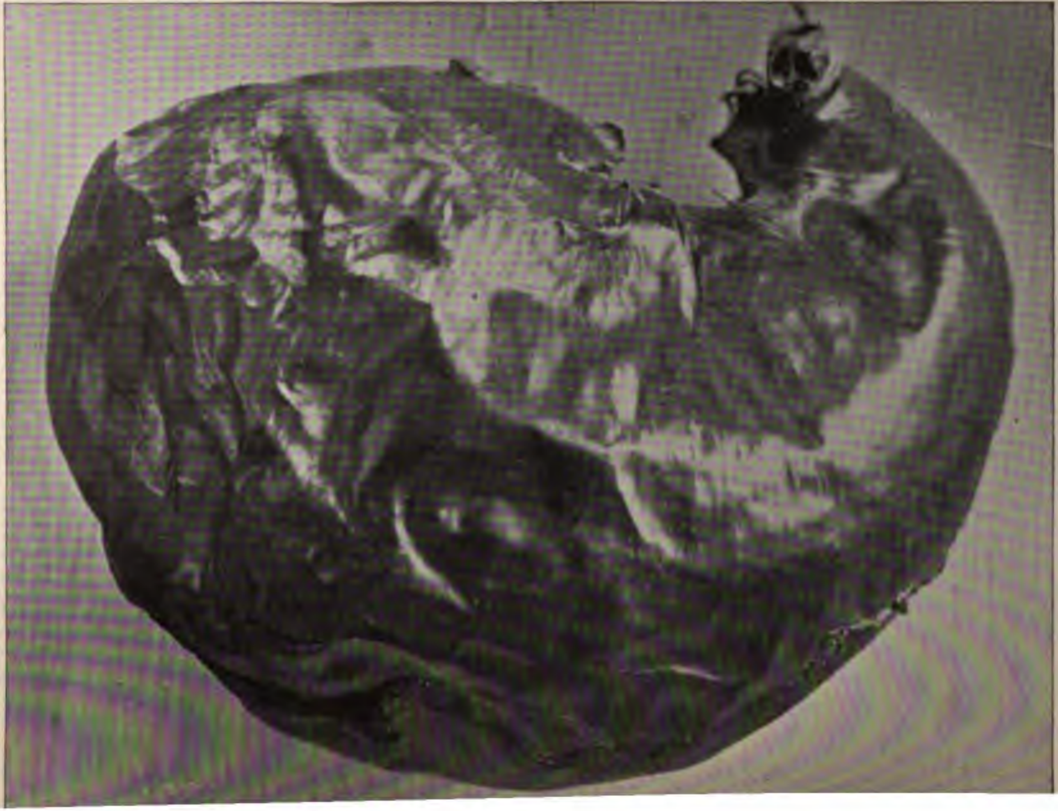
FIG. 29.



Stomach of infant 7 months old, natural size. Weight, 5500 grammes. Gastric capacity, 220 c.c

Dry preparation (page 89).

FIG. 30.



This next stomach (Fig. 29) came from an infant seven months old and weighing 5500 grammes (12 pounds). Its capacity is 220 c.c. (7⅓ ounces).

This stomach (Fig. 30) was taken from an infant nineteen months old and weighing 6270 grammes (13¾ pounds). Its capacity is about 300 c.c. (10 ounces).

I wish you to understand that I do not make any definite deductions from the last four cases. The stomach at nineteen months is especially unreliable as to its capacity. It was very distensible, and could easily by the mere weight of the water be made to hold 450 to 600 grammes (15 to 20 ounces). It gives, however, a very fair idea as to how the stomach looks at this age. This seven-months' stomach (Fig. 28) in its capacity corresponds to the weight, which is that of an infant of four months, while this other seven-months' stomach (Fig. 29) seems in its capacity to correspond to the infant's age rather than to its weight, which is that of the average infant at four months.

I have now treated this question of gastric capacity by the more exact methods of weighing and careful calculation, and also by the usually inexact method of direct measurement. Both methods, however, result in a general uniformity of figures and give us very fair data by which we can be guided when we come to the question of infant feeding.

It will be seen that the general principle of activity of growth is well represented in these figures.

The gastric capacity, however, in the third, fourth, and fifth months may appear rather small, and considerable differences will arise in the measurements of different observers. This, however, only impresses on us that we have not yet solved the problem of gastric capacity by any system of measurement. When all observers have agreed to make use of a mathematically precise and constant pressure in measuring the stomach, we may possibly arrive at more uniform results. Even then the degree of elasticity will be found to differ so greatly in the individual stomach that most diverse measurements will result.

There is no doubt that the value of these calculations lies in making us recognize evident changes in the activity of growth at certain periods; in making us allow that great differences arise irrespective of age and weight; in impressing us with the fact that the gastric capacity has been over- rather than under-estimated, and in enunciating that more exact clinical observations should be employed to reinforce our anatomical and physiological data.

During the last two and a half years I have been enabled through the aid of a milk laboratory to adapt exactly to the apparent needs of the infants under my care, as well as to their age and weight, the amounts of food on which they have seemed to thrive. It will be interesting and instructive to compare the following table with the figures and calculations which I have just shown to you, and thus see if my practical clinical results have corresponded to my experimental deductions.

The following figures represent the average amounts of food taken at different periods during their first year by three hundred and forty-one infa They were all well and strong, of average weight, and all were thriving steadily gaining during the year. They received only stated amounts food carefully ordered by prescription at the Milk Laboratory, and w watched with the greatest care to see when they evidently were hungry eno to have the total amount of their food increased. Of course the opportunity exact work is almost unlimited where one has a milk laboratory at his c mand, and it has therefore seemed to me that this method of determining gastric capacity is an unusually good one, and one which has never thoroug been carried out before. Before showing you the table of the general avera I will pick out one case to explain the significance of the general figures.

This infant (Case 21) was fed with the greatest care both as to quality and as to the quantity of its food. The following figures repre the amount of food given at each meal from birth to ten months:

TABLE 16.

Amounts of Food in an Especial Case.

Age.	Cubic Centimetres.	Ounces.	Age.	Cubic Centimetres.	Ou
Birth	30	1	6 months	150	
4 weeks	45	1½	7 months	150	
8 weeks	60	2	8 months	150	
12 weeks	75	2½	9 months	195	
16 weeks	90	3	10 months	240	
20 weeks	132	4½			

This case shows the necessity for frequent and great increase of the t amount in the first four or five months, the comparative quiescence of gro in the sixth, seventh, and eighth months, and the increase again in the ni and tenth months. It does not, however, correspond so closely to my vious results as does this table, where averages taken from the three hund and forty-one cases already referred to are given.

TABLE 17.

Three Hundred and Forty-One Infants fed at the Milk Laboratory.

Age.	Number of Cases for each Age.	Average Amount of Food each Feeding. C.c.	Ounces.
Birth	45	29.4	0.98
4 weeks	76	70.5	2.35
8 weeks	84	96.6	3.22
12 weeks	97	118.8	3.96
16 weeks	87	137.0	4.57
20 weeks	86	158.4	5.28
6 months	73	171.3	5.71
7 months	56	185.4	6.18
8 months	54	208.5	6.95
9 months	45	226.2	7.54
10 months	33	238.8	7.89
11 months	28	242.0	8.07

In this table the same infant has of course been recorded a number of times at ferent ages.

The whole question of gastric capacity is so closely connected with the subject of infant feeding that I shall leave it for the present, and speak of it more in detail later, when it will be seen to be of infinite importance in our attempts to regulate the substitute feeding of infants.

INTESTINE.—**Small Intestine.**—During the first month after birth, it may be reckoned that the small intestine will grow about two feet (about sixty-one centimetres), and a like rate of growth may usually be recorded at the end of the second month of extra-uterine life; but after that period its development proceeds in a most irregular manner. Thus, in a child of one year the small intestine measured eighteen feet (about five hundred and forty-nine centimetres), while in another, aged two years, the length was only thirteen feet eight inches (four hundred and seventeen centimetres). Again, in a child aged six years the small intestine was no less than twenty-one feet (about six hundred and forty and five-tenths centimetres) in length, while in another child, eleven years of age, its length was fourteen feet (about four hundred and twenty-seven centimetres).

I agree with Mr. Treves that the great variations which appear so early in the length of the small intestine bear no relation to the growth of the child. They probably depend on the diet. Not only the quantity but the quality of the food is an important factor in the growth of the intestine. The amount of residue, also, and the more or less irritating qualities of the food, must all have their effect.

As to the internal structure of the small intestine below the duodenum I can only say that I confirm the view now generally accepted, that Peyer's patches are found very early. I have seen them at three days and again at thirteen days.

In another case, sixteen months old, Peyer's patches were found, and one of them was five inches long.

Large Intestine.—Treves has also observed that up to three or even four months the length remains the same, but that nevertheless a remarkable change occurs. This is that the large intestine grows at the expense of the sigmoid flexure, which at birth is nearly one-half of the large intestine, while at four months it has assumed about its permanent proportion. Treves found the large intestine to measure at one year two feet and six inches (about seventy-six centimetres); at six years about three feet (about ninety-one and five-tenths centimetres); and at thirteen years about three feet and six inches (about one hundred and seven centimetres). I find among my notes the following measurements of the intestine.

TABLE 18. (Dwight.)

Sex.	Age.	Small Intestine.	Large Intestine.
Girl	13 days.	292.9 cm.	48.5 cm.
Girl	10 months.	670.0 cm.	78.0 cm.
Boy	10 months.	435.0 cm.	90.0 cm.
Girl	3 years.	490.0 cm.	84.0 cm.

Cæcum and Ascending Colon.—In about thirty-five observations on dren under four years of age, most of them new-born infants, the ca was found in about thirty cases to range from the right lumbar region t lower part of the iliac fossa. It was very frequently found at the jun of the rather vague lumbar and iliac regions. More or less would us be found between two parallel horizontal lines, one at the level of the hi point of the crest of the ilium and the other at its anterior superior spi process. In five cases the cæcum was either in the right iliac fossa or the true pelvis, the fact being that it was so free as to have no fixed l tation. It is comparatively recently that the truth has been recogniz America, England, and France that normally the cæcum is at every completely invested by the peritoneum, and that the idea that a large of the posterior surface rests on areolar tissue without any interve serous membrane is baseless, except in rare instances.

In young children the ascending colon differs in some respects from of the adult. Owing to the high position of the cæcum, to say nothin the relatively greater size of the liver, it is very short. There is no q tion that the ascending colon much more frequently has a mesentery in the adult, and also that a relatively larger portion of the part al the cæcum is also invested with peritoneum so as to be absolutely More than once Dwight has seen the cæcum and a large part of the asc ing colon in this condition. As to the question of how frequently mor less of the back of the cæcum may lack its peritoneal covering, in w case of course it is bound down to the parts beneath it, Dwight's obse tions are rather remarkable. Treves in his Hunterian Lectures stated in one hundred observations he never found the posterior peritoneal cove wanting. Tuffier examined one hundred and twenty subjects, adults, c dren, and fœtuses, and found the posterior surface uncovered in nine, of whom were old people. I have kept no systematic record of Profe Dwight's observations on adults, but have the following report of thi seven young children. In thirty-three the cæcum was completely inve with peritoneum. In four children, all new-born or only a few days the whole or a large part of the back of the cæcum was without periton

Considering that this condition is much more likely to occur in adult, and that, so far as we know, no one else has observed it in the inf I am inclined to think that Dwight's large number of cases (four out thirty-seven) must be considered an accident, such as is liable to hap where a series of observations is small. Professor Dwight believes that cæcum of the infant and that of the young child are much more mova than that of the adult, and are also usually situated higher.

Vermiform Appendix.—The length and direction of the vermiform pendix are very variable. I have found it six and a half centimetres (t and five-eighths inches) long in a girl of thirteen days, five and three-tent centimetres (two and one-eighth inches) in one of three years, eight cen metres (three and one-quarter inches) in one of ten months, and seven a a half centimetres (three inches) in a girl eleven weeks old. The vermiform appendix in the first of these cases was so peculiarly placed as to deserve a few words of description. Only a small part was free, the rest being held by a small mesentery to the cæcum and the ascending colon. It arose from the posterior side of the cæcum, and ran backward to above the crest of the ilium, where it entered a little peritoneal pouch in the rear wall of the abdomen, and then, turning on itself, ran forward again. The entrance to the pouch was guarded below by a semilunar fold of peritoneum, with its cavity looking upward. It would appear from Treves's researches that the fœtal shape of the cæcum is that of a pouch hanging down from the point of junction of the small and the large intestine and continued into the appendix, which grows symmetrically from the middle. Later, however, an irregular growth of one side of the cæcum generally leaves the origin of the appendix near the end of the ileum. Dwight has found that this condition usually prevails in the child. The position and direction of the appendix are most uncertain. It is, however, I believe, as a rule, on the posterior side of the cæcum. Its little mesentery passes to its beginning from the cæcum and is only exceptionally attached to the walls of the abdomen or pelvis.

The importance of the lymphatic glands about the cæcum as possible starting-points of inflammation is very great. Tuffier states that the lymphatics of the front of the cæcum follow the anterior ileo-cæcal artery to empty into two glands which he has found constantly in the superior ileo-cæcal fold, and which are very distinct in the child. The posterior glands are also found constantly on the posterior and inner wall of the cæcum itself beneath the peritoneum. They usually form a group of from three to six.

Sigmoid Flexure.—Dwight's observations on the sigmoid flexure in infancy show much diversity. In some cases it is obviously very long, in others apparently of about the adult proportions. As he has made accurate measurements in but few cases, I hesitate to make precise statements, but very frequently even at birth there was no evident departure from the normal adult proportions. A difficulty in this inquiry, which, however, is in itself an important point in anatomy, is to decide where the descending colon ends and the sigmoid flexure begins. Thus, in a girl of ten months the first impression was that the latter was not relatively longer than in the adult; but it was found later that what must be called the descending colon proper was very short, ending above the top of the crest of the ilium. This portion, a little over an inch in length, had a retro-peritoneal surface. The mesentery then began, and was attached obliquely across the psoas down to the front of the cæcum, where it became the meso-rectum. Thus the greater part of the descending colon formed one loop or series of folds with the sigmoid flexure; and this is by no means the only time that Professor Dwight has pointed out this arrangement. This loop which I have just described was also found to be very movable. The greatest breadth of the mesentery was four and eight-tenths centimetres (about two inches). In another child of the same age it was seven centimetres (about two and seven-eighths inches). I am inclined to think that even in infants, in whom the sigmoid flexure does not, as a rule, seem larg often has a relatively broad mesentery, allowing free displacement. In t children of three years the sigmoid flexure did not seem to exceed the ac proportion.

Descending Colon.—As is well known, the descending colon usually no mesentery, but still one is often found. Lesshaft, in his observati made on subjects of many different ages, found it once in six times. Dwig in rather more than twenty infants, found a mesentery to the descend colon in about half the cases. It is remarkable that Lesshaft found a m entery less often in young subjects than in others. I find that a great p of the large intestines in infants is less fixed than in adults. I unfor nately, however, have not had at my command sufficient material to ena me to say when the mature condition is reached.

TEMPERATURE.—The temperature of the infant at term, althou varying within a slight limit, is usually slightly raised. Very soon, h ever, as would be expected from the tax which is immediately made on vitality by so many new surroundings, the temperature falls rather bel the normal adult standard. In about a week the normal infant has rec ered its equilibrium, and, if its nutriment has also been properly adapted its digestive peculiarities, it usually presents the average normal adult te perature, 36.8° C. (98.2° F.).

TABLE 19.

Temperature of Infant at Term.

At birth	37.2° C. (99° F.).
Within an hour	36.1°–35.5° C. (97°–96° F.).
In about a week	36.8° C. (98.2° F.).

These figures are the average of a large number, and are subject to gr variations, as is seen on comparing them with a number of observati undertaken at my request by Dr. C. W. Townsend at the Boston Lying Hospital:

TABLE 20.

Townsend's Temperature Observations.

Age.	Temperature.	Age.	Temperature.
1 day	37.2° C. (99° F.).	9 days	37.4° C. (99.4° F
2 days	37.3° C. (99.2° F.).	9 days	37.1° C. (98.8° F
5 days	36.6° C. (98° F.).	9 days	36.9° C. (98.4° F
5 days	37.5° C. (99.5° F.).	10 days	37.1° C. (98.8° F
6 days	37.3° C. (99.1° F.).	13 days	37.2° C. (99° F.)
7 days	37.5° C. (99.5° F.).	13 days	37.3° C. (99.2° F
7 days	37.2° C. (99° F.).	16 days	37.3° C. (99.2° F
7 days	37° C. (98.5° F.).	20 days	37.3° C. (99.2° F

PULSE.—The pulse in uterine life is, as a rule, somewhat higher in gi than in boys, the former being about 130 to 140, and the latter 120 to 13 Anything over 130 points towards the female sex, but these figures as a mea of distinguishing the sexes before birth are not to be relied upon. At birth the pulse soon falls somewhat, and, as I have already told you, may be quite irregular. This, as a rule, is merely what we should expect would be the result of the sudden and great change which has taken place in the circulatory mechanism, and of the additional force which the heart is called upon to supply when it becomes the central station from which the blood is propelled. The lungs also are scarcely ready to perform at once their function, and are often somewhat more of an obstruction than an aid to the blood-current. The pulse in early life, especially during the first year, varies very much, but, as a rule, allowing that the girl's pulse is usually rather quicker than the boy's, the following table represents pretty well what you may expect in males.

TABLE 21.

Pulse-Rate for Males.

Age.	Pulse-Beats per Minute.
Early weeks	120 to 140
Until 2d year	110
2 to 3 years	100
5 to 8 years	90

From the eighth year up to puberty the pulse gradually acquires the adult rate. The pulse in children varies greatly under the many nervous influences which are continually affecting it in early life.

Dr. Townsend has also made a record of the pulses taken in the same infants whose temperatures were recorded in Table 20. They, as you see, do not especially correspond with the general averages of Table 21, but are what you may expect in the cases which you happen to see at random.

Clinically I have never arrived at very satisfactory results in my observations on the pulse in infancy. If, however, you care to investigate this subject more thoroughly, I will refer you to the excellent work done on the pulse by Keating and Edwards.

TABLE 22.

Townsend's Pulse Observations.

Age.	Quiet.	Crying.
1 day	130	158
2 days	120	156
5 days	152	164
5 days		160
6 days		152
7 days	120	154
7 days		160
7 days		152
9 days	148	
9 days	160	180
9 days	156	
10 days	152	
13 days	136	
13 days		168
16 days	168	172
20 days		168

RESPIRATION.—The respiration, although quicker in early life in adults and corresponding somewhat to the pulse, assumes the equilib of a later period of development much earlier than is found to be the with the pulse. It varies with changes of temperature and with exciten and has its rhythm much more easily affected by diseased conditions tha later life. This table represents fairly well what you will usually meet on counting the respirations when a child is quiet.

TABLE 23.

Respirations.

Age.	Respirations per Mint
At birth	45
Until the 3d year	15 to 40
3 to 5 years	25

I should now like you to notice closely this infant (Case 22) whic lying quietly in the nurse's lap.

It is a male, eight months old, and healthy. In the first place, you see that its ty respiration is decidedly abdominal. Counting the respirations by the rise and fall o ensiform cartilage, which stands out quite distinctly in this case, I find that they vary 60 to 70 in the minute. They are also, as you see, quite irregular, and by making w pencil an upward stroke for every inspiration, a downward stroke for every expir and a horizontal line for every pause, you will find somewhat the same lack of rhythm appears in the infant at term, which I described to you in this way in a previous le (Lecture II., page 48), and also the rhythm corresponding to that of the infant nine m old which I have already shown to you (Case 17, page 72).

Dr. Townsend has also observed for me the respiration of four case the Lying-in Hospital, with the following results:

TABLE 24.

1. Age, 1 hour	Respirations,	48 to 56.	(Awake.)
2. Age, 2 days	"	30 to 52.	(Asleep.)
3. Age, 3 days	"	24, 32, 44.	(Asleep.)
4. Age, 6 days	"	28 to 40.	(Crying.)

The respiration in all these cases was very irregular, and both abd nal and thoracic in type. In the baby two days old the respiration chiefly abdominal.

HEIGHT.—The average height of the male infant at term, I h already stated, is, according to a large number of measurements made Quetelet, Vierordt, and others, about 49.5 cm. (19⅘ inches). These fig correspond very closely to those which I have met with in quite a numbe infants whom I have myself carefully measured. Insufficient nourishn and improper food, especially as represented in rhachitic children, s to retard the growth, while, on the contrary, the various fevers seem increase the activity of growth in length, while decreasing the total wei In the first three or four months the growth is proportionally rapid to t in the latter part of the first year. In like manner the activity is gre in the first month than in the second, and in the second than in the th becoming still less in the fourth, fifth, and sixth months.

The average increase for the first month is about 4.5 cm. (1¾ in.).
" " " " " second month is about 3.0 cm. (1½ in.).
" " " " " third to the fifteenth month is about 1 to 1.5 cm. (½ to ¾ in.).
" " " " " first year is about 20 cm. (8 in.).
" " " " " second year is about 9 cm. (3½ in.).
" " " " " third year is about 7.4 cm. (3 in.).
" " " " " fourth and fifth years is about 6.4 cm. (2⅝ in.).
" " " " " fifth to the fourteenth year is about 6 cm. (2⅜ in.).

The height is about doubled in the first six years, and at fourteen years the final height has usually been attained to within about one-twelfth. The height at different ages will be shown in comparison with the weight in Table 27 (page 104), when we are considering the question of weight. The growth in height seems to be most active in the spring.

WEIGHT.—We now come to the subject of weight in children, the study of which has deservedly attracted considerable interest and scientific research. In quite a number of cases it has been found that the careful and systematic weighing of infants gives us warning of the approach of disease some days before any other symptoms are evident. This point was very clearly illustrated in a case which was under my care at the Infants' Hospital, and to which I shall refer in a later lecture more in detail (Case 279, page 627). This infant entered the hospital to have its food regulated. It was apparently perfectly well, but after a few days the daily weighing showed that it was losing. This loss of weight continued to be the only perceptible symptom for a number of days, when it manifested certain nervous phenomena and died a few days later of *cerebral thrombosis*. We sometimes notice a loss in weight preceding a chronic nutritive disturbance by several weeks, and if the coming disease is an acute one, or is of unusual severity, the loss is often sudden and great. You will therefore readily understand that the careful and systematic weighing of children may be of considerable value in preventive medicine. Thus, if we have noticed that a child has without perceptible cause lost weight, we can, by guarding it from an exposure which in health would not be too great, prevent it from having complications such as of digestion or from cold, and render the coming disease milder in its type and more readily dealt with. In a paper on the Relation between Growth and Disease, by Professor H. P. Bowditch, these changes in weight are especially dwelt upon, and it is apparently shown that this method of determining the onset of the disease is more useful in chronic than in acute diseases, though even in the latter class it is not impossible that the warning may be given in time to be of use, and to merit the term of "danger signal" which has been given to it by Dr. Percy Bolton. Bowditch shows in this interesting table (Table 25) the rate of growth of a girl between two and three years old, and the relation between growth and disease. The figures represent the absolute weight of the child obtained by weighing in the ordinary manner, and then deducting the weight of the clothes.

TABLE 25.

Date.	Age, in Weeks.	Weight.		
		Kilo.	Lbs.	
1880.				
September 19 . . .	107	11.40	25.08	
October 3.	109	11.40	25.08	
November 7 . . .	114	11.78	25.91	
December 5	118	12.25	26.95	
December 12 . . .	119	12.28	27.01	
December 26 . . .	121	11.90	26.18	
1881.				
January 2	122	12.15	26.73	
January 28	125	11.80	25.96	
January 30	126	11.65	25.63	
February 6	127	11.55	25.41	Enlarged cervical glands noticed F
February 13 . . .	128	11.55	25.41	Clay-colored dejections February 1
February 20 . . .	129	11.95	26.29	
February 27 . . .	130	11.75	25.85	
March 6	131	11.94	26.26	
March 13.	132	12.15	26.73	
March 20.	133	12 20	26.84	
March 27.	134	12.41	27.30	
April 3	135	11.91	26.20	Attack of measles beginning April
April 10	136	11.71	25.76	
April 17	137	11.98	26.35	
April 24	138	12.00	26.40	
May 1	139	12.03	26.47	
May 8	140	12.01	26.42	
May 15	141	12.34	27.14	
May 22	142	12.15	26 73	Cold in the head beginning about
May 29	143	12.09	26.60	

An examination of this table shows that the child, having grown during the autumn, suddenly, and without any manifest cause, bega weight about the middle of December. This loss of weight was irr progressive until February 6, when an enlargement of the cervical ly glands was noted, followed a week later by clay-colored dejections. symptoms yielded to appropriate treatment, and the child again gaine rapidly until March 27, when a sudden loss of weight occurred, foll an attack of measles. A subsequent loss of weight in May seems been associated with a rather severe cold in the head. We have he a case in which a disorder of nutrition manifested itself by enlarge and by clay-colored discharges, but in which these symptoms were for several weeks by a progressive loss of weight. It seems not u able to suppose that this loss of weight was the first symptom of a ance which afterwards manifested itself by more unequivocal signs in the case of the acute attack of measles it will be noticed that th weight preceded by at least a week the actual eruption of the diseas must not, however, suppose that loss of weight in a growing child is instance a precursor of actual disease. The weight of a healthy liable to oscillation within limits which have not been accurately det but it may sometimes amount to ten or fifteen per cent. in a week. (

ose in weight and regain their loss in a wonderful manner, so easily are they ffected by even slight physical disturbances, and so great are their recuperative powers. The weight of boys, as a rule, is somewhat greater than hat of girls at birth, and remains greater up to the age of puberty, when he girl rapidly overtakes the boy, surpasses him, and becomes a developed woman very soon, while the boy does not become a man until some years fter puberty. This fact you will see exemplified in the table (Table 27, page 104) which I shall presently show you, and which shows that the girls have surpassed the boys in their height at the eleventh year, and in their weight at the twelfth year, when they are found to be taller and heavier than the boys, as is the case also in the thirteenth and the fourteenth year.

The systematic and frequent weighing of infants during the first year of their lives I consider to be of great importance, and far more useful as a means for determining their nutritive condition than any other one method which we know of. For many years I have had the infants at the Infants' Hospital weighed every day as regularly as they are fed, and a glance at the olumn containing their weights in the various weeks and months gives information as to their general health, and serves as a guide to the changes which it may be necessary to make in their food. The information gained in this way is far beyond what the most careful physical examination could disclose. The weight is, in fact, an index of the nutritive processes to such an extent that it is representative of the child's well-being, while the height gives us information rather as to its cellular activity. I have already stated that the normal average weight of quite a number of infants at term is for males 3250 grammes (7¼ pounds), and for females 3150 grammes (7 pounds), and I have also stated that many individual cases occurred where the weight was either greater or less than these figures, and yet the infant was healthy. The increase in weight is in direct proportion to the original weight, and if the original weight is small the gain is usually correspondingly small. This, however, is only a general rule, for at times I meet with infants of light weight whose gains are remarkably large, and often surpass those of infants with a heavier initial birth weight. During the first three or four days of life there is usually a loss in weight, and the original weight is in a large number of cases regained only in the second week. If it is not regained by the third week, we should consider that it is a warning that the nutrition of the infant is at fault, and that especial measures should be taken to increase its vitality. This initial loss of weight is usually designated as *physiological*. We must not, however, be misled by this term, or place too much confidence in it, for, as a rule, this initial loss, which often amounts to from 270 to 300 grammes (9 to 10 ounces) can be accounted for only partially by natural physiological causes. The additional loss is evidently pathological, and is to be so regarded, in order that we should endeavor to obviate it, and thus prevent imposing an additional tax on the infant's vitality at a time when any tax whatever should be regarded as serious. Dr. Townsend has made some interesting investigations on this loss of weight at the B Lying-in Hospital, which show that the infants of primiparæ lose abo grammes (1½ ounces) more than those of multiparæ; also, deductin grammes (1½ ounces) as the average loss from removal of the vernix ca the meconium still remaining, that the loss in weight is reduced to grammes (8½ ounces) in the infants of primiparæ, and to 222 gramme ounces) in those of multiparæ. The whole loss should include the mecon which is computed to weigh about 60 to 70 grammes (2 to 2½ ounces), so a loss of from 90 to 150 grammes (3 to 5 ounces), which includes als urine, on the first day, can, in a very general way, be admitted to be p physiological. Dr. Townsend's figures also show that although the in of primiparæ lose more and are slower to recover the loss than are tho multiparæ, yet after the second week they overtake and keep pace wit latter. The whole question is simply one of nutrition, it being well k that the milk of primiparæ is somewhat longer in acquiring its equilib than that of multiparæ, but that finally it is equally nutritious. It wa found that the presence of the colostrum corpuscles in the milk had thing to do with the loss or with the failure to gain. Where the colos persisted the infants lost more than when it speedily disappeared. The trum should normally disappear in the first week. Where its prese prolonged into the third week, the infants do not thrive. Townsend three cases at the hospital illustrating this point: all the mothers se healthy and had plenty of milk.

(1) Multipara—no colostrum on third day,—infant's loss 8 ounces.
(2) " —colostrum until ninth day,—infant's loss 16 ounces.
(3) Primipara—colostrum until thirteenth day,—infant's loss 14 ounces.

The average loss in five infants of multiparæ where the colostrum absent by the fifth or sixth day was 10 ounces.

I am indebted to Evetsky and Foster for much valuable informatio this subject, and quote freely from their writings. The whole nervous tem of the young child is much more active and excitable than that o adult. The brain, for instance, besides being fifteen times as large pr tionately in the infant as in the adult, increases with much greater rap up to the age of seven years than at any other period. In connection, p bly, with the constructive labors of the growing tissues is the activity o lymphatic system. The absorption of oxygen is said to be relatively rapid than the production of carbonic acid,—that is, there is a conti accumulation of capital in the form of oxygen-holding compounds. food represents so much potential energy, but it must be converted into t before the energy can become vital, and in such conversion a large am of molecular energy must be expended. The metabolic activity is pronounced in the infant than in the adult, and is expended not so muc the energy required in the external world as for the rapidly increasing of tissue. Another reason for the presence of more active metabolis the infant than in the adult is the necessity of rapid molecular interchange to keep up the temperature. The infant having the smaller body, and yet the relatively larger surface (the extent of skin thus being proportionately greater), it loses more heat proportionately than does the adult, and thus suffers more easily from changes of temperature.

Disturbances of the nutritive processes from these conditions very easily arise, and the process of assimilation is much more important than in adult life, for the child's activity implies a greater consumption of nutriment in the form of food or tissue. The child's equilibrium is thus much more easily disturbed than the adult's, and this creates a greater susceptibility to disease and less power to resist external influences. This is well exemplified by the rule that the younger the individual the greater the mortality. There are three times as many deaths in the first half of the first year as in the second half, and a large proportion of those dying in the first half year die in the first month. Of those dying in the first month, death occurs in a large proportion in the first week. A considerable number of the deaths which occur in the early weeks of life, especially in the first week, are from asthenia. These facts are very significant in connection with the child's loss of weight in the early days of life over that which we have just described as being physiological. Lack of sufficient nourishment and an unstable equilibrium are the factors in the problem which represent this early loss of weight. These conditions are enhanced by the state of the mother, who, exhausted by the process of labor, is not able to supply a food for her infant which is adapted to its sensitive and incompletely developed digestive function.

In addition to these manifest causes for loss of weight, we must consider that the new-born infant is much more susceptible to external impressions than when after the first weeks its various functions have become adapted to their new surroundings.

The whole system is stimulated to greater activity of tissue interchange not only by the sudden change of temperature to which the skin is exposed, but also by the change from darkness to light, and from silence to a greater or less degree of sound. This transient early period of life, therefore, is marked by a superactive metabolism, insufficient nourishment, and resulting asthenic conditions which are analogous to starvation. This is represented as a whole by a loss of weight evidently of a pathological character, in addition to that which I have described as physiological. You will, therefore, now understand with what care the newly-born infant should be protected from too great changes of temperature, too much light, and too much noise. The analogy of this statement is found in the sensitive organization and habits of the lower animals. In this way only can the digestive function be made to correspond to such an extent, in the early days of life, to the work which is required of it, as to keep the loss of weight within the physiological limit. Starvation, as is well known, proves fatal primarily not from the amount of food furnished being too little for the processes of disintegration, but from exhaustion of the nervous system. The endura of the starvation is in proportion to the capability of resistance of t nervous tissue. This nervous tissue is so highly sensitive and has su great functional activity in the infant, proportionately to the adult, that needs much more nourishment, and succumbs much more quickly to depri tion from nourishment. Young animals die in a very much shorter ti when deprived of food than do older ones from this cause. It is not s prising, therefore, that when the early period of life is represented only hours and days, the various disturbances which would be of minor con quence at a later period of existence should have a decidedly pathologi effect and produce a marked loss in weight beyond the natural phys logical loss. The following case, taken from my records of this cl exemplifies the practical bearing of what I have just said.

Case 23.—A male infant was born December 16 at term. It was healthy and vi ous, and gave no evidence of organic disease. The mother, a multipara, strong and heal was twenty-eight years of age. Her other children were living and healthy. On the t day, December 19, the infant had a slight attack of icterus neonatorum, which disappe in twenty-four hours. On the fifth day, December 21, the weather was very cold and bl but the infant was taken to church and christened. The church was warm and the in reasonably well protected from cold, but there were a large number of people present, an unusual amount of noise. The infant, on being taken home, immediately began to s symptoms of asthenia, and on the following day was found to be cyanotic and breat rapidly, with a subnormal temperature and no apparent organic disease. It died ir afternoon. The asthenia seemed to be produced by too early exposure to change of perature, light, and sound.

As a rule, the average daily gain in the first two months should be below twenty grammes (two-thirds of an ounce). I have found at Infants' Hospital that if the gain is less than this the infant, as a rul being badly nourished, is sick, or is going to be sick. There are, of cou exceptions to this rule, and I would here also call your attention to fact that observations of weight including only that of two or three days very misleading, and that it is the week's weight which gives us the fa idea of loss or gain. Thus, I frequently find infants showing a daily of only five or ten grammes (one-sixth or one-third ounce), or even lo fifteen or thirty grammes (one-half or one ounce) on one day, and then g ing one hundred to one hundred and fifty grammes (three and one-t to five ounces) on the next day. From this you will readily unders that we should obtain from one day's observation too low and on the day too high an estimate of the nutrition. By the end of the week, h ever, the weights usually equalize each other, and we have fairly co figures to guide us. This table (Table 26) shows about what woul expected of the average infant as to weight during the first year. Girl a rule, gain less than boys, but this is only if they are of lighter wei The heavy girls make the same large gains as the heavy boys, but, as a their initial weight is smaller than that of the boys, and they therefore n smaller gains.

TABLE 26.

General Figures of Weight.

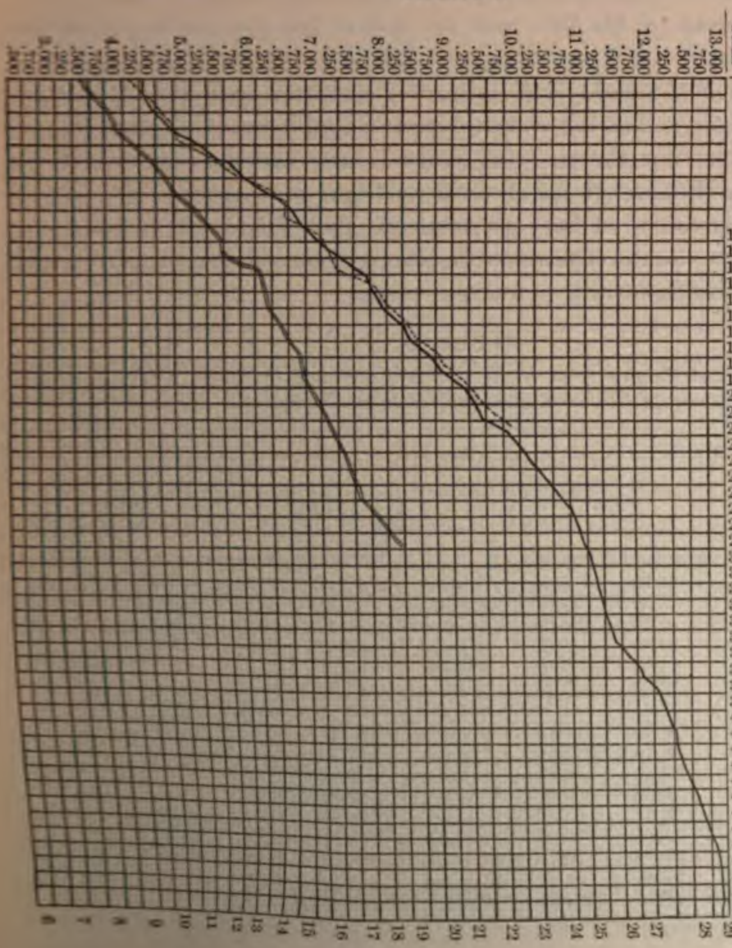
Age.	Weight. Grammes.	Weight. Pounds.	Average Gain per Day. Grammes.	Average Gain per Day. Ounces.
At birth	3000 to 4000	6.6 to 8.8		
From birth to 5 months			20 to 30	⅔ to 1
From 5 months to 12 months			10 to 20	⅓ to ⅔

Age.	Weight. Grammes.	Weight. Pounds.
At 1 year	9,500	20.90
At 7 years	19,000	41.80
At 14 years	38,000	83.60

[The above figures are on a basis of 3500 grammes (7.7 pounds) at birth, and of a gain of 30 grammes per day for the first four months and 10 grammes per day for the last eight months of the first year.]

Useful figures to remember are that the initial weight is doubled at five months and trebled at fifteen months; also that the weight at one year is doubled at seven years, and that this weight is again doubled at fourteen

CHART 3.



Name, ...
Date of Birth, June 21.
Initial Weight, 4650.

Weeks.	Actual Wt.	Date of W'g.
1	4,500	June 27.
2	4,612	July 4.
3	4,916	" 11.
4	5,332	" 18.
5	5,684	" 25.
6	6,004	Aug. 1.
7	6,292	" 8.
8	6,644	" 15.
9	6,852	" 22.
10	7,172	" 29.
11	7,476	Sept. 5.
12	7,802	" 12.
13	7,994	" 19.
14	8,170	" 26.
15	8,362	Oct. 3.
16	8,586	" 10.
17	8,912	" 17.
18	9,136	" 24.
19	9,376	" 31.
20		
21	9,968	Nov. 14.
22	10,112	" 21.
23		
24		
25		
26	10,912	Dec. 20.
27		
28		
29		
30		
31		
32		
33		
34		
35	11,680	Feb. 20.
36	11,904	" 27.
37	12,032	March 5.
38		
39		
40		
41		
42	12,544	April 9.
43		
44		
45	12,640	April 30.
46		
47		
48		
49		
50		
51	13,104	June 10.
52	13,376	" 20.

years. There are, of course, both gains and losses in weight du year, the weight acting as an index of the disturbances which ari rule, what may be called the line of nutrition rises from the initi in the first week, week by week, up to the fifty-second week. A increase is, however, exceptional, on account of the many disturban as from food, the dental periods, weaning, improper hygienic care, contraction of disease.

Instances of continual weekly gains during the first year have ally come under my notice in both hospital and private practice, chart on the preceding page (Chart 3) gives the exact weights of a male infant fed by a wet-nurse for over a year, and will serve as an of the ideal line of nutrition.

The infant was gaining so regularly that the weighing was on several weeks, which fact is unfortunate, as the weights would have shown the same uniform gain. A weekly gain is also show same chart of a male and a female infant, brother and sister, nu their mother. The double line represents the boy's weights in twenty-nine weeks of his life; and the dotted line the girl's we twenty-one weeks.

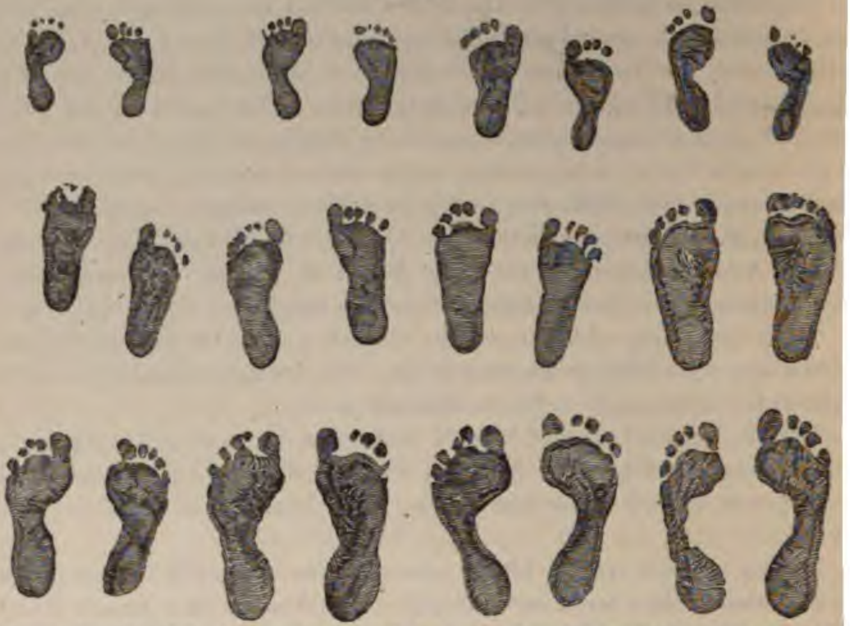
The question of weight is so intimately connected with that of that I shall reserve showing you the charts recording the daily we the infants which for the past ten years I have had an opportu studying at the Infants' Hospital, until we begin to investigate the principles of nutrition. I have prepared this table (Table 27) to sl how at a glance you can determine the average normal height and of boys and girls from birth to fourteen years.

TABLE 27.

Average Heights and Weights from Birth to Five Years, and of Boston School Girls, irrespective of Nationality, from Five to Fourteen Years.

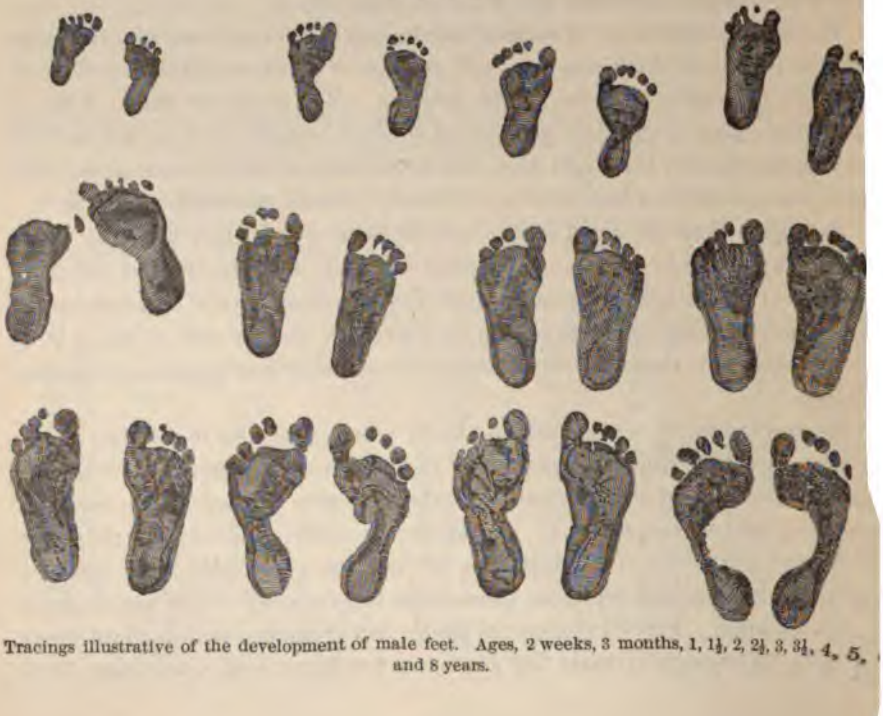
Boys.				Age.	Girls.		
Height.		Weight.			Height.		Weig
Centimetres.	Inches.	Kilogrammes.	Pounds.		Centimetres.	Inches.	Kilogramm
49.37	19.75	3.25	7.15	Birth.	48.12	19.25	3.15
61.87	24.75	6.50	14.30	5 months.	59.12	23.25	6.80
73.82	29.53	9.54	20.98	1 year.	74.17	29.67	9.00
84.55	33.82	13.80	30.36	2 years.	82.35	32.94	13.81
92.65	37.06	15.90	34.98	3 years.	90.77	36.31	15.07
98.27	39.31	17.27	37 99	4 years.	97.00	38.80	16.5
103.92	41.57	18.64	41.00	5 years.	103.22	41.29	17.9
109.37	43.75	20.49	45.07	6 years.	108.37	43.85	19.
114.35	45.74	22.26	48.97	7 years.	113.80	45.52	21.
119.40	47.76	24.46	53.81	8 years.	118.95	47.58	23
124.22	49 69	26.87	59.00	9 years.	123.42	49.37	2
129.20	51.68	29.62	65.16	10 years.	128.35	51.34	2
133.32	53.88	31.84	70.04	11 years.	133.55	53.42	
137.77	55.11	34.89	76.75	12 years.	139.70	55.88	
143 02	57.21	38.49	84.67	13 years.	145.40	58.16	
149.70	59.88	42.95	94.49	14 years.	149.85	59.94	

FIG. 31.



Tracings illustrative of the development of female feet. Ages, 1 week, 3 months, 1, 1½, 2, 2½, 3, 3½, 4, 5, and 8 years.

FIG. 32.



Tracings illustrative of the development of male feet. Ages, 2 weeks, 3 months, 1, 1½, 2, 2½, 3, 3½, 4, 5, and 8 years.

BONE MARROW.—In a previous lecture I spoke of the red marrow as characteristic of the bones in early life. The marrow of the bones at a later period of life changes from red to yellow. This change of red marrow to yellow begins, according to Professor Charles Minot, before birth, and progresses in each bone from the centre towards the periphery, or in long bones towards the end. It begins earlier in the distal bones, and then goes on from bone to bone centripetally. Concerning the exact time when these changes take place very little is known, and nothing definite. I will now show you, for comparison with the infant's bone with red marrow already described, this section of an adult bone with its yellow marrow. You see that one is quite distinct from the other. (Plate II.)

SKIN.—In the early weeks of life there are two comparatively normal conditions of the skin which may be met with, besides the more common shades of pink and red described in Lecture I. They are called *Icterus Neonatorum* and *Erythema Neonatorum.*

ICTERUS NEONATORUM.—Icterus occurs from a number of causes in the new-born infant as symptomatic of disease. There is one form, however, which is of so slight a grade and is characterized by so entire an absence of pathological symptoms that it is usually looked upon as representing a physiological condition occurring in the transition from the intra-uterine to the extra-uterine circulatory mechanism. It is to this condition that the name icterus neonatorum is given. It occurs in the first few days of life, and may not entirely disappear for several weeks. The most common time for it to begin is from the second to the third day, and, according to its intensity, the usual time of its continuation is from eight to fourteen days. It is not accompanied by any special symptoms. The conjunctivæ are somewhat tinged with yellow in a certain number of cases, but it does not seem to affect the color of the fæcal discharges or to appear in any quantity in the urine.

Careful examinations of the blood in cases of icterus neonatorum fail to show any changes beyond what would be expected in the early transitional stage of blood development commonly found at this age.

This infant (Case 24, Plate II.), a male, was born ten days ago. Its weight at birth was 3400 grammes (7½ pounds). It now weighs 3200 grammes (7 pounds). It was perfectly healthy at birth, and its skin was of the usual pink color which is seen in healthy new-born infants, such as I have already shown you. (Plate I.) On the fourth day of its life the skin began to show a yellow color, which soon became intensified, and, although it is now beginning to fade away, it represents very well the picture of a physiological icterus neonatorum. You will notice especially the yellowish-brown color of the abdomen, and the slightly icteric color of the conjunctivæ. The urine in this case is apparently normal, and the fæcal discharges are still tinged with the dark color of the meconium. In another week this yellow color will almost entirely disappear, and the skin will assume the natural pink color of a healthy infant in the first month of life. Later it will become whiter and more like the skin of the older child.

Among the many conditions which might cause this icterus neonatorum, the investigations of Birch-Hirschfeld seem to be the most thorough and to offer the most rational explanation for this condition. This author says th it is difficult to avoid associating the icterus in some way with a disturban of the hepatic circulation, owing to the transfer of its chief blood-suppl from the umbilical vein. This is especially to be seen when we consider th very evident congestion and œdema of the liver, so well described by Webe which occurs in cases in which the circulation through the umbilical cord interrupted before the respiratory movements, by their effect on the rig side of the heart, afford an adequate compensation.

The vessels in the hilus of the liver are surrounded by a dense layer connective tissue, which is continued into the organ along the branches the portal vein. In cases where there is venous obstruction in the liver consequence of delayed birth this tissue is the seat of much œdema. broad layer of gray pulpy tissue encloses the vessels and is also seen arou the umbilical vein in its diaphragmatic portion, extending also to the gal bladder. The microscopic appearances of this tissue are those of œdem with more or less abundant accumulation of round cells in the interstic That this swelling of the tissue must compress the bile-ducts is sufficient obvious, and Birch-Hirschfeld has found that not only under these circum stances are the bile-ducts distended, but there may be a positive difficulty i squeezing the bile out of the gall-bladder into the duodenum, while in th latter there is a manifest deficiency of bile. In cases where death occurs the first day of life, a beginning icterus may be distinctly detected, an Birch-Hirschfeld has reported cases demonstrating this condition, and h also observed the gradual increase of the jaundice where life had continu longer. Birch-Hirschfeld has also shown that the presence of the bile-aci may always be demonstrated in the pericardial fluid in fatal cases where th icterus neonatorum was present, whereas they cannot be found in other chi dren who do not present a jaundiced condition. This may be regarded strong evidence in favor of the hepatogenic origin of *icterus neonatoru* I shall speak of the graver forms of icterus in a later lecture.

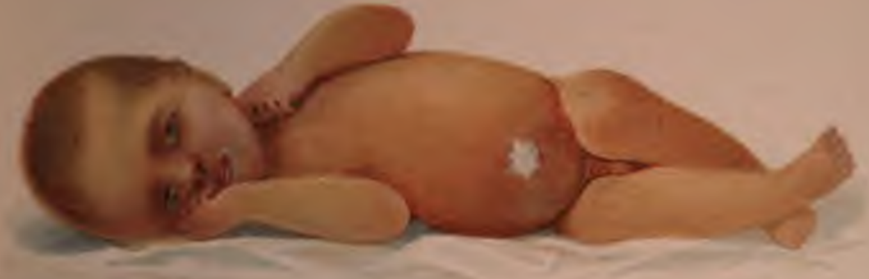
This explanation of the cause of icterus neonatorum must not, howeve be regarded as conclusive; for Cohnheim has in a number of autopsi made on this class of cases failed to substantiate the conditions described b Birch-Hirschfeld.

The ordinary bathing of the infant's skin is all that is necessary i these cases of *icterus neonatorum*, and I have never seen any indicatio for especial treatment of this condition beyond great care in establishin the equilibrium between the food and the digestion.

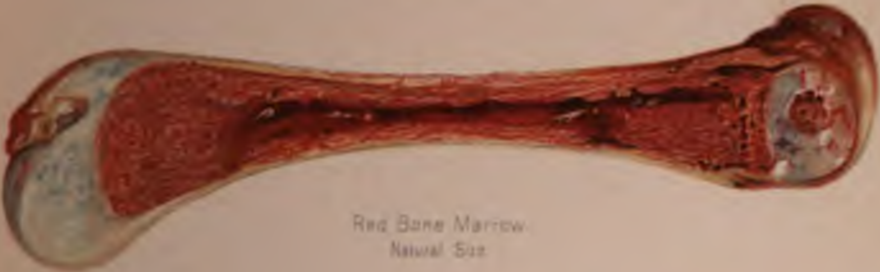
ERYTHEMA NEONATORUM.—At birth the skin is exceedingly sensiti to external influences, and in every case shows variations in color accordi to the degree of this sensitiveness, and to the greater or less amount of ir tation, whether from temperature or from mechanical causes, to which i exposed.

The delicate layers of epithelium are commonly thrown off to such degree as almost to represent a physiological desquamation, and it is ofte

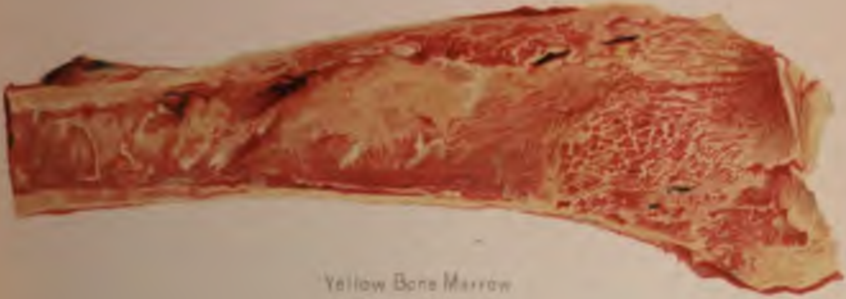
PLATE II.



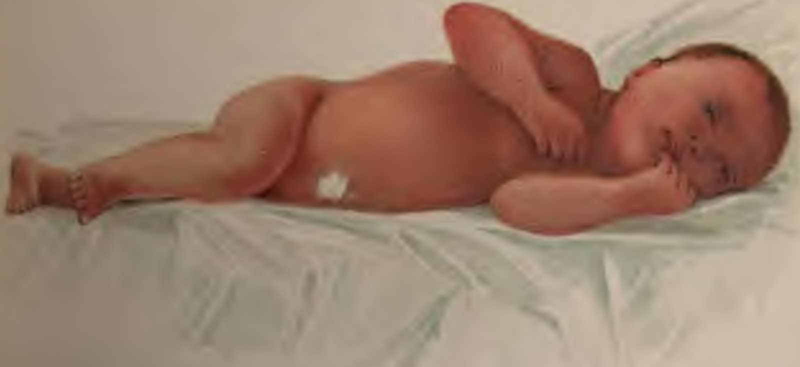
Icterus Neonatorum.



Red Bone Marrow.
Natural Size.



Yellow Bone Marrow.
¾ Natural Size.



Erythema Neonatorum.

CORD.—The cord should be carefully wrapped in antiseptic absorbe cotton, and no water should be allowed to come in contact with it. It w thus become dry sooner, and will gradually loosen and fall off.

FUNCTIONS.—The different functions of the infant vary considerab as to the time of their development, in the same way that is shown by t physical development. It is difficult, therefore, to give exact avera figures, and in fact my observation of individual cases has differed so oft from these average figures that I can only warn you that you must allc much latitude in stating the proper time for an especial function to develo

VOICE.—During the first year of its life the average infant uses voice merely in crying to express its discomforts and desires. At about t twelfth month it usually begins to enunciate single words, and in the midc or toward the end of the second year it learns to form short sentenc Children vary very markedly as to the time when they really learn to ta connectedly, but this is usually accomplished by the third or fourth ye though it is somewhat later before they master the details of language, su as the proper use of prepositions.

MENTAL IMPRESSIONS.—The infant seldom SMILES before the fifth sixth week, the change of expression of the mouth before that time bei usually an indication of some discomfort. In individual cases, howev there is no doubt that the true smile of enjoyment comes earlier, even the fourth week. The infant usually does not RECOGNIZE objects befc the sixth or eighth week. Its HEARING is soon established. The fur tions of TOUCH, TASTE, and SMELL I have already told you are apparent more or less developed at birth.

LACHRYMAL GLANDS.—The development of the function of the lac rymal glands varies considerably, but the infant will usually be found shed tears when it is three or four months old. I have, however, kno tears to appear as early as the first month. They do not at first come eve time the infant cries, so that a number of observations must be made on t same individual before deciding whether this function is present. I ha also noticed that even older infants do not shed tears with each cryin spell. These facts are at times quite important to remember, as a suppr sion of the lachrymal secretion occurs where the infant's vitality has be profoundly affected by disease, and a return of the tears is an indicati for giving a favorable prognosis, and often that convalescence is about to established.

SWEAT GLANDS.—The sweat glands are developed at about the third the fifth week. I have seen an infant in the second week of its life sufferi so much as to have its circulation seriously interfered with from the hi temperature of a bath-room where it was being bathed, while the nurse w was bathing it was perspiring profusely and was apparently perfectly co fortable. There is, however, a great variation in the time when these glan develop, and at times even in the second week of life I have noticed ca where the head was seen to perspire quite freely. I have already told that my observations lead me to think that in certain individuals the function of the sweat glands must be fairly developed at birth.

SALIVARY GLANDS.—The saliva is a secretion which is somewhat slow in being established, both in quantity and in its amylolytic property. There is not much flow of saliva in the infant's mouth for the first three or four months of its life, and even when the function of the glands has become so developed that the saliva appears in the mouth in abundance, a comparatively small amount reaches the stomach by being swallowed. It flows out of the mouth over the chin, and until the latter part of the first year, when its amylolytic action has become established, it probably plays but an insignificant *rôle* in digestion. The salivary secretion contains a certain amount of ptyalin, but its diastatic powers seem to be in process of development, and this should indicate to us that this function ought not to be forced into use in digestion until it has become much better established, as towards the end of the first year.

PANCREAS.—The amylolytic action of the pancreatic secretion I have already told you is but little, if at all, developed at birth. Towards the end of the first year the function seems to have become fairly well established, and to a degree which will not be harmed by a moderate call upon it for the digestion of small quantities of starch. The pancreatic power of digesting fat also seems to be slight in the early months of life, but to increase gradually and to be well established by the end of the first year.

BILE.—The large size of the liver at birth and during infancy is well adapted to the great metabolic activity which is needed for the development of this period of life. The investigations of Jacubowitsch show that the bile in children is poor in inorganic salts, with the exception of the iron salts. It is also distinguished by its small amount of cholesterin, lecithin, and fat, and the smaller percentage of its glycocholic and taurocholic acids, as compared with the bile of later life.

BLOOD.—The blood of infants and children is so important a subject and will in the future play so great a *rôle* in the treatment of their diseases that I have thought it better to devote an entirely separate set of lectures to its discussion. I shall, therefore, speak of it later (Division VII.).

LYMPHATIC SYSTEM.—The high development of the lymphatic system in early life is very marked. According to Foster, not only are the lymphatic glands largely developed and more active than in the adult (as is probably shown by their tendency to disease in youth), but the quantity of lymph circulation is greater than in later years. The observations of Kramstyk show that particles of fat are very easily absorbed in early life. Brunner's and Lieberkühn's glands are only partially developed in early life; the solitary and agminate follicles are rich in lymphoid tissue.

THYROID.—The thyroid body is relatively greater in the infant than in the adult.

URINE.—The urine, as I have already told you, is small in amount at birth, and during the first twenty-four hours it is not uncommon to find

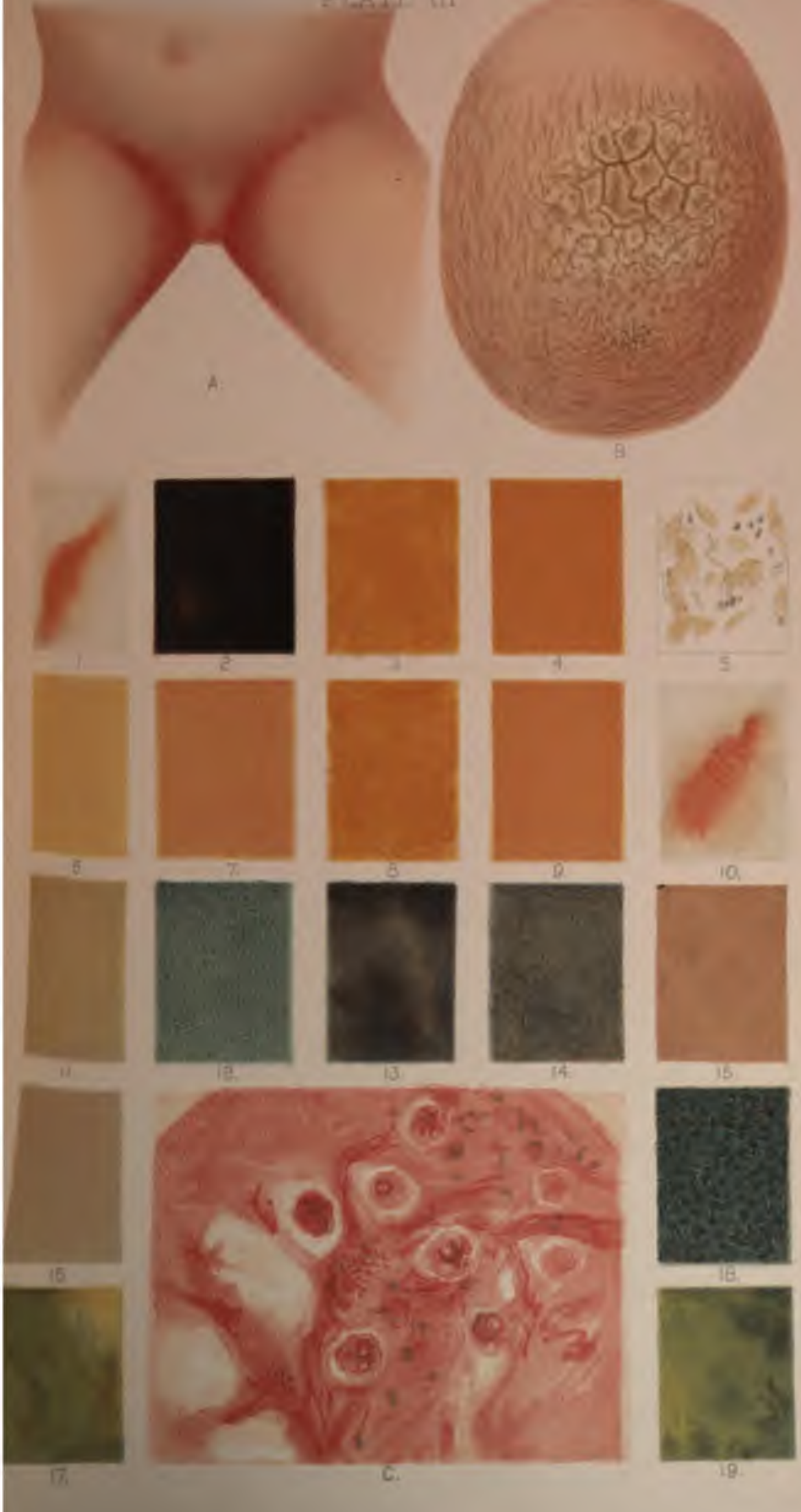
PLATE III.

A. Intertrigo.
B. Seborrhœa capitis of infants.
C. Amœba coli. Section of mesocolon. (Leitz oil immersion $\frac{1}{12}$, ocular No. 3.)

Napkins.

1. Detritus of uric acid infarction (stain during early days of life).
2. Meconium.
3. Breast-milk.
4. Breast-milk.
5. Crystals of uric acid and urate of ammonium (hedgehog crystals) taken from
6. Substitute feeding. Fat, 2 per cent.; milk-sugar, 5 per cent.; proteids, 1 per cen
7. " " " 3 " " 6 " " 1 "
8. " " " 4 " " 7 " " 1 "
9. " " " 4 " " 7 " " 1 "
10. Detritus of uric acid infarction in excess.
11. Bile-stain.
12. Color of fæces after bismuth 3 grains every two hours for six doses.
13. " " " " 4 " " " "
14. " " " " was omitted for twenty-four hours.
15. Color on napkin commonly seen, but in this case excessive in amount and p logical from uric acid.
16. Color of "clay-colored" fæces.
17. Color of the change in milk-fed (breast or otherwise) infant's fæces just befo just after they are passed (not necessarily pathological).
18. Pathological color seen in Case 417.
19. " " " Case 418.

PLATE III



or none passed. The function of the kidney begins quite early in fœtal and the bladder has been found to be full of urine at birth. The urine ch is first passed is usually dark and thick, but it soon becomes of a light ow color, and is generally slightly acid in its reaction. Its specific gravity l0 at birth) falls in two or three days to 1003, and by about the fifteenth is found to be 1006. By the end of the first week and throughout dhood the amount of urine passed in twenty-four hours is relatively ater than in adult life. This in early infancy may be due to the pre-derance of liquid food, but is in part the result of the infant's more ve metabolism, for the urea is also found to be proportionately increased. ording to Foster, the presence of uric and oxalic acid in unusual quan-s is a frequent characteristic of the urine of children. It is also stated the phosphates are deficient, being retained in the body for the purpose building up the osseous system. The *uric acid infarction*, which I have ady referred to, and evidences of which may last for two or three weeks, sists of urate of ammonium (hedgehog crystals), amorphous urates mixed h uric acid crystals, and some epithelial cells (Plate III. 5, facing page). The variations in the amount of urine which has been computed to passed during the early days of infancy and childhood are very great, as amount in all probability depends very largely on the quantity of liquid ested. It is well, however, for you to have some general idea of the rmal total amount of the urine at different ages when you begin to study diseased conditions of the kidney.

The difficulties in accurately measuring the amount of urine excreted by y young infants are such that few positive statements can be made as to quantity. It is sufficient to say that it is about ninety grammes (three es) a day for the first few days, and then rises in amount very rapidly. Rietz states that during the first four days of life the urine contains or less albumin, and that this disappears at about the seventh or eighth It also frequently happens that the first urine that is passed is cloudy. he following tables (Tables 28 and 29) give approximate figures for y and childhood:

TABLE 28.

Age.	Total Urine in 24 hours.
months	250–410 c.c. (8⅓–13⅔ ounces) (Pollak).
months	986 c.c. (33 ounces) (Camerer).

TABLE 29. (Schabanowa.)

Age.	Total Urine in 24 hours.
5 years	760 c.c. (25 ounces).
years	1043 c.c. (34½ ounces).
years	1430 c.c. (47 ounces).

ften convenient to know how much urine is excreted for each kilo-body-weight. The following table (Table 30) represents the ome careful work which has been done on this subject:

TABLE 30. (Vierordt, in Gerhardt's Handbuch.)

Years.	Number of Cases.	Average Body-Weight, in kilogs.	Total Amount of Urine in 24 hours, in c.c.	Number of c.c. of Urine excreted for each kilog. of body-weight.
3–5, boys	4	13 82	743	53.08
3–5, girls	4	14.73	708	48.00
6, boy	1	15.5	1209	78.00
7, boy	1	22.42	1055	47.06
11, boy	1	24.0	1815	75.64
13, boy	1	32.69	756	23.12
Adults	—	63.0	1700 to 1800	28.00

URINE OF ADOLESCENCE.—It is well to bear in mind, in connec with the conditions of the kidney which exist during the period of deve ment, what has been termed the *urine of adolescence.* At puberty t appears to be a disturbance of the equilibrium of the renal circulation curring so frequently, and presenting so distinctively the characteri of a simple hyperæmia, that we are justified in looking upon it as a phy logical rather than as a pathological condition.

This physiological congestion of the kidney is probably closely nected with the development and increased activity of the uterine circula in the female, and with the prostatic and genital blood-supply in the n The importance not only of knowing that such a condition exists at pub but also of bearing it in mind when we are called to treat children who ar the border-line between childhood and adolescence, is too little recogn and this want of recognition often leads to unfortunate mistakes. Nume instances of the truth of this statement must arise in the practice of e physician: so that I need refer only to one of a number of cases of kind which have come under my notice.

CASE 29.—A girl, thirteen years old, was brought to me for advice with the follo history. She had always been somewhat delicate, but had never had any special di and was considered to be fairly healthy, until she was twelve years old. She then beg grow very fast in height without a corresponding development in weight and general cular strength. When she was twelve and a half years old the catamenia appeared were accompanied by severe pain. This was in November. In December, six weeks the catamenia again appeared, and were accompanied by considerable pain and general tration. The child at this time looked pale and thin, had very little appetite, and was fatigued. A physician was consulted, who prescribed strong food, such as meat, a t and gymnasium exercise. This advice was followed implicitly, and the child was ma exercise especially the muscles connected with the abdomen and pelvis three or four a week at the gymnasium, and by daily home exercise, such as lying on the back and ra the legs. Under this treatment the child rapidly grew worse, and the catamenia di return in January. The physician then examined the child carefully, with negative re until the following analysis of the urine was made (Analysis 1):

ANALYSIS 1.

March 19.

Specific gravity	1035.
Reaction	Acid.
Albumin	0.05 per cent.

Epithelial and hyaline casts were found.

The child at this time was thirteen years old. The physician now became much alarmed, and informed the parents that their child had a form of Bright's disease. This statement completely demoralized the whole family, carrying with it as it did to their minds the impression of a fatal issue of the disease. The father, who was just starting on an important business trip involving much money, was so distressed that his business was entirely thrown aside, as he wished to remain near his child. Under these circumstances further advice was sought for, and the case was placed in my hands. A careful physical examination revealed nothing abnormal about the child beyond overgrowth, with a resulting anæmic condition. The urine was sent to Professor E. S. Wood for expert analysis, with the following result:

ANALYSIS 2. (Wood)

March 21.

Color	Normal.
Reaction	Acid.
Urophæin	Normal.
Indoxyl	Normal.
Urea	Increased.
Uric acid	Increased.
Albumin	Very slight trace.
Sugar	Absent.
Bile-pigments	Absent.
Specific gravity	1023.
Chlorides	Normal.
Earthy phosphates	Normal.
Alkaline phosphates	Slightly diminished.
Sediment	Excess of mucus—a little vaginal epithelium—an occasional hyaline granular and epithelial cast—an excess of renal epithelium — an occasional blood-globule.
Total amount in 24 hours	960 c.c. (a little less than 2 pints).

I was enabled from this report to tell the parents that the examination showed simply a slight renal hyperæmia, the very small trace of albumin and the very few casts all pointing towards that condition and against any serious renal disease. The parents' minds were much relieved, but no new treatment was instituted, and, as the child was weak and languid and did not appear to be improving, I decided to have another urine analysis made before giving any further advice.

The next analysis showed the following conditions:

ANALYSIS 3. (Wood.)

April 4.

Color	Normal.
Reaction	Acid.
Urophæin	Normal.
Indoxyl	Increased.
Urea	Increased.
Uric acid	Much increased.
Albumin	Very slight trace.
Sugar	Absent.
Bile-pigments	Absent.
Specific gravity	1027.
Chlorides	Normal.
Earthy phosphates	Increased.

Alkaline phosphates	Normal.
Sediment	Much calcic oxalate — much vaginal epithelium and uric acid crystals—excess of renal epithelium — a few blood-globules—an occasional hyaline and granular cast of small diameter with renal cells and blood adherent.

This analysis showed the urine to be so concentrated that the indications for treatm were very evident.

The child was not allowed to go to school or to the gymnasium. She was made to in bed for several hours twice a day. Her diet was largely milk in considerable quant meat especially being withheld. She was also made to drink freshly distilled water, 250 (about eight ounces) once in six hours. She was allowed to take a slight amount of e cise out of doors, but to a very limited degree.

This treatment, so radically different from what she had previously received, was i tuted on the ground that while there was no organic disease of the kidneys, yet the hy æmic condition was so pronounced as to show that the renal tubules were being kept condition of chronic irritation to a considerable degree. This irritation was so promine factor in the girl's generally debilitated condition that it became for the time being primary importance. The causes for the irritation were very evident. The catamenia just being established; accompanying this was the irregular and varying congestion of pelvic organs originating with the uterus and ovaries, and extending to the kidneys. addition to this was the adolescent condition so common in children growing too rap for their general nutrition. The girl had been made to exercise the very muscles w exercise would naturally tend to increase pelvic congestion, and was fed largely on m which would not tend to lessen the renal congestion. The indications for treatment evidently rest for the pelvic organs and dilution of the irritating concentrated urine w was passing through the renal tubules.

Under this course of treatment the child began slowly to improve. She became anæmic; her appetite increased, and was less capricious; she began to gain in wei to sleep well, and to have more strength. On April 11 another analysis was mad Professor Wood, with the following result:

ANALYSIS 4. (Wood.)

April 11.

Color	Normal.
Reaction	Slightly acid.
Urophæin	Diminished.
Indoxyl	Normal.
Urea	Slightly diminished.
Uric acid	Increased.
Albumin	Very slight trace.
Sugar	Absent.
Bile-pigments	Absent.
Specific gravity	1015.
Chlorides	Normal.
Earthy phosphates	Normal.
Alkaline phosphates	Diminished.
Sediment	Excess of mucus and renal cells — few blood-globules — one hyaline cast detected (after a search of more than an hour) —vaginal epithelium.

analysis showed such marked improvement that it was evident that we were th an exaggerated physiological rather than with a pathological condition, and eatment was a wise one. I think it may be of interest to you to follow the provement which took place later, and which resulted in complete recovery in year from the time when the albumin and general renal irritation were first This improvement is shown in the following table:

TABLE 31.

Analysis.	May 2.	May 6.	June 7.
umin	Slight trace.	Slight trace.	Very slight trace.
cific gravity	1008	1033	1018

ediment was very similar in all these analyses, and consisted of mucus and of ithelium, a little secondary calcic oxalate, and an occasional hyaline cast and ule.

l analysis (Analysis 5), made January 29, enabled me to give the following satisort, namely, that there was no evidence of any renal disturbance whatever, and ine was normal in every way.

ANALYSIS 5. (Wood.)

January 29.

r	Normal.
ction	Acid.
phæin	Normal.
oxyl	Normal.
a	Normal.
c acid	Normal.
umin	Absent.
ar	Absent.
-pigments	Absent.
cific gravity	1020.
orides	Normal.
thy phosphates	Normal.
aline phosphates	Normal.
iment	Vaginal epithelium and mucus.

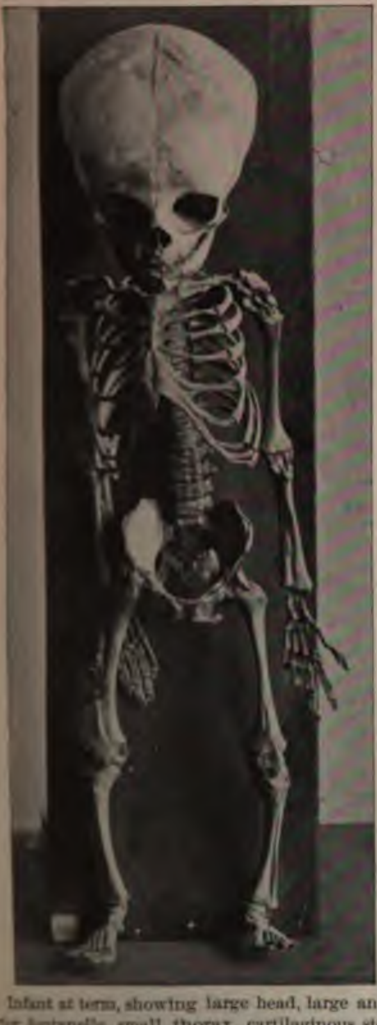
ESTINAL DISCHARGES.—The contents of the intestine continue xed with meconium for three or four days or a week, the longer ng when the infant is weak and does not nurse well. After this infantile discharges, which have a characteristic appearance as dis- d from those of the older child, appear. It is especially necessary to familiarize yourselves with their characteristics, as they are an t guide to the proper feeding of the infant and are an index show- her the food is properly digested and assimilated. When the nutri- milk, with the percentages of its different elements corresponding to normally found in good average human milk, the discharges are of yellow color, smooth, unformed, of medium consistency, showing a portion of water, and sometimes changing on exposure to the air to sh yellow. They as a rule contain undecomposed bile-pigment and , while the older child's and the adult's discharges do not contain the ecomposed. The amount of fæcal discharge in the first day of life forty-five grammes (one and one-half ounces), and increases in the following days to fifty grammes (one and two-thirds ounces). It consist mucus, fat, epithelial remains, and a slight amount of albuminoid mate In early infancy there are from two to four discharges daily. As the c grows older there are two and finally one in the twenty-four hours. T do not lose their yellow color until amylaceous or albuminoid food is gi when the different shades of brown begin to appear; they are not for until something besides milk is swallowed. Starting at birth with the st meconium, infection by the mouth and rectum quickly occurs, and in a s time almost any form of bacteria may be found in the discharges, but ch such putrefying forms as Proteus vulgaris (Jeffries). With the sucklin the infant and the substitution of the refuse of the milk and the secre of the digestive tract for the meconium, a sharp transition occurs. Ins of the generally distributed forms, causing decomposition, only two k of bacilli are now regularly found, the Bacillus lactis aërogenes and Brie bacillus, the first chiefly in the upper parts of the intestine, the second i lower part. When the infant begins to take a mixed diet, quite a numb forms of bacilli appear, among them the Streptococcus coli gracilis, the p fying green fluorescing, a tetrad coccus, and several kinds of yeast. color of the infantile intestinal discharges when the nutriment is milk a whether human or animal, seems to depend somewhat on the percentag fat, as you will see by examining these napkins with discharges on produced by milk of varying percentages (Plate III., 3, 4, 6, 7, 8, 9). consideration of the fæcal discharges of the infant is so closely conn with the subject of infant feeding that I shall leave anything further w I have to say about it until we begin to consider that important bran our medical studies.

We have now, gentlemen, studied the principal anatomical and ph logical facts concerning infants and children which will be of practica in aiding us to diagnosticate and treat their diseases. Before beginnin study of these diseases I should like to present for your inspection actual illustrations of normal infants and children. I have explaine shown to you in a general way the normal condition of the external po of the body, and also what it contains.

INFANTILE SKELETONS.—It may aid you to remember wh have said if you will also first examine these two skeletons. One (Fig is the skeleton of an infant at term. The other (Fig. 34) is the skelet an infant at nineteen months.

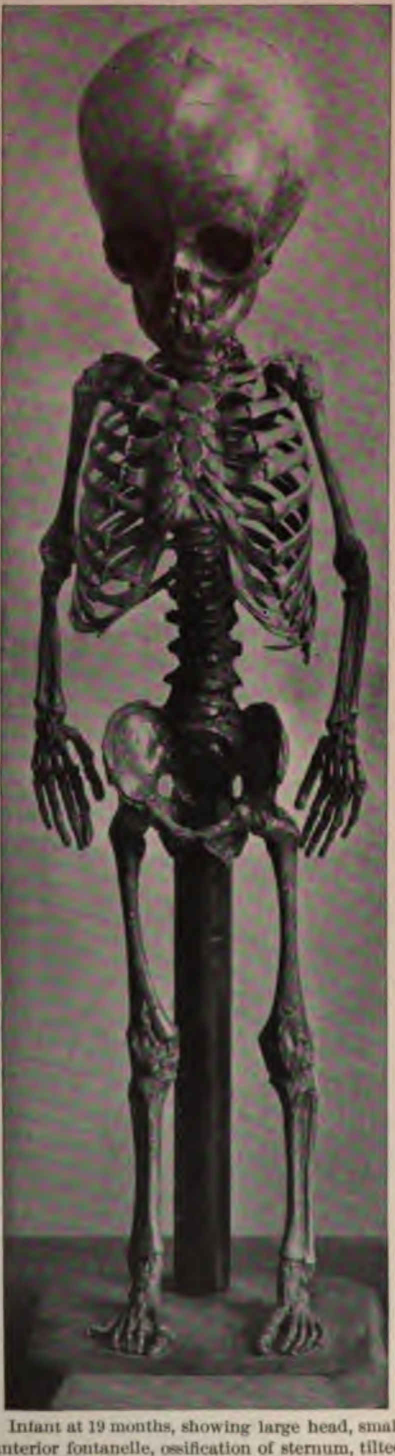
You see in the younger subject the large head in proportion to the thorax, and the lack of development of the face in comparison wit head, which is very evidently due to the rudimentary development o jaws. You will also notice the widely open anterior fontanelle. On exa ing closely the sternum you will see that it is not in one piece, as in the but that the centres of ossification with the intervening cartilaginous nections, which I have already described in a previous lecture (Divisio Lecture III.), are well marked. You will also notice what I hav

FIG. 33.



Infant at term, showing large head, large anterior fontanelle, small thorax, cartilaginous sternum, tilted pelvis, and bow-legs.

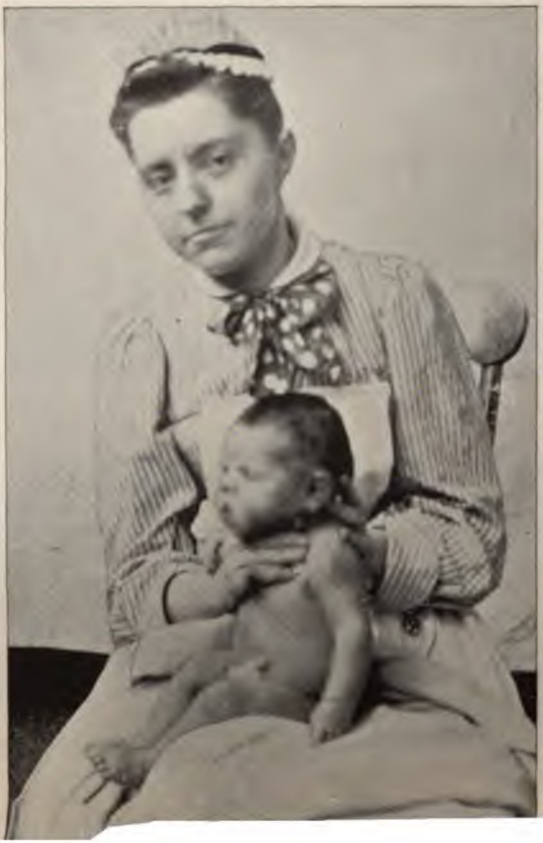
FIG. 34.



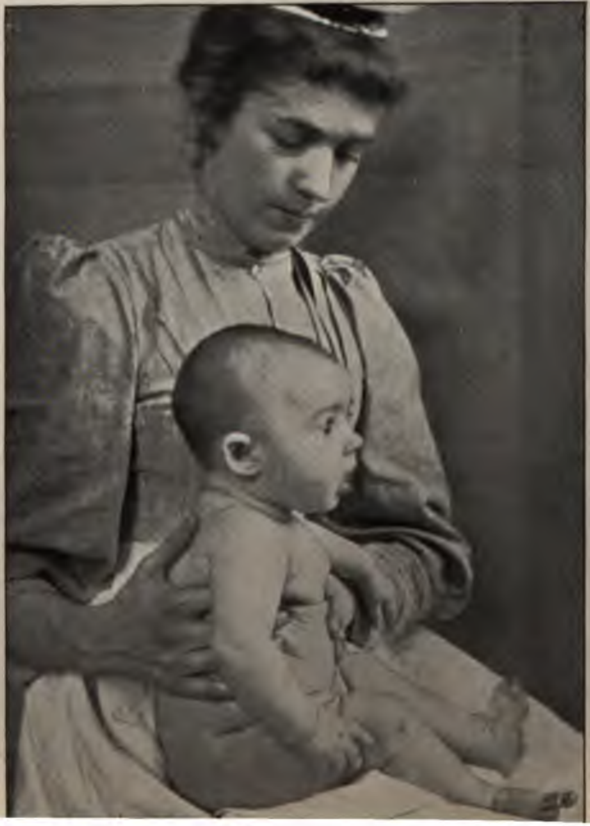
Infant at 19 months, showing large head, small anterior fontanelle, ossification of sternum, tilted pelvis, and straight legs.

Warren Museum, Harvard University.

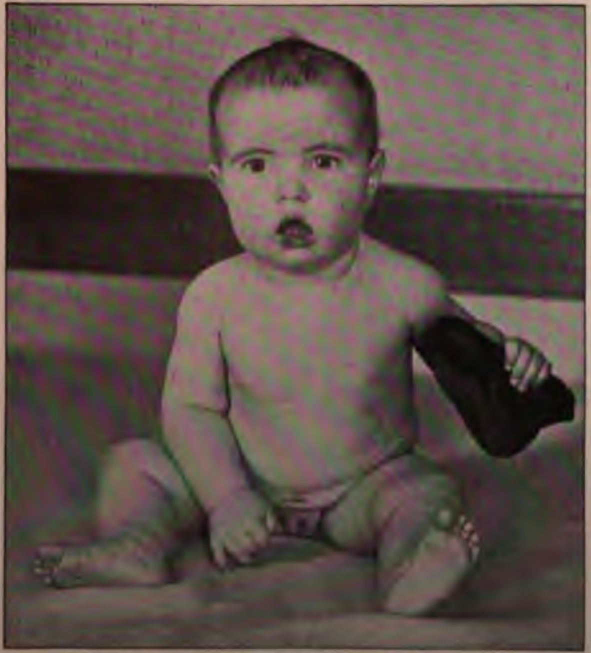
Case 30.



Case 31.

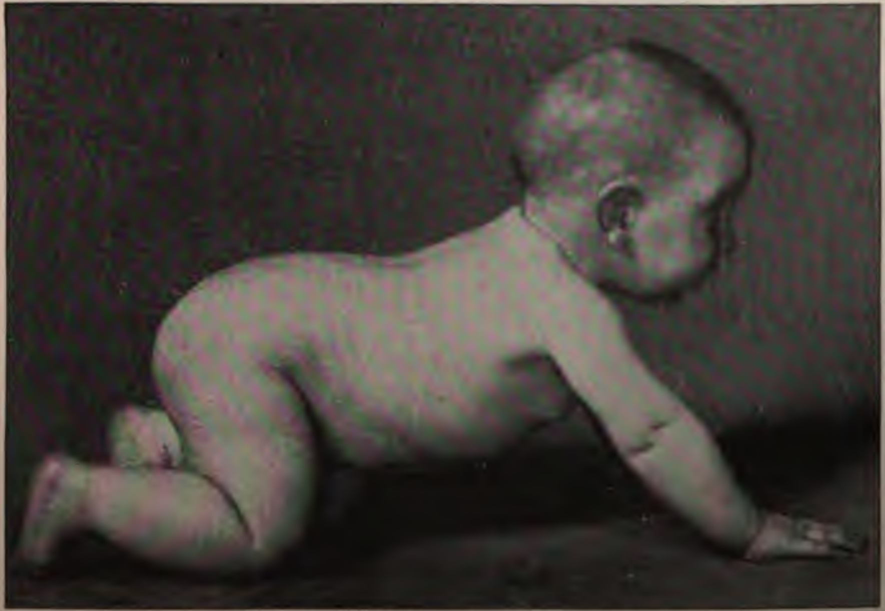


CASE 32.



Infant 8 months old, sitting alone. (Vide page 120.)

CASE 33.



Infant 10 months old, creeping. (Vide page 120.)

referred to before in speaking of the pelvis, how it is tilted forward, as compared with the adult's, and how small and contracted it looks. You will observe that the legs are not straight, as in the older child, but show decided bowing of the tibia and fibula. This characteristic condition of the legs in intra-uterine life is present at birth and continues for some months, the bones usually becoming straight by the time that the period of walking has been reached. In this skeleton of an infant nineteen months old, you see that the legs have developed naturally in their growth and are straight. The pelvis still tilts somewhat, but is evidently less contracted, or rather has begun to enlarge. The thorax has broadened in comparison with the head, and the cartilaginous sternum has become to a large degree bone. The head is still large proportionately to the face, although the jaws have developed considerably beyond what is seen at birth. The anterior fontanelle is, as you see, quite small in comparison with the fontanelle of the new-born infant.

These are the chief characteristics of the infant's and child's skeleton, and you will now appreciate this series of infants and children which I have carefully selected to impress upon you the ages at which the various stages of physical development should naturally be found.

NORMALLY DEVELOPED INFANTS.—You must not consider this exhibition of healthy infants too trivial for your closest study. I believe that one of the greatest drawbacks to the proper appreciation of the kind of knowledge which is needed to examine children successfully and intelligently when they are sick, is the lack of precise facts concerning healthy children. To know at a glance whether it is normal for a child not to sit alone or not to stand alone,—to understand its childish actions, whether in creeping or in walking,—these are data which will be of infinite use to you in your nursery practice. I therefore do not hesitate to occupy a certain amount of time in showing you these infants whose physical development and strength represent about what you will meet in a large number of average individuals at these especial ages.

This infant, a few hours old (Case 30), is, as you see, absolutely *unable* to sit up or *to hold its head up*. The swollen condition of the face which is so frequently seen during the early hours of life after a prolonged labor is well exemplified here, and will pass away naturally by to-morrow. When the head is not supported, it falls in any direction on the thorax. You must, of course, impress upon the nurse that care should be taken to support the head gently as well as the back in lifting and carrying the infant at this age, and until the muscles have developed to a degree which will render it possible for the infant to support its own head, or until, as is still more important to remember, it has learned to co-ordinate sufficiently to make use of these muscles. The time when the infant begins to sustain its own head varies considerably, certain individuals being decidedly precocious in this respect, while others, without showing any sign of disease, are much later in sustaining their heads than is the case with the average infant. From *two to three months* is about the time when the normal infant, according to my experience, *sustains its head* without assistance, although this is usually done in a very vacillating way up to the fourth or fifth month.

This infant (Case 31) is two and one-half months old. It is apparently normally developed as to weight, height, and general growth, and, although it cannot sit alone, and has to have its back supported, it holds up its head quite steadily.

We have now arrived at a period of growth when the infant can be pu on the floor without having to be held by the nurse. This is usually fron the *seventh to the ninth month.*

The nurse has just undressed this infant and placed it on the floor (Case 32), so that yo can see it from all points of view. The infant is *eight months* old, and is normally devel oped. She, as you see, *sits alone perfectly well,* and can be allowed to amuse herself on th floor without fear of her falling over.

The next infant which I shall have brought in to show you is a little more advanced i its physical development, as it is ten months old. While the one at eight months (Case 32 can sit very well, you see that it cannot as yet move about the floor, and in fact does no attempt to do so; but watch how this active infant, *ten months old* (Case 33), as soon as yo place it on the floor, turns over on its hands and knees and moves across the floor, rathe awkwardly, perhaps, and not very fast, but it certainly can be said to *creep.* It is natura for the average infant of from ten to twelve months to move about in this way. The loco motion of infants at this age, however, is not always on their hands and knees. Man individuals never creep, but their first efforts in progression are represented by sitting o the floor and dragging themselves along with one leg.

Now we will see what this next infant, which is *twelve months* ol (Case 34), can do when we place it beside the others.

This infant has arrived at a period of development when it is strong enough to pul itself up and *stand by a chair,* and you see that it immediately performs this feat, and is evi dently very proud of the accomplishment.

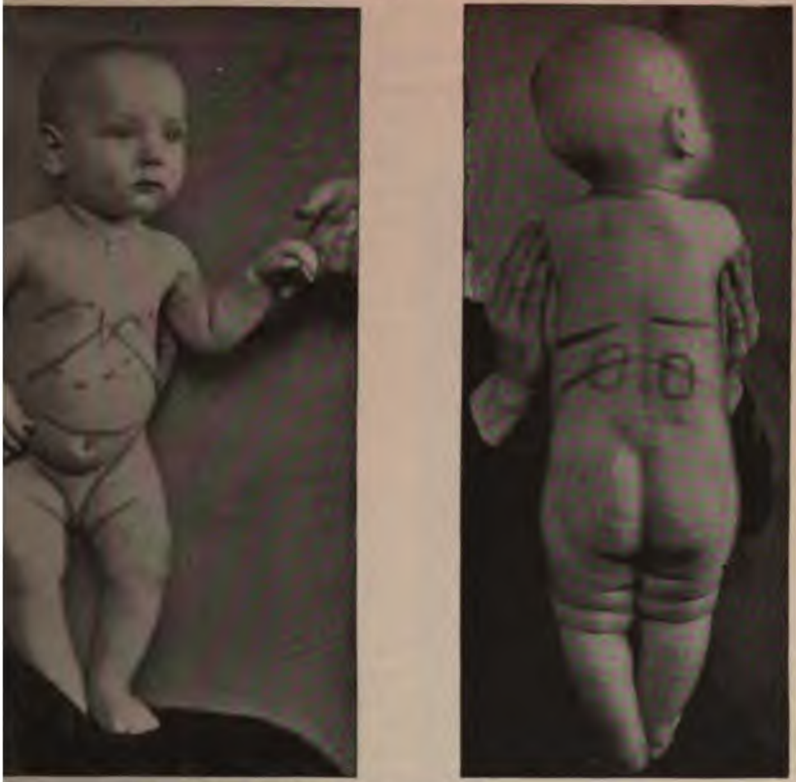
Finally, here is another infant, fifteen months old (Case 35), and normally developed, a you will notice if you carefully examine it. The head proportionately to the adult's is still large. The thorax is well formed, with the natural curves of the back, and the legs ar straight. It can walk very well, and although it is rather averse to performing for you benefit and is crying, still you see that it can go across the floor to its mother perfectly wel without falling. The age at which the average infant walks of course varies, and man infants never attempt to creep, but begin to walk before they are twelve months old. Th average infant, however, *walks* from the *twelfth to the fifteenth month.*

TOPOGRAPHICAL ANATOMY OF THE EARLY PERIODS OF LIFE.—I have already spoken of the importance, for purposes of diagnosis, of recognizing the fact that the organs differ in the space which they occupy in the body according to the stage of development of the child. Well-marked periods are thus shown to exist by physical examination as well as by anatomical research, and the results of these different methods of investigation are found to correspond. I have always found that a careful consideration of the period of development is of the first importance when beginning to make a diagnosis of disease, especially of the heart and lungs. The large size of the liver in infants and the comparatively greater proportionate size of the heart to the lung in the middle years of childhood are striking instances of the truth of this statement, and should warn us that more than ordinary care should be employed in diagnosticating a pneumonia of the right lower lobe behind in infancy, or a dilated heart in childhood. Three periods of growth are of especial significance in this connection: 1. The development of the organs in the first year, especially in the first half of the year. 2. A period occurring during the fourth, fifth, sixth,

, and perhaps eighth and ninth, years. 3. The later years of child-

epresent the first period I have taken this infant (Case 36), seven months old and
developed, and I have outlined in black the principal points both in front and
hich will be useful for you to remember when making a physical examination at

CASE 36.



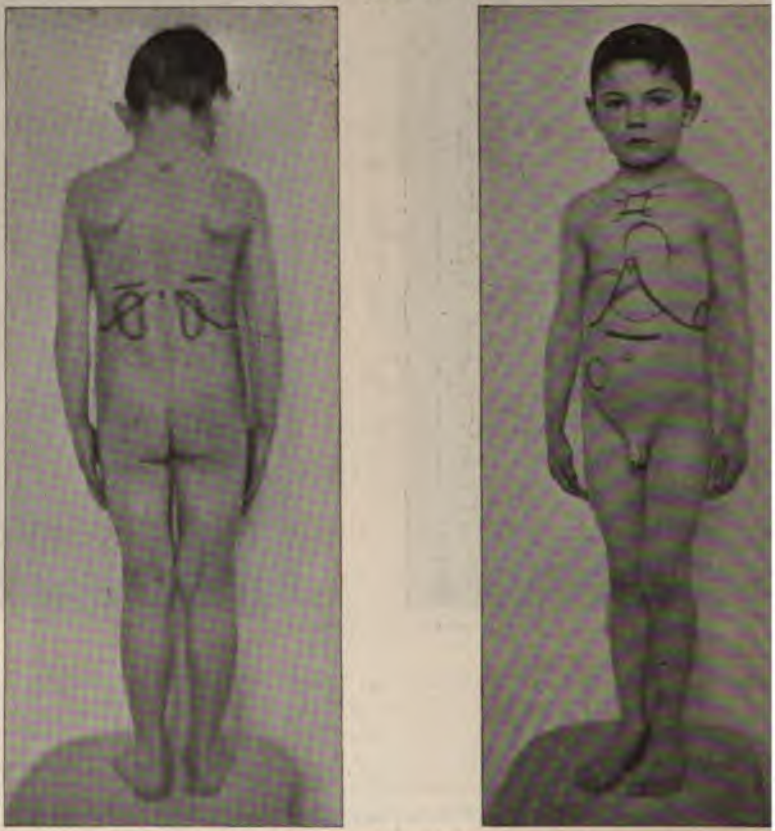
Normal infant seven months old.

look at him in front. The plain dark lines have followed the lower margin
bs and the outline of the ensiform cartilage and manubrium. To the left of the
rt of the left parasternal line you will notice a small curved line. This represents
ute dulness of the heart. The relative dulness is very slight, and indeed almost
tible even on light percussion over the sternum. This area of dulness can almost
d by the end of the finger used for percussion. It is bounded by the fourth rib or
rspace above, and is just within the mammary line. There is very fair resonance
e whole length of the sternum. The interrupted lines represent the upper and
rders of the liver. There is not much to say about the upper line, but the lower
teresting and instructive as illustrating the large size of the liver in early infancy,
see how little of the stomach, which is here represented by a dotted line between
of the liver and the left border of the ribs, is to be reached by percussion. The
h, of course, in this infant, empty. When full, it comes out much further under
of the liver. This general idea of its position, however, is very important when
to consider cases of improper feeding where we have to determine whether we have
l stomach to deal with. The broad black line just above the level of the umbilicus

marks the transverse colon, which in infancy has a relatively low position. The
which is marked by a black circle, stands, as you see, high in the abdomen, near the
superior spine of the ilium. I have also outlined the upper piece of the sternum a
cated the clavicle and first rib. On looking at this infant's back you will see that
marked the lower borders of the thorax, the kidneys, and the lower borders of th
The left kidney is decidedly higher than the right at this age. While the lower b
the lung on the left comes down as far as the tenth rib, the corresponding border of t
lung, owing to the large size of the liver, descends only as far as the ninth rib.

I shall now show you a child in the second period of growth (Ca
In this middle period of childhood the heart has developed more
proportionately than the lungs, and takes up more space in the a
portion of the thorax.

CASE 37.



Normal development at six years.

This boy, six years old, and properly developed for his age, presents certain p
interest which differ from the infant and the adult, and which should be carefully ta
account when we are making a physical examination at this age. You see I h
marked the manubrium, indicating the clavicles, the first and second ribs, the
cartilage, and the lower borders of the thorax. The area of cardiac dulness is far
than in this infant (Case 36). This dulness should, so far as the sternum is conce
determined by light percussion directly over the sternum from above downward.
way we can detect the change in the percussion note over the lower part of the

better than by percussing from the lung to the sternum, since the former is so much more resonant that the sounds are more difficult to distinguish and are often misleading. The upper resonant part of the sternum, on the other hand, presents an excellent opportunity for comparison, and brings out the delicate shades of sound which are needed in getting the relative dulness. This relative dulness, however, is usually pronounced under the lower part of the sternum in this period of development, and you hear as I percuss to the left how it shades off into the absolute dulness of the precordia. Absolute dulness under the sternum, unless depending on pathological conditions, is rare even at this age, when it is also rare not to have this physiological relative dulness. In this period the dulness of the heart extends higher in the left parasternal line than at any other time of life. The lower border of the third rib usually marks the upper border of the absolute dulness, which extends also to the left parasternal line and keeps well within the mammary line. The relative dulness, on the other hand, reaches as high as the lower border of the second rib. It then passes to the right under the upper third of the sternum, descends obliquely to the fourth right costal cartilage, and then keeps closely to the right parasternal line.

To the left it extends well out to and perhaps a little over the mammary line. The area of dulness in this special boy I have outlined where as I percussed his precordia you heard a marked absolute dulness between the mammary and left parasternal lines gradually shading into the marked relative dulness of the lower third of the sternum. You will notice that this is a far different result of percussion from that which is found in the adult, and in this infant (Case 36), where, as I have shown you, there is no dulness under the sternum, and the absolute dulness rises only as high as the fourth costal cartilage in the left parasternal line, and the relative dulness only to the third interspace. The relative dulness also extends only as far as the mammary line. The impulse of the heart is usually found a little higher in infants and in young children, irrespective of these periods, than in older children and in adults.

You will next notice that a much smaller space is occupied at this age by the liver than in infancy. This I have indicated by the double line, which rises as high as the fifth rib in the mammary line, and to the attachment of the sixth or seventh right costal cartilage to the sternum. The dotted line of the stomach, on the other hand, occupies, as you see, a much larger space than in the infant. The line of the transverse colon stands proportionately higher, the cæcum rather lower. On examining the back, you see the lower border of the right lung is still a trifle higher than that of the left, and comes to about the upper border of the tenth rib, while on the left side it extends to the lower border of the same rib. At this age the liver has diminished in size relatively to such an extent that the difference of the position of the lower borders of the lung is but slight.

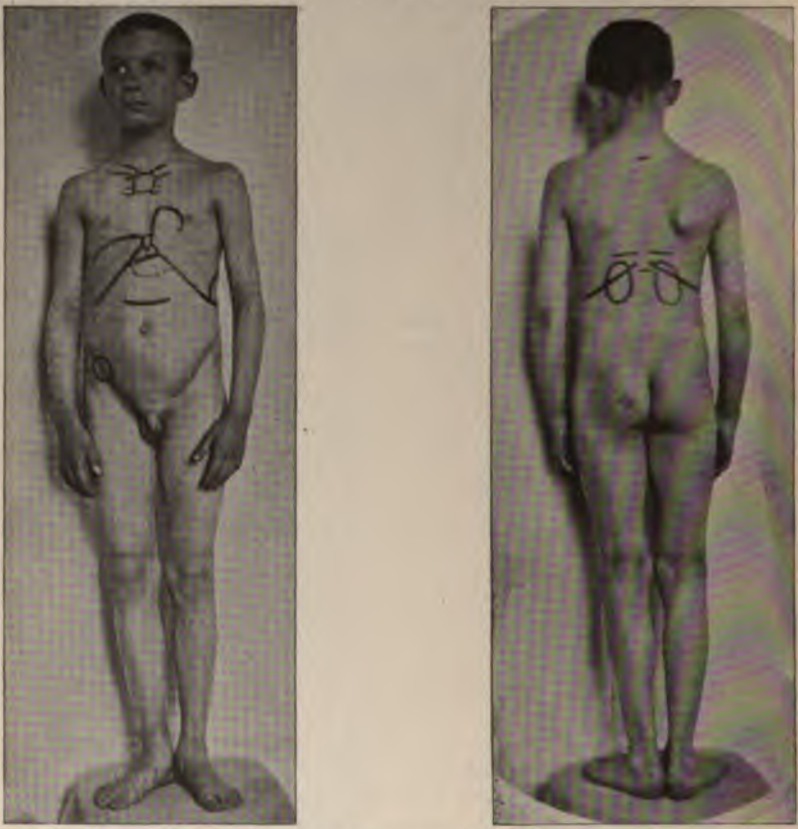
The kidneys are about on a level on both sides. I have also indicated as landmarks for your study the first and twelfth dorsal vertebræ. You see that this child is passing through transitional stages of physical development, and is gradually approaching the adult type of perfected growth.

This perfected growth, so far as the topography of the organs is concerned, is reached in the last years of childhood and at about the age of puberty. The organs of the child seem at this age, although they have not yet acquired their complete growth, to present for purposes of percussion the outlines which we are accustomed to see in the adult, with the exception possibly of the position of the cæcum.

This normally developed boy (Case 38), twelve years of age, illustrates remarkably well the relative topographical correspondence of later childhood and adult life.

I have, as in the boy of six years (Case 37), outlined the manubrium, clavicle, first and second ribs, ensiform cartilage, and the lower borders of the thorax. The curved line passing up the left parasternal line to the fourth rib and keeping within the mammary line marks the absolute dulness of the heart, and corresponds to the topography of the adult's heart. The upper line of the liver is, you will notice, found to be about at the level of the fifth rib in the mammary line, and does not extend beneath the lower border of the rib but is just below the tip of the ensiform cartilage. The dotted line represents the stomac The spleen has its upper border at the ninth rib, and its lower portion comes down as far the lower border of the eleventh rib. The cæcum you will notice is marked in the upp

CASE 38.



Normal development at twelve years.

part of the right groin. The transverse colon is about midway between the stomach an the umbilicus. Looking at this same boy from behind, you will see that I have marked hi kidneys and the lower borders of his lungs in about the same relative position as occurs i the adult. I have also indicated the first and twelfth dorsal vertebræ.

These representatives of the normal development of important period of life have not only been carefully mapped out by myself by percussio and in accordance with the anatomical knowledge which we possess on thi subject, but have also been verified by Professor Dwight, who has examine each child carefully and has satisfied himself that my marking is correct I shall at present say nothing more about these various stages of development, the knowledge of which I hope you have now mastered sufficiently t utilize in connection with the subjects to which I shall next direct you attention.

DIVISION III.

HYGIENE OF THE NURSERY.

LECTURE V.

THE NURSERY.—INTERTRIGO.—SEBORRHŒA CAPITIS OF INFANTS.—CLOTHING.—FEET AND SHOES.—SLEEP.—OUT-DOOR AIR.—NURSERY-MAIDS.—SCHOOL.—IMPORTANCE OF CORRECTING DEFECTS OF POSTURE.—VACCINATION.

WE have studied the infant at term with regard to its normal anatomy and physiology. We have also examined it at different periods of its growth up to the age of puberty.

I must now, before undertaking to explain and to show to you the various diseases of early life, impress upon you the importance of a knowledge of the care of the infant and child in health. I am accustomed to place what I have to say on this subject under the title of "Hygiene of the Nursery." It is essentially in the nursery that we should study the healthy child, as the nursery is its home, where it feels most at ease and behaves in the most natural manner. The general hygiene of the child is represented in its nursery, and we should therefore by our knowledge and advice so direct these questions of nursery hygiene as to give this sensitive, easily impressionable young human being the best opportunity to develop into a healthy and vigorous adult.

NURSERY.—We cannot, of course, in every case procure for the child the surroundings which are best for it, but we can at least impress on the parent what these surroundings should be, and how important they are for the general health of the child. The nursery should be high from the ground and out of reach of the dampness which arises towards the latter part of the day.

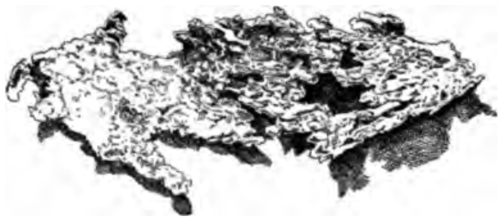
SUN AND WINDOWS.—It should have a sunny exposure and large windows high enough from the floor to avoid having the younger children continually pressing their faces against the glass to look out, and thus frequently catching cold from the little currents of air which penetrate most window-casings. The mothers often overlook this simple manner of catching cold, and wonder how their children, who are so closely watched, could have contracted the catarrhal conditions which you will be summoned to treat.

Papers and Carpets.—In my opinion it is much better not to hav paper on the walls or a carpet on the floor. Young children are very s ceptible to inhalation poisons, and to organisms of all kinds. Many a c of anæmia, naso-pharyngeal catarrh, and stomatitis ulcerosa has in my ex rience apparently arisen from arsenic in the paper. Dust also, with its mu titude of organisms, which with the most careful sweeping it is impossi to get rid of, is another source of irritation to the respiratory tract. I sh speak of arsenic in the wall-paper later, but here merely state, in support what I have just said, that very minute amounts of arsenic appear to aff young children, and that the paper itself is a receptacle for micro-organis which are difficult to eradicate.

Picture-Mouldings.—It is advisable not to have any picture-moul ings on the walls, as they are a place for dirt to gather which it is imp sible to remove properly.

Floor.—There should be as few cracks as possible in the floor, and should be smooth, so as to be easily cleansed. The floor, however, sho not be highly polished, for children frequently fall while playing, and son times quite severe accidents occur in this way. I have known of one lit boy four years old (Case 39) who broke his arm by simply slipping a falling on the floor. It is t often the case that blows a resulting injuries are ov looked because it is thoug that all children naturally f and strike their heads. T little boy, two years old (C 40), fell on his nursery fl six months ago. Nothi especial was noticed at t time, but one week later a swelling appeared on the right frontal bone, a later three small ulcers were noticed in the same locality. The child w brought to the hospital, and Dr. Augustus Thorndike examined and moved this sequestrum, 6½ cm. (2⅝ inches) long, exfoliated from the rig frontal bone and extending from the temple and line of the hair backwa including a little of the sagittal suture.

Fig. 35.



Sequestrum from frontal bone, natural size. Child two years old.

Walls and Ceiling.—I prefer the floor, the walls, and the ceiling be painted. Not only can they then be frequently washed and scrubb but when the child happens to have any of the contagious diseases, t whole room can so easily be disinfected that it saves much trouble a expense.

Rugs.—A rug is desirable in the middle of the room. It should nev be an antique; in fact, it is better to have new, simple carpet rugs. T rug should not be too large nor too heavy to be frequently taken out into t open air and thoroughly beaten.

Bed.—The child's bed should be iron, painted so that it can be careful cleansed by wiping, and its sides, as the child grows older, should always be kept high enough, by some simple contrivance, to prevent the child from climbing over them. As few hangings and useless curtains, with which the mother is usually so desirous of draping the bed, should be used as possible.

Pillow and Mattress.—The pillow and mattress should be of hair, and the latter should be protected by a rubber sheet and aired thoroughly every day. Especial precautions should be taken that the child does not kick off the clothes at night. It is well for the nurse's bed not to be close to that of the child. This entails a little extra trouble on the nurse's part, but her breath is not a healthy pabulum for the child's lungs, which require fresh, pure air of their own.

CLOSETS AND DRAWERS.—The child should have its own closet and its own drawers. The nurse's belongings ought to be kept in a separate room. The closets and drawers should be cleansed at least once a week.

FURNITURE.—There should be sufficient furniture in the room for comfort, but stuffed furniture should be avoided. As little as possible that is complicated or cumbersome should be kept in the child's nursery.

CURTAINS.—Only simple muslin curtains, which can be washed, should be used at the windows.

HEATING AND VENTILATION.—The heating and ventilation of the nursery are of great importance. The child requires pure, warm air. The temperature of the room can vary somewhat according to the climate, but, as a rule, the average should be from 18.8° to 21.1° C. (66° to 70° F.). The open wood fire is best both for the character of the heat which it gives, and for its value as a means for promoting ventilation.

DRAUGHTS.—We must take into consideration the currents of air in the nursery, so that the mother, understanding the atmospheric conditions which surround her child, can give the simple directions, which she has learned from us, to the nurse. This is by no means an unnecessary precaution, for one of the worst cases of rheumatism in the hip-joints (Case 41, Division XVIII., Lecture LIII., page 1085) which has come under my notice was that of a child two years old who was allowed to sit on the floor with its back to the open door, and directly in a line with the open fireplace. The direction of the currents of air between the doors, windows, and open fireplace is admirably and scientifically described by Mr. John Pickering Putnam in his valuable work entitled "The Open Fireplace," and I have represented the direction of the cold-air current in a picture (Fig. 36, page 131) which I shall presently show you. If the child is much on the floor, a sheet can easily be placed over the cracks of the door; and plain white sheets are always the best articles for screens or portières.

WINDOW VENTILATORS.—A plain piece of wood the width of the window, about 10 cm. (4 inches) high, and made to fit closely to the window-sill, is the best ventilator, but is rarely needed where a wood fire is burning in the room. The upper sash can also be lowered for a few inches if more air is needed.

TOYS.—Remember that a child puts everything that it gets hold into its mouth, so be careful not to allow it to have toys with colors can be soaked off by its saliva, which would perhaps poison it. Toys which are made of woollen materials or of feathers should be avoided particles easily come off them.

SCALES.—The weight of the infant is so important, as I have told in a previous lecture (Division II., Lecture IV., page 97), that I cons properly adjusted scales an important part of the nursery equipm The scales which are usually provided are, as a rule, very inadequate the minute and daily weighing, the results of which are at times of s great assistance to the physician in the management of the infant's f Never hang an infant in anything on a hook to weigh it. Such weights usually, from the continual kicking of the infant, quite incorrect. Do think that the kitchen grocery scale is good enough for the infant. We afford to have incorrect and approximate grocery weights, but cannot aff to apply these methods to the growing infant, with its unstable equilibri The scales should be of a small but solid platform variety, which can placed on a firm table by the tub where the infant is to be bathed, for before the bath. Here are the scales which I am in the habit of usi (Fig. 36, platform scales on table, page 131.)

These scales weigh from four or five grammes (one drachm) up to nin kilogrammes (two hundred pounds). A basket, with a small soft blan lining it, is placed on the platform of the scale, and the naked infant weighed in the basket. The scale is balanced, and the infant immediat taken out of the basket without stopping to read the weight, so as not expose it too long while uncovered. When the infant has been dressed t scale can be read, and the balance-weight minus the weight of the bas and blanket (which can, of course, always be a constant quantity) gi us the exact weight. Weighing with the clothes on I have found a ve unsatisfactory procedure.

BATHING.—The question of the bath is one which you will frequent be asked about, and is indeed of a good deal of importance in the ear months of life. Unless there is some definite contra-indication, I think th an infant should be bathed every morning. The contra-indications are the skin or nails turn blue, or if the infant seems in any way to show sym toms of weakness or lowered vitality after bathing, such as are represent by cold extremities and nose, or an unusually quickened respiration. these cases sponging, merely sufficient for cleanliness, is to be substituted f the bath. The bathing should be done with celerity, the tub being plac on the side of the fireplace opposite from the window, and fronting t latter, so as to avoid draughts and insure a good light, care being take at the same time to protect the infant's eyes from a strong light. I wi now describe to you the manner in which I prefer the details of the bath t be carried out. The nurse sits with her face to the light and has the infan on her lap, wrapped up in a warm blanket, with its feet towards the fireplac and its head in such a position as regards the window as to avoid having too much light in its eyes.

Temperature of Bath.—The water should vary in its temperature somewhat with the age of the infant, but should never be so cold as to cause blueness or cold extremities. We must also be careful not to have the water too hot, as this has sometimes proved to be injurious. Each infant, however, must have the temperature of its bath adapted to its own vitality. This table will, in a general way, guide you in determining which temperature at each age you had better begin with.

TABLE 32.

Temperature of the Bath for Different Ages.

Age.	Centigrade.	Fahrenheit.
At birth	36.6°	98°
During first three or four weeks	35°	95°
One to six months	34°	93.2°
From six to twelve months	32.2°	90°
Twelve to twenty-four months	30°	86°
Then gradually reduce in summer to	26.6°	80°
In the third or fourth year, if possible, reduce to	23.8°	75°

The nurse first washes the face in clear water, keeping the body and limbs wrapped up in a warm blanket. She should gently cleanse the nose, the corners of the eyes, and the external ears. The nose is especially important, for the infant's vitality is easily affected by occluded nares. The face is then wiped with a soft towel. The nurse should then soap, wash off, and dry the scalp. The sponge and water in the other division of the bathing basin are then used for soaping the body and extremities. Especial care should be paid to the folds of the neck, the axillæ, groins, genitals, and anus. The temperature of the water in the basin and bath should be tested from time to time with the bath thermometer until the washing is over. The proper warmth of the water is to be kept by adding when necessary a little hot or cold water from cans within easy reach.

Tub.—The tub, which is preferably made of rubber hung on a simple wooden frame and sufficiently high to prevent needless stooping on the part of the nurse, is placed, as I have arranged this room to show you (Fig. 36), on the nurse's left, at a convenient distance from her chair.

Basin.—In front of the nurse is the double washing basin, which, as you see, is merely a china basin divided into two compartments, and fitted to a wicker stand, also sufficiently high to prevent the nurse from stooping as she uses it. To the right of the nurse is the table, with the scales on one end and the toilet basket on the end towards her.

Soap.—The soap should be white castile, or any kind which is free from irritating elements.

Sponges.—There should be two sponges: one goes in one side of the washing basin, and is for the head and face; the other is to be used in the opposite side of the basin, and is for the body and extremities. The body and limbs having been thoroughly and quickly soaped, the nurse shou gently lower the infant into the clear water in the bath, being careful n to frighten it or drop it. This is not an unnecessary warning. I ha known infants, even in the hands of ordinarily careful mothers, to dropped from the bath or scales, with a resulting permanent injury of t spine or hip. After allowing the infant to kick and splash for a few secon it is taken back into the nurse's lap and carefully dried with a warm s towel. Never soap and wash the infant in the bath, but always on the la

POWDER.—When the skin is perfectly soft, clear, and in a normal co dition, no powder is needed. Where there is any slight irritation, which, times, is liable to occur when the skin has not been kept sufficiently dry, a especially if there is a decided redness in the folds of the skin, as of t neck, axillæ, or groins, this powder can be applied, for which you can wri the following prescription:

PRESCRIPTION 2.

Metric.	Gramma.	*Apothecary.*	
℞ Pulv. zinci oxidi	7 | 5	℞ Pulv. zinci oxidi	ʒ
Pulv. amyli trit.	60 |	Pulv. amyli trit.	℥
M.		M.	

No perfume of any kind should be added to the powder. The infa should be sweet and pure in itself, without accessory odors. In additi to this room arranged to show these various details of nursery routi I have had this diagrammatic picture of the nursery drawn for you to illu trate what I have just said in regard to ventilation and bathing (Fig. 3 page 131).

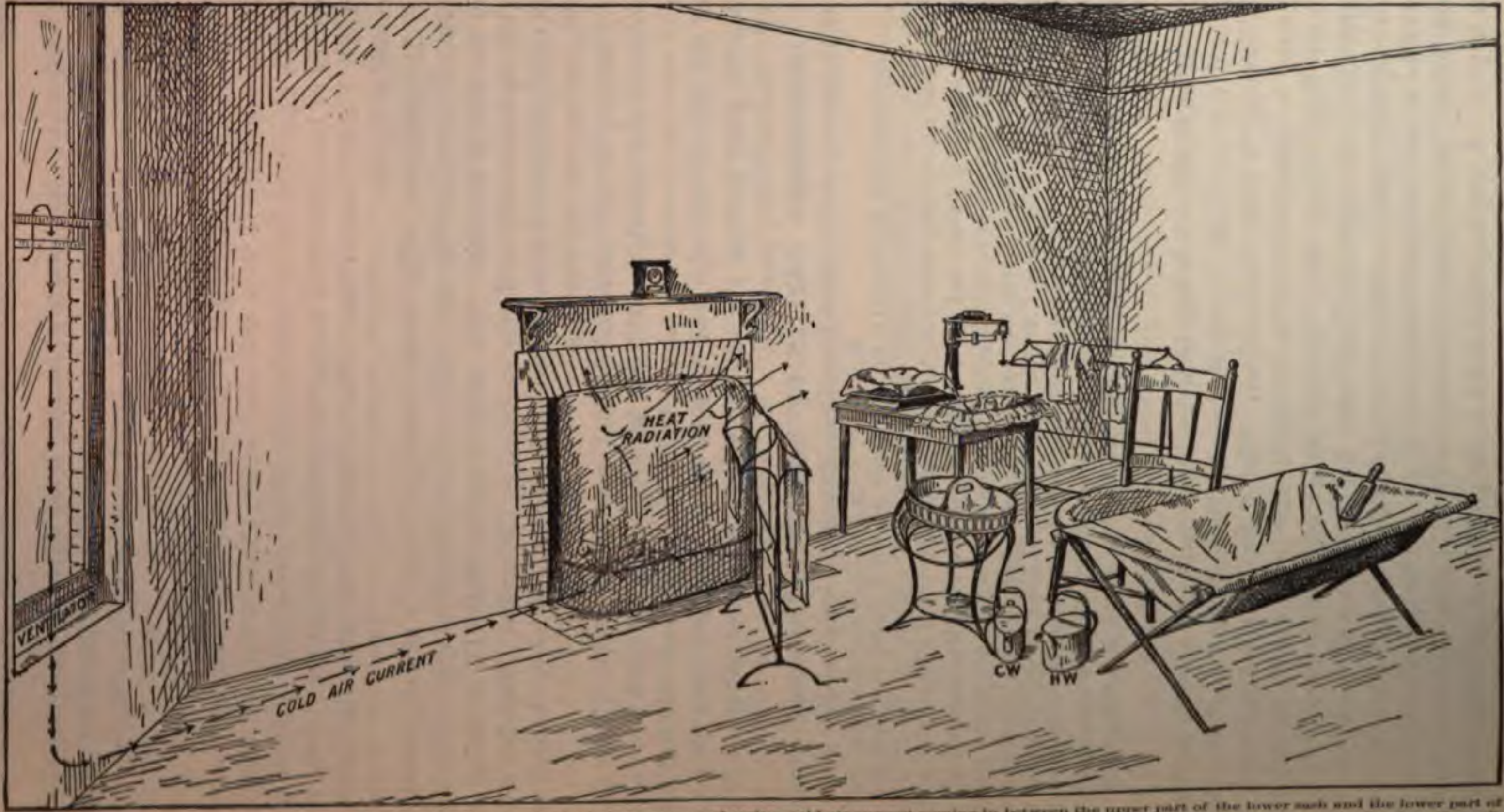
You see the simple wooden *ventilator* under the lower sash of the wi dow, and the arrows marking the entrance of the cold-air current. Whe this current is too strong it can be tempered by pinning a towel across t opening between the upper and the lower sash. The cold-air current pass from the window at a point near the floor directly across the room to t open fireplace. This should at once suggest to the mother that parts of t room, on account of these currents of air which from doors and windo pass over the floor to the fireplace, should be avoided not only for bathi but also for playing on the floor.

A high *fender* covering the entire opening of the fireplace, and fasten so that the older child in playing cannot pull it down, is an important pa of the nursery equipment. It answers two purposes,—one to prevent t sparks from flying out on the child, the other to prevent the child fro falling into the fire. Serious accidents have happened from a lack c proper precaution regarding this apparently self-evident necessity. Tl hot air from the fire radiates in all directions, as is shown by the arrows.

There should be a rack for the towels, which should be kept warm i front of the fire while the infant is being bathed.

The clothes should in like manner be neatly spread out on anoth rack, ready to be put on as soon as the infant has been dried.

Fig. 36.



Nursery with preparations for bath; ventilator fitted to lower sash of window; arrows showing cold-air current coming in between the upper part of the lower sash and the lower part of the upper sash, passing down to within a few inches of the floor and then directly across the room to the open fireplace; arrows showing heat-radiation coming from the fireplace through the high fender; table; scales; toilet-basket; double basin; chair; tub; thermometer; cold- and hot-water cans; towels on rack; clothes on rack.

The bath thermometer is represented at one end of the tub; it is usuall guarded from breaking by a wooden frame, which also allows it to float i the water, and the nurse is thus enabled to see at a glance that the bat water is remaining at the proper temperature.

CLOCK.—There should be a good clock in every nursery.

There are two conditions of the skin that quite commonly occur i infants, especially in their first year, which, although they are abnorm usually come from lack of sufficient care in the nurses, and can therefore spoken of here rather than among the pathological conditions of the ski which I shall mention later. One is *intertrigo,* the other the *seborrh capitis of infants.*

INTERTRIGO.—The former, intertrigo, is merely an exaggerated h peræmic condition of the skin, usually of an erythematous type and occurri in the folds of the skin. This infant (Case 42, Plate III. A, facing pa 112) represents very well this condition in the groins. Napkins soak in urine and allowed to remain for some time without being changed are frequent cause of this condition.

Keeping the skin clean and dry and applying the powder will, as a ru soon cure this intertrigo. At times, however, it becomes much more inten and runs into a pronounced eczema, which is a much more difficult lesio of the skin to deal with and requires special treatment such as I sha describe when showing you cases of eczema in a later lecture (Divisio IX., Lecture XXI., page 470).

SEBORRHŒA CAPITIS OF INFANTS.—The second conditio which also can well be described in this connection, is represented by

This infant (Case 43, Plate III. B), two months old, which I shall now show you. I has, as you see, a collection of crusts of a brownish-yellow color on the top of its hea These crusts are especially thick over the anterior fontanelle. This condition is called t *seborrhœa capitis of infants,* and you will often be asked whether it is safe to remove it. I should never be allowed to collect, and when present it should be gently and graduall removed by first soaking it with warm sweet oil to loosen the crusts, and then washing off with soap and warm water. A little simple ointment should be applied to keep t scalp at this point soft and thus prevent the reaccumulation of the crusts. The whole sca of the infant should be perfectly clean. Seborrhœa capitis is simply a tendency to ove production by the sebaceous glands of their secretion, which, mixed with dirt, produces th condition.

CLOTHING.—It is very important that those who care for the infan should not only clothe it properly but should understand why one metho of clothing is better than another. The surface of the infant's body i greater in proportion to its entire weight than is the case in the older an hence larger human being. Greater surface means that there is a greate opportunity for evaporation, and hence that the smaller subject will cool off more quickly, other conditions being equal, than the larger one. We therefore see at once that much care should be given to the question of warmth i the infant. Any exposure of the body or limbs in either infants or children is unwise. A very important factor in the problem of growth in the infant s perfect freedom of motion for its legs and arms and for the respiratory and abdominal muscles. It should also be thoroughly understood that pressure on any portion of the body or limbs must produce evil results, by displacing organs which should be allowed to have entire freedom of position in their respective cavities.

Too little warmth will do harm, by preventing the proper metabolism of the tissues and thus reducing the animal heat. Too great warmth, on the other hand, by causing inequalities in the circulation, will in like manner be detrimental to the child's growth and vigor. Clothes which bind any part of the infant tightly cannot but press out of their natural position whatever happens to be beneath the point of pressure, whether it be the liver, the intestines, or the toes. The clothes, then, must evidently be warm and loose, and we must bear in mind that loose clothes are warmer than tight ones, from the very fact that they do not interfere with the natural activity of the circulation, and that they give freer play to all the muscles which produce the normal warmth arising from exercise. We must remember that the only way in which the infant can obtain the exercise so much needed for proper growth, and which is so easily obtained by the older child in running about, is by continually moving its legs and arms and thus accelerating the muscular action of its thorax and abdomen.

An important item in the proper management of the infant in its nursery is that it should be irritated as little as possible by unnecessary delay in dressing it after its bath. Useless stitches, buttons, and articles of clothing should be dispensed with, and a method adopted which, while combining the necessities of dress which I have just spoken of, will allow the dressing to be finished before it has time to annoy the infant.

ABDOMINAL BAND.—There is no necessity for using beyond the first two or three weeks the usual flannel band supposed to be so indispensable by the average nurse. Herniæ, whether umbilical or inguinal, cannot be obviated, and in fact may be produced, by undue abdominal pressure.

This form of abdominal band (Fig. 37 A), which is made of light soft flannel, can be smoothly applied over the dressing of the cord and kept in place with moderate pressure by means of safety-pins.

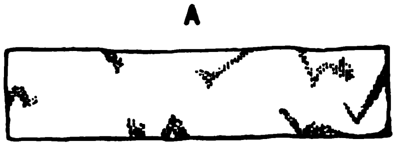
The band can soon be replaced by a somewhat elastic knitted garment (Fig. 37 E, A), half band and half shirt, with shoulder-straps of the same material to hold it in place, and a tab in front to fasten it with a safety-pin to the napkin (Fig. 37 E, B).

This shirt can be made of soft wool or silk, or, as I have recently found, can be knitted in any form or size from half cotton and half silk.

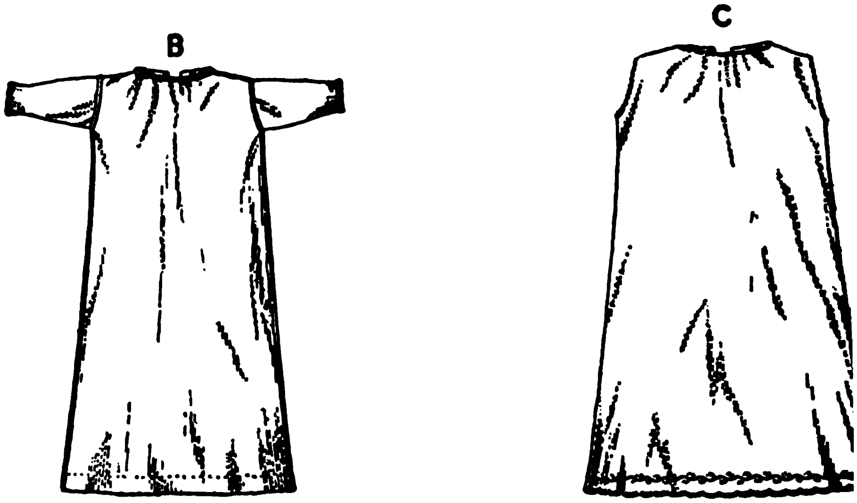
This knit material can also be used for these other undershirts which I have here to show you (Fig. 37 B and Fig. 38 F, page 137). Garments made in this way are the best that I have ever seen. They are warm, soft, and delicate, have no seams, can be washed without shrinking, and retain their elasticity much better than those made from the other materials which I have mentioned.

FIG. 37.

(*Long Clothes.*)

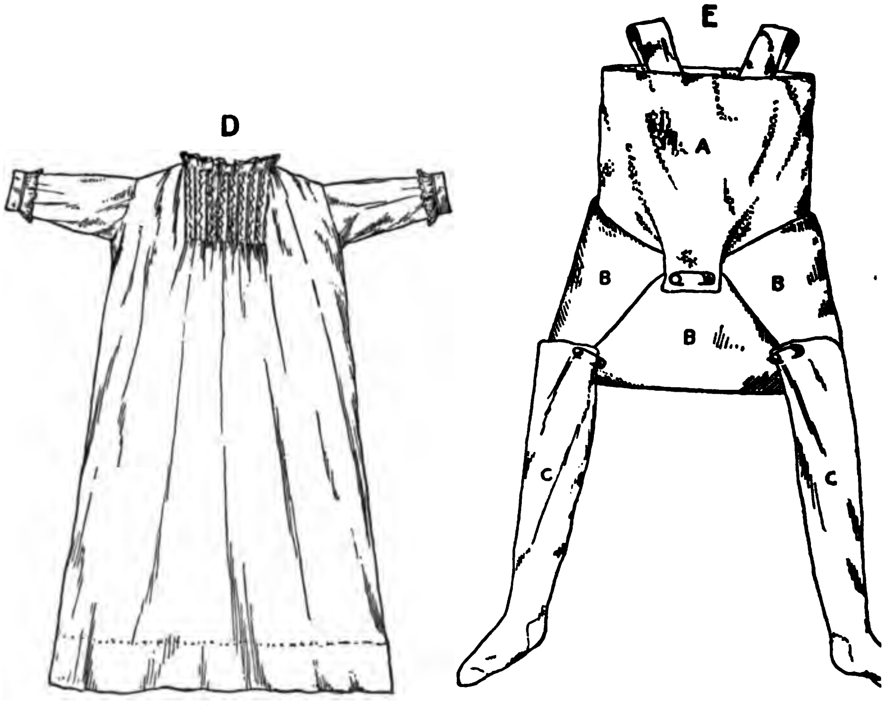


Flannel band for early weeks.



Shirt.

Petticoat.



Dress.

A, knit band; B, napkin; C, stocking.

NAPKINS.—This napkin (Fig. 37 E, B, page 134) is folded and fastened with safety-pins as is customary for keeping it in place. The usual napkin is very cumbersome and heavy, besides being expensive. It can be replaced by rolls of soft absorbent gauze, which absorb the urine from the skin, an important quality in cases where the skin is easily irritated. These napkins can simply be cut from the roll, which is kept in the nursery, and, when removed from the infant after a movement of the bowels, can be burned, thus avoiding the trials resulting from the objections of the nurse or the laundress to washing the napkins. If, however, the mother prefers the regular old-fashioned napkin, small squares of this gauze can be placed in the middle of the napkin, and this will in great measure obviate the more disagreeable part of the napkin-washing, as the square of gauze will hold most of the movement and can at once be burned.

The infant while in long clothes need not have any further covering for its legs, and need have nothing on its feet. There is no particular objection to little knit socks if the mother wishes to use them.

After the nurse has put on the band and the napkin there are left three garments which are usually the clothes needed to complete the infant's outfit of *long clothes.*

These garments are the shirt (Fig. 37 B, page 134), the petticoat (Fig. 37 C), and the dress (Fig. 37 D).

SHIRT (Fig. 37 B).—The shirt is a garment with long sleeves and high neck, cut almost as long as the outside white slip or dress. Unless it is knitted, as I have before described, it is well to have it made of some soft, fine, all-wool material, with the seams finished on the outside to prevent irritation of the skin. It is made to button in the back. A fresh garment of this kind is also sufficient for the infant's dress at night, except during the early weeks of life.

PETTICOAT (Fig. 37 C).—A flannel shirt cut all in one piece, as the shirt is, made of fine flannel with no sleeves and with low neck, represents the petticoat. It should be made large enough to go over the shirt, should be of the same length as the dress, and should also be made to button in the back. The taste of the mother can be gratified by any reasonable degree of embroidery which she may wish to put on this second garment, but the shirt should be perfectly plain.

DRESS (Fig. 37 D).—The outer garment should be made of some soft white material, such as nainsook, should be large enough to go over the shirt and petticoat, should not be starched, and is usually about one yard long from the neck to the bottom of the skirt. It should have high neck and long sleeves, and should button behind.

The advantage of this costume is that it is loose but warm, and that the three pieces which constitute it can be put on together, the infant having to be turned over only once before the clothes are buttoned. The other methods of clothing usually necessitate turning the baby over several times in the process of dressing.

Before the infant has had its bath, these three articles of dress are to arranged one inside of the other, ready to be slipped on all three at on This can be done with great celerity, and the dressing process can thus gone through without the usual accompaniment of irritated cries which a so frequently heard in the nursery, and which are to be deprecated.

When the infant is old enough to have its long clothes changed to sh ones, which is at about the time when it learns to creep, the under-garment c be replaced by a knitted or fine all-wool undershirt with high neck and lo sleeves (Fig. 38 F, page 137) made short, with an additional white pettic in winter if desired. The infant should now also have its feet and le covered with long white wool stockings, which are kept in position by bei pinned to the napkin (Fig. 37 E, B, page 134). When the child begins walk, soft kid shoes should be used with the soles adapted to the natu curves of its feet, as I have explained in a previous lecture (Division I Lecture IV., page 105), and as I shall presently show you (page 139).

Stockings.—A word more in regard to the stockings may not be o of place, and is especially needed in reference to the older child in its thi fourth, and fifth years. It is a mistake to think that if we keep the f and abdomen warm the legs can be left uncovered with impunity. Sh stockings and bare legs, in my opinion, should be abolished, as a proli source of catarrhal conditions. The argument is a poor one that certa children have been known to grow up well and strong with uncovered le or even that our ancestors were in the habit of depriving their children suitable coverings for their necks and arms as well as legs, while they the selves were warmly clothed from head to foot. Our ancestors did and s many things which, to us, convict them of great ignorance. I have sa that the stockings should be white. This is to insure freedom from poiso ous dyes, which at times seriously affect the delicate skin of the young chi Colored stockings are a source of great gratification to lazy nurses and those who wish to lessen the size of their laundry.

There are three garments which are usually put over the shirt and a considered to complete the short clothes. These are the flannel petticoat, t white petticoat, and the dress, and they are to be made large enough to one over the other and thus to be put on all at once.

Flannel Petticoat (Fig. 38 G, page 137).—The inner garm next to the shirt has a flannel skirt, a cotton waist, low neck, no sleev and is fastened with buttons in the back.

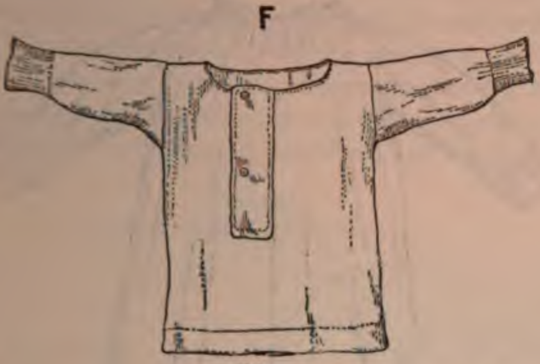
White Petticoat (Fig. 38 H, page 137).—Next to the flannel pet coat comes a garment with a skirt of some soft white material, with a cot waist, low neck, no sleeves, and also buttoned in the back.

Dress (Fig. 38 I, page 137).—Finally, over all the other garme comes the dress, which is made with high neck and long sleeves, and buttoned behind.

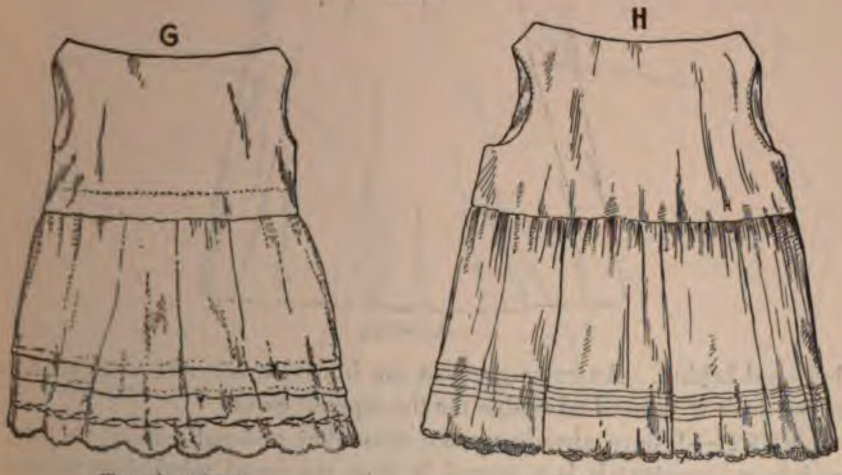
Night-Dress (Fig. 38 J, page 138).—A regular night-dress c now be used, made of soft flannel, with high neck and long sleeves, a

FIG. 38.

(*Short Clothes.*)

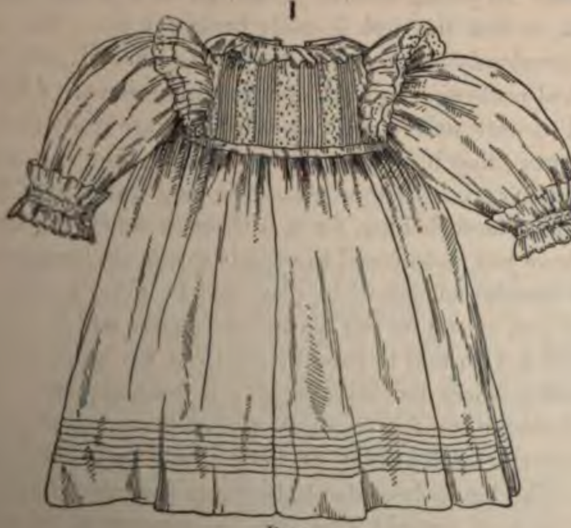


Shirt.



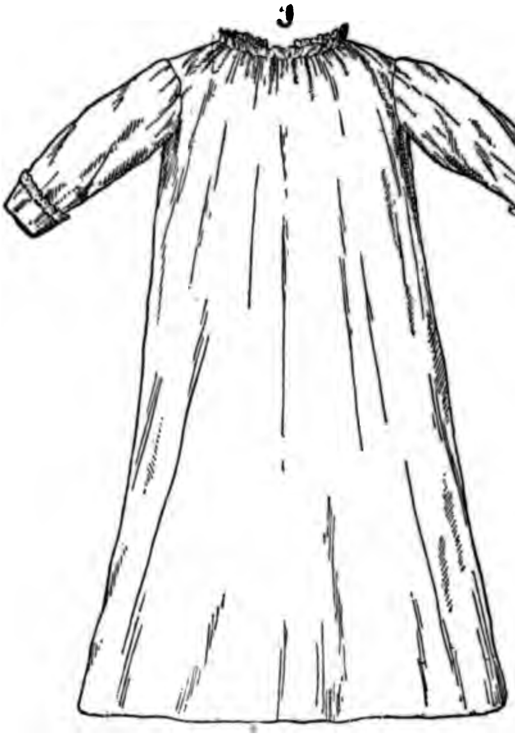
Flannel petticoat.

White petticoat.



Dress.

FIG. 38.



Night-dress.

buttoned behind. An extra garment can in cold weather be worn u
night-dress if deemed advisable for the especial child.

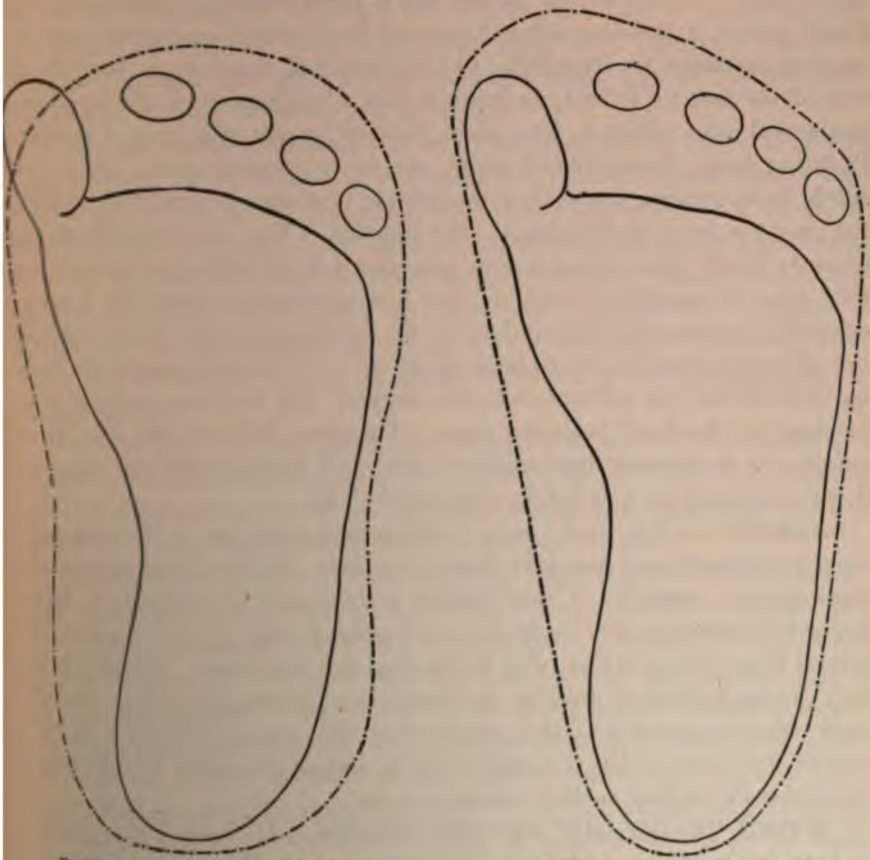
FEET.—I have already spoken somewhat at length about th
and how important it is to guard it from the usual injudicious t
which it receives. In young children, although the foot may be well
it is very weak, so that the arch is easily broken down. The pad
which I have previously referred (Division I., Lecture II., Fig. 13,
is a physiological protection against such breaking down. Childre
not be allowed to walk until some time after they are ready to do s
allowing, of course, that if they insist on walking they can seldo
strained from doing so. As they get older, long walks with their
should, if possible, be forbidden, for it is through these long walks
evils which I have just endeavored to explain to you are brought abo
child will get exercise enough at its play, and in doing so will not
the arch of its foot, or use its feet beyond the degree which nature i
Children should not be told to turn the toes out too much, as this
arch in a position where the muscles give it least support. The
dancing-school master is a fair example of what over-zealous ignora
bined with the respected traditions of the past can do to children's

SHOES.—Children's shoes should be rights and lefts, like those
as the present style of straight shoe gives no support to the arch

very important period of its growth; this, moreover, also tends to push the great toe towards the median line of the foot, and so to cause enfeebling of the muscles which have so much to do with the proper elasticity of the feet.

We should, therefore, have shoes properly adapted to the child's foot,—shoes that will at once be comfortable and leave the feet free to develop and fulfil all their functions. The children's shoes as we find them in the stores have the two sides of each shoe symmetrical and equidistant from the middle line; the right and left are told only from the arrangement of the buttons, and are frequently worn interchangeably. Now, the foot has no such median line on each side of which the parts are equally disposed; and its two edges are very different, as a glance at the soles of this one-and-a-half-year-old child's feet will show (Case 44, Fig. 39).

FIG. 39.
CASE 44. (Natural size, 1½ years.)



Unsuitable shape for sole of child's shoe.
Suitable shape for sole of child's shoe.

We must note especially that the phalanges of the great toe do not naturally point towards the outer border of the foot: such a position, common as it is in the adult, must be considered as an acquired deformity which started, in all probability, with the first pair of leather boots.

I will now show you how contrary to all anatomical rules are the sh which are usually sold for young children. Dr. Dane, to whom I am debted for all these valuable suggestions concerning children's feet and sh has made a tracing of this child's foot to show how the lines of the s ought to run, in order to be adapted to the anatomical conditions. T dotted line around the left-hand tracing shows the shape of the shoe that w provided for the child's foot at the shoe-store.

That this matter of forcing the first toe out of its normal position m bring with it very serious consequences is easily shown: as it inclines agai the terminal phalanx of the second toe, it often crowds it backward, a finally makes it the distressing "hammer toe," which may even requir surgical operation for its relief. On the inside of the foot, as soon as axis of the first toe is bent, we begin to find a bulging out of the metatar phalangeal joint, which in later years, fostered by pair after pair of tight a ill-fitting boots, is capable of giving the most exquisite pain. Still m subtle in its working than this is the trouble that often comes from disabli the great toe from performing its full function. The elasticity of our s depends largely upon our power to press down firmly with the great toe a then raise the weight of the body over it as a support; when this is lost crippling the toe with ill-shaped boots, the muscles not only of the first di but of many adjacent groups begin to atrophy. This soon leaves the int nal arch of the foot without sufficient support, and the long series of w incident to "flat-foot" is started upon. Therefore, for one and all of th reasons, let us demand that children's feet shall have at least the chance develop properly in well-fitting anatomical shoes.

SLEEP.—Infants and young children vary much as to the amount sleep which they need and take during the day. At first they sleep alm continuously, especially if they happen to be somewhat premature. In few weeks, however, they begin to have regular periods of rest, consisting several hours' sleep, at first twice in the day, and later once. The more sl they can be induced to take in the twenty-four hours, the better. As th grow older the amount of sleep which they take grows less, but in the fi four or five years of life it is well to try to induce the child to rest quiet on its bed for at least an hour during the day.

WHEN TO GO OUT OF THE HOUSE.—If the infant happens be born in the winter months and the weather is at all severe, it is better keep it in a well-ventilated nursery, such as I have already described, th to run the risk of its vitality being lowered by exposure to cold. I belie that infants in our Northern climate are exposed to cold far more than th ought to be, and that they need fresh, warm, dry air, rather than the co and often damp air of our winter months. When they are born in a mild climate, or at a warmer season of the year, they can after the first few wee be taken out in their carriages often twice a day. When the infant is five six months old I am in the habit of giving the following directions to t mother as to when she shall send it out. I explain to her that it makes much difference whether the air is damp or dry, and what the rate of the wind may happen to be, as does the number of degrees indicated on the thermometer. If the sun is shining, the air dry, and there is no wind, the infant can without harm go out for an hour in the middle of the day even at a temperature of —6.6° to —3.8° C. (20° to 25° F.). Where, on the contrary, the air is damp, or the rate of the wind is great, it is better for the infant to remain in its nursery, and, at any rate, not to go out, if the temperature is below 0° C. (32° F.). The practice of allowing the infant to sleep in the open air in its carriage in every kind of weather is, I believe, a bad one; but on the days when it is proper for it to go out, such as I have already described, it can without harm sleep in the open air. The nurse should be directed to protect the infant's eyes from the direct rays of the sun, and not to allow a strong wind to blow in its face.

Where the weather has been too severe or damp for the infant to go out in its carriage for some time, it is advisable to have it dressed warmly and wheeled up and down in its nursery with the window open for fifteen or twenty minutes. To avoid too much draught, blankets can be placed over the cracks of the doors and the open fireplace while the infant is breathing the fresh air. The room being far above the ground, the dampness is avoided, and even a considerable velocity of the wind outside the house will in this way be unable to affect the air of the room, and will not make too strong a draught.

Not only should an injudicious administration of cold air be avoided, but extreme care also should be taken in hot weather that the child is not exposed to too great direct heat from the sun, and it should never be kept in a hot atmosphere where currents of fresh air cannot have access.

NURSERY-MAIDS.—The idea that the child should be taken care of by an old, experienced nurse is a vicious one. The experience of nurses, as a rule, is that of ignorance rather than of intelligence. Every mother, as she is presumably more intelligent than the nurse whom she employs, and is surely more interested in the welfare of her child, should personally supervise and unhesitatingly investigate all that the nurse does to the child. The nurse's ideas as to what is needed for the child's hygienic surroundings, food, and clothing can well be dispensed with. The mother, learning from the physician what is best for her child, should give her directions to the nurse and see that these directions are strictly carried out. A nurse between the ages of thirty and forty is preferable to one who is younger or older. She should be neat, healthy, strong, cheerful, gentle, and patient. She should be willing to refer small details of the nursery routine to the mother, as well as those which appear of greater importance. The chief attributes of a good child's nurse, in my opinion, are a desire to obey implicitly the orders which she receives from her mistress, and a temperament in harmony with the sensitive nervous organization of her charge.

MOUTH.—I shall ask you to join me in entering a protest against the way in which the nurse, and in fact almost every one who comes near the

infant, put their fingers into its mouth on all occasions. It would see
though the infant's mouth was considered by those who ought to know b
as something which was especially made to be felt. Infants are much
likely to have various diseases in their mouths than are adults, and prob
one reason for this is that dirt of all kinds is constantly being introd
into them. The fingers should always be thoroughly washed before e
ing the infant's mouth, and yet unwashed fingers are continually feeling
baby's gums to ascertain if a tooth can be found.

The nurse should be instructed that she is never to kiss the infant o
mouth, or allow any one else to. The germs of disease can well be transmi
in this way. It is partly through ignorance of its doing harm, and pa
through timidity on the part of the mother in prohibiting it, that a stop is
at once put to this bad habit of nurses and friends, and it is the physician's
to warn mothers on this apparently trivial but really important questio

In a later lecture, when speaking of tuberculosis (Case 263, page 6
I shall report to you a particular instance where the child was, in all p
ability, infected by the breath of its nurse.

SCHOOL.—I have not a great deal to say about schools. I think, h
ever, that much ignorance of the child's nervous organization is shown
those who should best know how to care for it, at a period of life whe
hygienic surroundings, both mental and physical, are extremely import
No one system is good for all children. I am sure that I have seen
kindergarten system do harm to a number of children, although it seem
suit others. Each child should be gauged for itself, and not be forced
any general system, even if that system has proved to be good for the m
No time is lost, in my opinion, in sending children to school at a somew
later age than is usually supposed to be necessary. I am continually hav
to take little children out of school who are fretful and have loss of appe
Neither parents nor teacher seem to appreciate that the little, actively grow
brain is overtaxed by too great stimulation and is protesting against s
treatment by these general symptoms. Many a child is being dosed w
tonics who merely needs rest from school. The parents should keep the m
rigid supervision over their children while at school, and notice from their
havior whether they are mentally tired. This supervision should not be
to the teachers alone, however interested they may be in their little pup
It seems hardly necessary to state that the school-room should be well v
tilated, and that at stated intervals during the school hours the windo
should be thrown open and the atmosphere of the room completely chang
This should not, however, be done with the children in the room. Att
tion should be paid not only to what the children eat at lunch, but to h
and where the lunch is eaten. A child really needs nothing but dry bre
between its meals, so far as its nutrition and digestion are concerned.

DEFECTS OF POSTURE.—How can we better appreciate the i
portance of following nature as closely as possible in its methods of d
veloping young human beings so as to perfect their various functions to t

fullest extent, than by examining carefully this group of malformed children which I have brought here to show you? (Cases 45, 46, 47, 48, and 49.)

BACK.—The extreme flexibility and slow development of the spine clearly point out to us that nature intends to leave its function in abeyance and bring it into use slowly. If the young infant is allowed to sit or stand at too early an age, the superincumbent weight of the large head tends at once to exaggerate the physiological curves of the spine to a point where they may become pathological. As I have already told you in my lecture on Development, during the first year of life the strength of the spinal column is slowly increasing. Not before the seventh or eighth month has it acquired sufficient rigidity to warrant the child's being allowed to sit up. Artificial methods, therefore, of making the young infant assume a sitting posture at a period of development when the spine should be comparatively straight should be deprecated. I have met with numerous instances where both parents and nurses were anxious to have the infants, at a very early age, sit for quite a long time strapped in small chairs. In like manner the same infants were encouraged to stand and walk long before the apparatus for locomotion was ready for use. We may ask, how many of these individuals developed a spinal curvature in later childhood? Possibly the risk in a perfectly healthy child may be small. We often, however, in early infancy, cannot determine which individual may become rhachitic, and where rhachitis is present the tendency to abnormal curvature is well known.

We should, then, in our advice as to the proper physical management of the early years of life, be guided by our knowledge of the normal average development. Free play for the infant's legs, when lying on its back in bed, should be a point to be noticed and considered, since we know that pressing down the legs causes strain and curvature in the lower spine. Knowing the great lateral flexibility of the infant's spine, we should advise the nurse not to hold the infant continually on one side. Symmetry of development and free opportunity for natural movement should be our aim in the management of the infant from the very earliest period of its existence. Our knowledge of the great flexibility of the growing spine provides us at once with a most valuable means for treating lateral curvature in childhood, and we are continually seeing the benefit of encouraging the promotion of elasticity by moderate pressure and bending. A case which is now under observation in my service at the Infants' Hospital beautifully illustrates the truth of what has just been said.

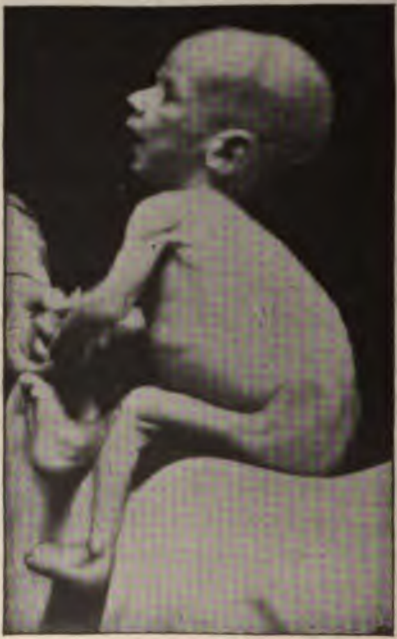
A feeble, rhachitic child (Case 45), nineteen months old, was presented for treatment with a marked lateral curvature in the dorsal region, the convexity being towards the right, combined with decided rotation, following the type of the worst adult cases.

The condition seemed to be purely the result of habit, the patient having been made, when very young, to sit up beyond the limit of endurance of the still undeveloped bones and ligaments. The treatment instituted by Dr. R. W. Lovett, who took charge of the case, was based entirely on the elasticity of the spine, and consisted simply of manipulation and recumbency, resulting in a very great degree of improvement both as to the curvature and the twisting.

Dr. Lovett also tells me that in the surgical out-patient clinic Children's Hospital the improper treatment of the young subject's s in infants, for instance, where they are carried altogether on one well recognized as an important factor in the etiology of rotary curvature. I have seen in this clinic a number of examples of thi and have been much impressed with the important relation which ana knowledge bears to clinical prophylaxis, diagnosis, and treatment.

If you will bear in mind what I told you in speaking of the ossi of the different parts of the spinal column, you will readily understa so long as an infant can be made happy in the prone position, wheth nursery or in its carriage, it will be better for it to be kept in this always protecting the eyes when out in the open air from the stron and the face from the wind. During the first year when it begins t in its carriage its back should be carefully supported by a pillow.

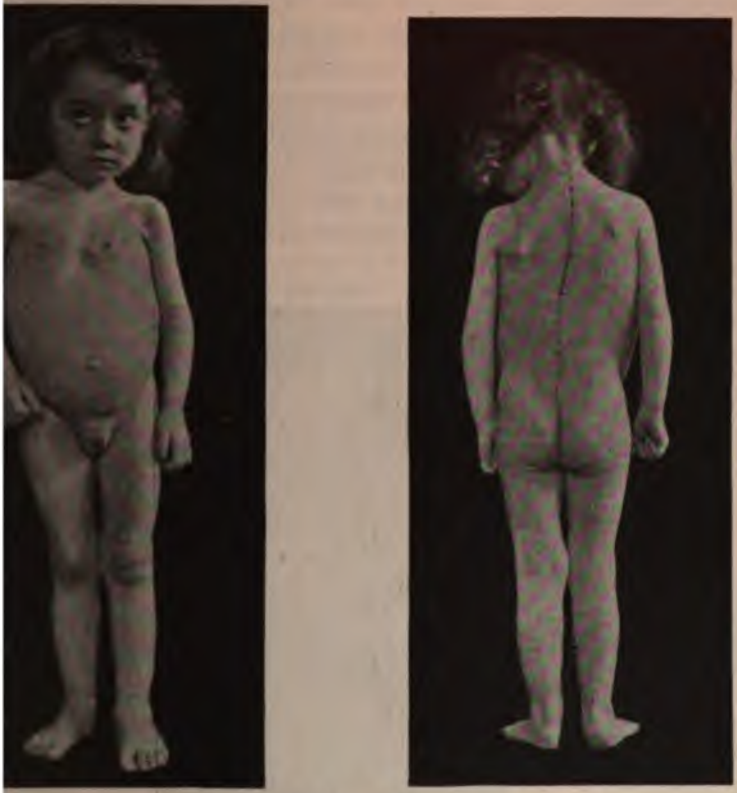
Case 46.



Posterior spinal curvature from sitting too soon.

I have here to show you an infant (Case 46) who is a fitting example of which can be done by encouraging children to sit up before their spinal columns ciently strong. This infant, six months old, has been made to sit in a chair for time, strapped in a position which allowed it to use its arms, but such as to rend possible to fall back and rest itself. You see the exaggerated curve of its back, w responds to that which would be seen normally at birth. Such a curve I hav shown you in Diagram II., Curve 1. If this infant had not been made to sit un developed sufficiently to acquire the physiological curve (Diagram II., Curve 2), not at this age show any spinal curvature. It has, however, through improper t reacquired the posterior curvature (Diagram II., Curve 1) of the early hours of li child grows older, weak undeveloped muscles have a tendency to al and posterior curvatures to be produced. Habit, of course, has with these faulty positions of later childhood.

CASE 47.



Lateral curvature of the spine. Child four and one-half years old.

tle girl (Case 47), aged four and one-half years, shows a lateral curvature, not of the spine, but one which is usually explained as a result of superincumbent ing upon muscles which are unable to support it properly.

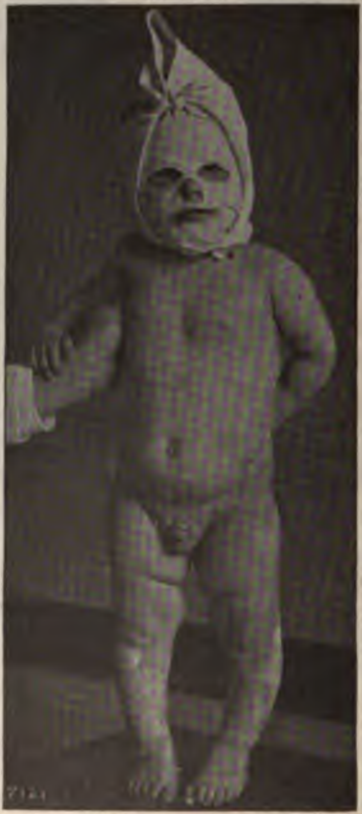
ll notice, on looking at her from behind, the curve which the line of the spinal es to the right in the dorsal region, so different from the straight line of the veloped boy which I showed you in my lecture on Normal Development (Di- Lecture IV., page 124, Case 38). On looking at this same child in front, you will the right shoulder is higher than the left and how the whole thorax is in a dis- ion. These deformities are always more readily recognized by looking at the nt and preferably across the room, as the outline of the chest and hips is much y defined on the anterior aspect of the body than on the posterior. Posteriorly cases even of the slightest lateral curvature at once notice the difference in the tips of the scapulæ. This child stoops, and has what is commonly called lders.

should teach you that in any case of round shoulders lateral should be thought of and carefully eliminated.

Faulty attitudes in sitting and standing play a great *rôle* in pr these curvatures. We must, however, acknowledge that such spina tures have been differently explained on the ground that they are th of a lack of development of all the tissues upon one side of the spine. explanations have also been given ; but in certain individual cases it is sible to formulate any reasonable cause for the curvature.

Legs.—At birth the infant's legs are curved rather than straig have already described to you (Lecture IV., page 118), when I showed infant skeletons at birth and at nineteen months. The natural tend the growth of the legs is to become straight, but if the child is enc to stand and walk too soon, especially if the bones have not been p nourished, the weight of the head and trunk becomes too great to ported by the legs, which curve outward in the form of an ellipse, a co which is called "*bow-legs.*"

Case 48.



Bow-legs. Child three and one-half years old.

This little boy (Case 48), three and one-half years old, has, as I learn, been en by his parents to stand and walk before he was a year old.

His nourishment has also been rather imperfect, but he is not rhachitic. Y the result of this combination of circumstances a decided bowing of both legs. He treated in my ward for facial eczema, which accounts for his rather startling head- shall describe him as a case of eczema in a later lecture.

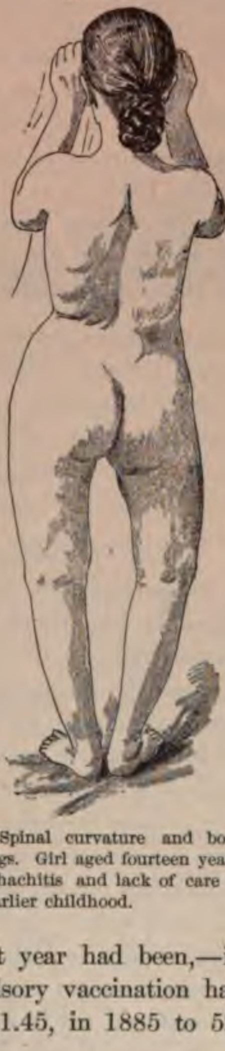
The deformity called "knock-knee," in which the leg at the knee bends in rather than bows out, may occur from simple weakness, but is so rare except when rhachitis is present that it is better to speak of it in connection with that disease.

Finally, I should like you to examine carefully this girl's back (Case 49).

She is fourteen years old, and presents, as you see, a typical case of bow-legs and of lateral curvature.

These conditions are not the representatives of disease of the bone existing now, but are the result of improper nutrition causing the bones to become softened (rhachitic) and easily bent. They are also the outcome of lack of care to correct, by proper gymnastic exercises, weak muscles and bad positions of the trunk. When we consider that such conditions as we see so marked in this girl could have been obviated by proper treatment at an earlier period of childhood, when they were beginning, we can readily understand the importance of careful medical supervision in preventing the acquisition of various deformities.

CASE 49.



Spinal curvature and bow-legs. Girl aged fourteen years. Rhachitis and lack of care in earlier childhood.

VACCINATION.—It is now pretty well accepted throughout the world that the introduction of the vaccine virus into the circulation protects the individual from variola. The physician in general practice, however, is so often questioned as to the advantages in contrast with the dangers of vaccination, that it is particularly advisable in regard to infants and children to know a few facts, especially concerning primary inoculations. According to the careful investigations of McCollom on the history of variola and vaccination, compulsory vaccination was suspended in Zurich, Switzerland, in obedience to popular clamor, in 1883. The deaths from variola, out of one thousand deaths from all causes, for the previous two years and that year had been,—in 1881, 7; in 1882, 0; and in 1883, 8. After compulsory vaccination had been done away with, the deaths rose in 1884 to 11.45, in 1885 to 52, and in the first eight months of 1886 to 85 per 1000.

In this connection it is of interest to note that during the epidemic of variola in Prague in 1888, 76.57 per cent. of the unvaccinated died, while only 10.58 per cent. of the vaccinated succumbed to the disease.

In Boston from 1721 to 1792, a period of seventy-one years, there were three very severe and fatal epidemics of variola, or one in about every twenty-three years. From 1792 to 1892, a period of one hundred years, there had been only one severe epidemic of this disease, and even this could not be compared in severity with those in the last century. The protective power of vaccination is the only possible explanation of this comparative immunity from variola during the last hundred years. In the past ten years th percentage of deaths among the unvaccinated at the Boston Small-Pox Hospital has been 75, while that of the vaccinated has been only 3 per cent. I the past twelve years no person who has been successfully vaccinated withi five years has died of variola, and those who have been attacked by variol have had the disease in a very mild form.

Dr. Barry, in his report of an epidemic of variola at Sheffield, England during 1887 and 1888, gives a very clear idea of the relative frequency of deaths occurring in the vaccinated and in the unvaccinated. I have arrange a table (Table 32 *a*) which illustrates his results very well, and shows the percentages of those who, living in houses invaded by variola, were attacked by the disease, and also how many of these died. It also gives the percentage for all ages, for under ten years and for over ten years.

TABLE 32 *a*.

Individuals living in Houses invaded by Variola.

		(1) All Ages.	(2) Over 10 Years.	(3) Under 10 Years.
Vaccinated.	Attacked . .	23.0 per cent.	28.1 per cent.	7.8 per cent.
	Died	1.1 "	1.4 "	0.1 "
Unvaccinated.	Attacked . .	75.0 "	68.0 "	89.9 "
	Died	37.2 "	37.1 "	38.1 "

The low percentage of children as shown in column (3) is very strikin in comparison with column (2), which represents older individuals and enunciates the importance of revaccination. A glance at the table at once impresses upon us the significance of the difference in the number of deat between the vaccinated and the unvaccinated. We can hardly imagine a other explanation for this great difference in the mortality rate than the supposition that the vaccine virus is highly protective against variola. St more striking are the actual figures recorded as representing very large numbers of cases of variola. These figures show that among the vaccinated, nineteen individuals out of twenty recovered, while of the unvaccinated, fi individuals out of one hundred died. It is not held by the advocates vaccination that one vaccination will protect for a lifetime. On the contra revaccination is just as important as the primary operation. One attack variola does not always protect an individual from a second invasion, a more should not be expected from the operation of vaccination.

Dr. Josef Körösi, Director of the Buda-Pesth Statistical Bureau, has lat published the statistics of 112,000 observations made with reference to t deleterious after-effects of vaccination. As a result of these observatio the author concludes that, even if any slight increase of mortality can charged to vaccination in certain specified diseases, there should be placed its credit a saving of life at least three hundred times as great. Kör stands at the head of living statistical authorities upon vaccination; hen his conclusions are entitled to great respect. He attaches to vaccination greater preventive power than to any other known means or appliance in t whole field of medicine.

Variola has been communicated to the cow by direct implantation of the virus. The efforts to accomplish this were numerous and at first unsatisfactory. The first successful inoculation of this nature was at Berlin, in 1801. Since then many observations have been made in this direction, and the conclusions of those who have carefully studied the subject and are therefore qualified to judge are that:

1. Variola is inoculable on the bovine species when the method of operation is good and when the virus is taken at the proper time.

2. Inoculation of the calf with variola forms a valuable source, in a new direction, for obtaining animal vaccine. This is of great practical value not only for the vaccine institutions of Europe, but also for those of warmer climates, where variola is frequently endemic and where vaccine rapidly deteriorates.

3. Variola inoculated on the calf is transformed after several transmissions into vaccine by its passage through this animal. Dr. Fischer, Director of the Vaccine Institute at Karlsruhe, in Germany, performed at about the time when Körösi was investigating this subject a series of similar experiments, and he arrived at practically the same conclusions. These experiments, according to McCollom, refute the argument of ignorant theorizers, that the vaccine disease cannot protect against variola because there is no connection between these two diseases. Finally, I am authorized by Dr. John H. McCollom, the city physician of Boston, to state that no death from variola has occurred during the last ten years here in Boston of a child who had been vaccinated before it was five years old. With these facts before us, I shall not discuss further the merits and demerits of vaccination, but shall take it for granted that it is well to vaccinate the young infant. The time at which this should be done is, I think, of considerable importance. The infant should be vaccinated early, before it begins to be exposed to the danger of contagion from sources outside of its home. We must, however, remember how low is its vitality at birth, and how readily this vitality is affected by what would be considered trifling conditions for the older child or for the adult. A time should be chosen when the infant is not subject to the other disturbing conditions which naturally arise in the first two years of life, such as weaning and the irritation of the dental periods. If it is found necessary to vaccinate the infant after the sixth or seventh month, or before the twentieth, it should be done in an interdental rather than in a dental period, and not at the time when its food is being changed, or when it is suffering from either slight catarrhal conditions or some definite disease. I prefer to vaccinate the infant when it is four or five months old,—that is, just before the period when the first tooth appears. At this age it has usually become accustomed to its food, its digestion is in equilibrium, and its vitality is much above what it was in the early weeks of its life. By the fifth month also it will usually have developed the outward symptoms of syphilis if it has inherited that disease from its parents. You will thus not be so likely to be blamed for having inoculated with something besides the

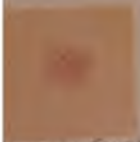
vaccine virus, which might happen if after vaccinating in the early weeks o
life a syphilitic efflorescence should appear. The vaccine virus can be intr
duced into any part of the body through the skin, and according to the fan
of the physician or parents. Girl infants can be vaccinated just below th
knee on the outer side of the leg, so as to avoid having a scar on the arm, t
which women usually object. I am accustomed to vaccinate boys on the oute
side of the upper arm. Whether the vaccination is performed upon the le
or the arm, we should first inquire if the person who is to take care of th
infant is right-handed or left-handed. If the nurse, for instance, is right
handed, she will naturally hold the infant on her left arm, and in this cas
the infant's right arm being towards the nurse, it is better for the vaccina
tion to be on the left arm. The process should be reversed where the nurs
is left-handed, and in this case, for the same reason, it is better to vaccinat
on the right arm or leg. The form of virus which I have been accustome
to use, and which I consider the best, is taken from cows rather than fro
human beings. It should be very carefully prepared by those who ha
made a scientific study of the subject, and, if possible, on farms which a
under State supervision.

I shall now show you the details of vaccination such as I have found
my practice to be the best. It has been pretty well proved by careful o
servation of large numbers of primary vaccinations that those who in la
life contract variola have the disease in a less severe form where in th
primary vaccinations they have been inoculated in three places at o
rather than in two, and in two places at once rather than in one. The g
eral constitutional disturbance also does not appear to be greater where
inoculation has been in two or three places rather than in one. The e
dence therefore seems to be in favor of inoculating in two or three pla
in primary vaccinations. A very small surface is amply sufficient for t
proper introduction of the virus. This pointed ivory quill (Plate IV.)
charged, as you see, with virus, and can be used directly for removing t
epithelium, for exposing the smaller blood-vessels, and for introducing t
virus. I prefer not to use any more instruments than possible, in order
avoid the possible introduction of some foreign substance which might int
fere with the natural course of the vaccine virus and cause unnecessa
inflammation.

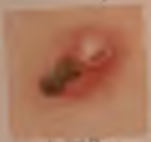
I will now vaccinate before you this infant (Case 50), a girl, four and one-half mo
old, and I have chosen as the place for the introduction of the virus this point just be
the knee on the left leg. I first wash my hands very thoroughly. I then wet
end of a freshly-boiled clean towel in water that has just been boiled. The skin is t
thoroughly rubbed with the hot water, and not dried. This procedure accomplishes
purposes. The first is to remove all dirt or extraneous matter from the spot where
are about to expose the blood-vessels, and thus lessen the danger of septic absorpti
The second is to remove the external layer of the epithelium, which has been softe
by the hot water, and thus render the subsequent scratching less painful and shorter
its duration. I now make a series of short scratches about one-half centimetre (ab
one-fourth inch) long, four or five in number, and in two sets, one crossing the other, u

PLATE IV

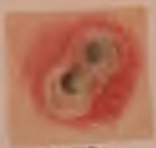
Vaccine Quill



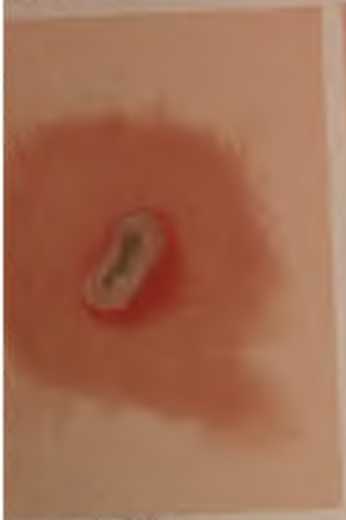
accination Scratch



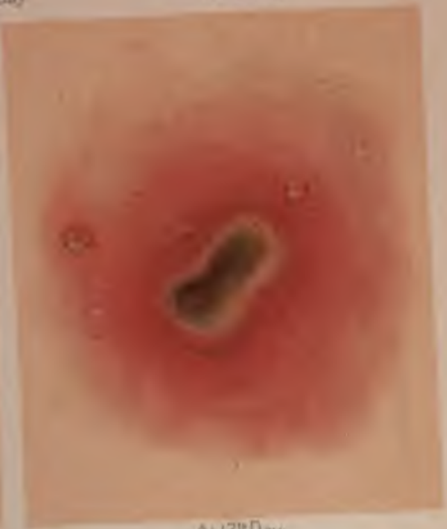
At 5th Day



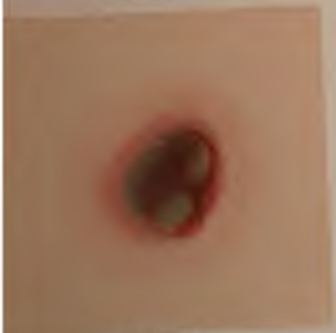
At 8th Day



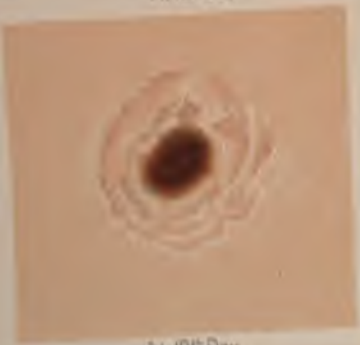
At 10th Day



At 12th Day



At 16th Day



At 19th Day

Scar at 1 year

Scar at 21 years

pithelium is sufficiently removed to show that the blood-vessels are exposed, but a degree that bleeding should take place, for in the latter case the virus may be pre-d from gaining an introduction to the general circulation. (Plate IV.) The point e quill should now be dipped into water which has been freshly boiled (sterilized). The part of the quill which is charged with the virus is then thoroughly rubbed into the nd. The skin should be protected for four or five minutes from contact with anything; this the infant can be bathed or go out as usual. In some cases I have waited until scratch has dried, and then have covered it with a small piece of aseptic cotton which I d at the edges with collodion. After three or four days this cotton can be removed, , unless the subsequent lesion is broken, this measure is an additional safeguard against ction from extraneous matter in the first few days.

Having now shown you the details of the vaccination of this infant, I l also show you a number of cases at different periods of the evolution the vaccine virus. (Plate IV. shows the different stages as they occurred one carefully observed case seen by the artist and myself every day.)

This infant (Case 51) was vaccinated *five days* ago. Nothing especial was noticed until terday, when a little red papule appeared over the side of the vaccination, and to-day see at the end of the vaccination scratch a round clear vesicle, while at the other end re happens to be left a little brown crust. (Plate IV.)

This next child (Case 52) was vaccinated *eight days* ago. You see an irregular-shaped on about ½ cm. (¼ inch) long, and 1 cm. (½ inch) wide, somewhat depressed in the middle, with a clear vesicular border. (Plate IV.)

Here is a case (Case 53) which was vaccinated *ten days* ago. You see that the lesion he last case (Case 52) has now increased in length to 2 cm. (⅞ inch) long, and to a little r 1 cm. (½ inch) wide, but we now have an erythematous condition of the skin forming reola with a diameter of about 2 cm. (⅞ inch), in the middle of which is the lesion just ribed. This areola is a light shade of red, and on its outer border are, as you see, gularly distributed little light red maculæ. (Plate IV.)

This next child (Case 54) was vaccinated *twelve days* ago, and you see very nearly the appearances as have occurred in the last case, except that the areola is very much more se in its red color, and has grown to the size of a circle 3 cm. (1¼ inches) in diameter. of the little maculæ have become vesicles. (Plate IV.)

This child (Case 55) was vaccinated *sixteen days* ago, and in place of the vesicular n with its depressed centre you see that a crust has formed with a narrow line of redness nd it, and on the outer border of this areola the redness is gradually becoming fainter shading off into the normal skin. (Plate IV.)

Finally, here is a child that was vaccinated *nineteen days* ago (Case 56). The crust aller than in the one which I have just shown you at sixteen days, the redness has peared, and where the areola was most pronounced there is slight desquamation. e IV.)

This child and its nurse (Cases 57 and 58) present one of the usual appearances of the ination scar at *one year* and *twenty-one years*. (Plate IV.)

Of course every case of vaccination does not present exactly the same earances. The lesions may differ in shape and size, and one individual be affected more intensely by the virus than another; one may have ompanying severe constitutional symptoms and another have none. The in of lymphatics may be affected as far as the axilla or the groin.

As a rule, the following description represents pretty well the usual rse of the disease. After the vaccination, the skin shows nothing new til the third, fourth, or even fifth day, when a small red point appears. is soon becomes a papule; by the next day a vesicle is developed; about the sixth day this vesicle usually becomes umbilicated, and is surrounded b a faint red zone. By the eighth day the vesicle is fully developed, and b the ninth day the red zone increases rapidly and the vesicle soon becomes pustule. By the eleventh or twelfth day a crust is formed, and this cru falls from about the fourteenth to the twenty-first day, in some cases an ulc being left which heals by another crust being formed, in others the ski remaining intact. From the eighth to the twelfth day there may be a sligh amount of fever and coated tongue, with some loss of appetite, and th glands of the axilla or groin may become enlarged and tender. The sca though perhaps not typical, can usually be recognized by its small depressions (pits) and its location.

In a certain number of cases, instead of this regular progression of th vaccine disease with its characteristic development in a single lesion of th skin, the virus appears to give rise to the original disease cow-pox (vaccinia Vaccinia is characterized by the appearance of papules, vesicles, and pustul of different sizes in different parts of the body and limbs as well as on th face, and running a definite course. I happen to have a case of this kind t show you (Case 59).

This little girl, two years old, was vaccinated ten days ago. You see the characterist lesion of vaccination on the arm. You will notice, however, on the side of the nose, o the forehead, behind one of the ears, and on the chest, a number of papules, umbilicat vesicles, and a few pustules. These lesions evidently represent something more than t usual course of a vaccination. It is, in fact, a case of vaccinia (cow-pox). The cons tutional symptoms are not pronounced in this case, and there is no doubt that the child w make a rapid recovery.

Vaccinia is in my experience a rare disease; its lesions when followi vaccination appear at about the fifth day after the inoculation. At the e of four days, however, minute vesicles can be seen with a magnifying glas

In some cases, instead of the healing of the scratch in a few days, or t formation of the vesicle of a successful vaccination, irregular excrescenc of a fungus-like character may appear. These in all probability have n connection with the true vaccine virus, and are not protective. In additi to the rather rare cases of vaccinia to which I have just alluded, vario efflorescences at times appear on the skin, not only in the neighborhood o the vaccination lesion, but also in other parts of the body. They may b present on the fourth or fifth day, or even later, in the second week, and a probably caused by some reflex connection with the vaccination lesion. The vary considerably in form, but are usually represented by a multiple o papular erythema or an urticaria. It should be remembered, where an un vaccinated child has been exposed to variola, that if you vaccinate it withi forty-eight hours it will probably be protected, and if within five or six da the variola poison will be so modified as to produce only a mild form of th disease. Following the advice of Dr. McCollom, if such a case were pr sented to me I should vaccinate the child in two places. I should then wa for forty-eight hours and repeat the vaccination in a third place.

DIVISION IV.

FEEDING.

LECTURE VI.

THE GENERAL PRINCIPLES UNDERLYING ALL METHODS OF INFANT FEEDING.

JUST as the highest aim of medical art should be directed to the province of preventive medicine, so the highest and most practical branch of preventive medicine should consist of the study of the best means for starting young human beings in life. They should be preserved from the perils which surround the early hours of their existence, and be given strength and vigor to resist the attacks which must inevitably be made on their vitality, and which are greater and more dangerous in inverse proportion to their age. With these objects in view, the preventive medicine of early life becomes pre-eminently the intelligent management of the nutriment which enables young human beings to breathe and grow and live. In fact, it is a proper or an improper nutriment which makes or mars the perfection of the coming generations. The feeding of infants is, then, the subject of all others which should interest and incite to research all who are working in the domain of pediatrics. The subject is a great one, and is worthy of the attention of the greatest minds of the age. The responsibility of discussing so serious a question is a grave one. It should be taken up carefully. It should be dealt with broadly. We must acknowledge for the present that in the status of feeding, as it has existed up to the last few years, the average human breast-fed infant was more likely to live, other conditions being the same, than the infant which was fed by any other method. But we must remember that the latest investigations of this subject show very clearly that it is not human milk as a whole which is pre-eminently good, but that it is a varied combination of the different elements of the milk which makes it the best food during the first year of life. It is our province to study and make use of these elements of the food, which were once somewhat mysterious, but which are now rapidly becoming known through the work of patient and careful investigators.

sources, has been proved to be the nutriment on which the greatest number of human beings live and the least number die.

MAMMARY GLAND.—In regard to the early months of life, a knowledge of the changes which take place in the mammary gland from many causes is of vital importance and must be kept in view. The methods of modifying the milk in the mammary gland, however limited in their scope, should be carefully investigated and adapted to the individual infant according to its age and size and general physical condition. The mammary gland, in its perfect state, uninfluenced by disease or nervous disturbance, or by the improper living of its owner, is a beautifully adapted piece of mechanism constructed for the elaboration and secretion of an animal food. When in equilibrium it represents the highest type of a living machine adapted for a special purpose,—mechanically, physiologically, and economically. When from any cause this sensitive machinery is thown out of equilibrium, its product is at once changed, sometimes slightly, but again to such an extent that the most disastrous consequences may follow when it is taken by the young consumer. The breasts of all mammals are elaborators and producers. They are not storehouses for preserving sustenance until it is needed. They are delicately constructed mills, turning out, when demand is made for it, a product which has been directly formed within their walls from material which has been brought through their portals from various parts of the economy. The breast is a compound racemose gland, lined with glandular epithelium, which forms sugar, fat, and proteids, and these are mixed with water and salts from the blood. The epithelial cells are so finely organized, and so sensitive with their minute nerve connections, that changes of atmosphere, changes in food, the emotions, fatigue, sickness, the catamenia, pregnancy, and many other influences, throw their mechanism out of equilibrium most readily, and change essentially the proportions of their finished product. Then again this delicate mechanism adapts itself to the quantity of its product, elaborating a smaller or a greater supply, according to the demand actually made upon it by the consumer. The same breast will either supply the proper amount of milk demanded for the requirements of the average age or a greater amount for the same age in case of a greater gastric capacity. Again, this machinery is regulated as to the time which it takes to produce the average food required for the different ages, a shorter interval of feeding being needed for the younger infant and a longer one for the older. This fact is made evident by the decided qualitative changes which result when the gland is called upon to produce its product at improper intervals. Thus, a prolonged interval lessens the solid constituents in their proportion to the water, while a shortened interval, by exciting the epithelial cells to frequent work, over-stimulates them, with the result of increasing the solids in their proportion to the water. In fact, too long intervals produce a product too dilute, while too short intervals produce a product too concentrated. The analyses of large numbers of specimens of human milk at different periods of lactation show us that not only do the constituents vary from month to month, and even from day day, but that this variation takes place as much in the early as in the la periods of lactation. We are not warranted, therefore, in assuming that t milk grows stronger as its age increases, provided that it still remains normal equilibrium. The mammary gland acts both as a secretory and an excretory organ, so that it cannot be classed as a metabolic tissue in t limited meaning which we now attach to these words. Yet the metab phenomena (Foster) giving rise to the secretion of milk are so marked, distinct, and have so many analogies with the metabolism which we meet adipose tissue, that we must look upon the mamma chiefly as a secret organ. This, however, is only within certain limits, for we know that times foreign elements may be excreted from the gland. This at on suggests the interesting question as to when the mammary gland is m likely to have what we might call its normal secretory function int fered with and to assume temporarily the function of an excretory org This seems to occur both before the gland has attained its equipoise, during the colostrum period, and later when any of the above-mention influences occur which affect the general mechanism of the gland. these instances we find the colostrum reappearing in the milk. The fore in the beginning of lactation, during lactation when normal metab lism is interfered with, and as lactation draws to a close, we have ana gous conditions in which the mammary gland instead of being a norm secretory organ becomes abnormal and more or less an excretory org During these periods of abnormal gland excretion we must remember t drugs can be eliminated by the milk more freely than when the gland is equipoise. We assume, therefore, that the mamma during that early peri of lactation, which essentially represents a condition of lack of equipoise, h a double function, partly secretory, partly excretory. The greater the exc tory function of the gland is at any time in proportion to the secretory, t more abnormal will be the finished product; while the nearer the gla approaches to a purely secretory organ, the more perfect and normal w be its product. The mechanism of the mammary gland is therefore in most perfect condition after the colostrum period has ceased, and at a ti when the general organism, both physical and mental, is freed from cau detrimental to a perfect metabolism.

General principles are vital in their importance when we come to stu the subject of feeding in all its phases, whether the nutriment to be pr vided for the infant is to come directly from its mother, a wet-nurse, an animal, or indirectly from the product of the mammary gland. Th principles are, (1) That nature throughout all ages has clearly indicated means of natural selection what the source of supply should be; that that the mother should during some early period of its life supply food t her offspring from her mammary glands. (2) That where, owing to d ease, over-civilization, or any causes which prevent the offspring fr receiving its sustenance directly from the maternal mammæ, some nut ment must be substituted which will correspond as closely as possible to the natural food-supply. (3) That this substitution can be obtained most exactly through the product of the mammary gland of another woman. (4) That, owing to the strong analogy between human beings and all animals which suckle their young, we should in our study of copying good human milk make use not only of what we have learned from human beings, but also of what is known of lactation as it occurs in animals. This entails acquiring a knowledge of the investigations and experience of those who have studied commercially the breeding of animals and their food, and the production and modification of their milk.

I have already explained to you the conditions which are normally found in early life from birth to puberty. All these conditions representing the various stages of a physiological development must be thoroughly understood and remembered if you wish to appreciate the many difficulties which are to be dealt with in a practical investigation of infant feeding. In my next lecture I shall begin the consideration of feeding during the first twelve months of life. This I have designated as "*The First Nutritive Period.*"

LECTURE VII.

THE FIRST NUTRITIVE PERIOD.

I. MATERNAL FEEDING—II. DIRECT SUBSTITUTE FEEDING—III. INDIRECT SUB TUTE FEEDING.

As in my lecture on Development I endeavored to emphasize only th facts which would be of practical use to you from a clinical stand-point, in dealing with the subject of nutrition, I shall not attempt to discuss finer and more intricate questions of physiology and chemistry. While pecting to receive great aid from the physiological chemistry of the fut we must not allow this fascinating branch of our art prematurely to set as evident clinical truths which for years have emanated from nursery pract and have proved to be of great value in it. The nutrition of young hum beings may be divided into three distinct nutritive periods, corresponding the degree of their development. The first period consists of the first ten twelve months of life. The second period comprises the second and th years, and the third period the remaining years of childhood. The scie of feeding depends almost exclusively, in addition to the general principles which I have already spoken, on the knowledge of what elements of the f are required by the growing tissues in these nutritive periods, and also the time when the various digestive functions are ready and able to disp of them. I shall therefore begin with the discussion of the first nutrit period, which is essentially the only one where human milk need be cons ered. I have already referred to the marked analogy which exists betw the nutrition of human beings and other mammals, and the necessity of derstanding the lactation of animals when we endeavor to explain that human beings. In order to acquire this knowledge I have received much aid from Mr. G. E. Gordon that I wish to acknowledge my indebt ness to him for placing at my disposal the fruits of his many years of stu and practical observation on the feeding, breeding, and lactation of cows.

The first nutritive period, which for purposes of simplicity I have ar trarily made to represent the first twelve months of life, is obviously, fr what I have already told you, the most important one of the three. In t period the infant may be fed by a number of methods. It may be nurs by its mother, or a wet-nurse, or an animal, or it may be nourished by fo especially prepared from the milk of one of these.

I. MATERNAL FEEDING.—The first of these methods, the *matern* is so far superior to any other which has ever been known that I sh assume that it is the best, and the one from which in almost every particul all others should be copied.

The relative advantage of the milk-supply received from a primipara or a multipara is not of so much importance in the case of mothers as in that of wet-nurses. I shall therefore defer what I have to say on this subject until I speak of the latter, merely reminding you of what I have told you concerning them in a previous lecture (Lecture IV., page 100).

Normal Maternal Conditions.—The assumption that the maternal is when normal the ideal source of infant food-supply presupposes many important conditions concerning the mother and the function of her mammary glands. She should be strong and healthy, of an even, happy temperament, desirous of nursing her infant, and have time to devote herself to this special duty during the whole period of her lactation. She should have a sufficient supply of milk, and should be willing to regulate her diet, her exercise, and her sleep according to the rules which will best fit her for her task. These may be said to be the ideal conditions which we endeavor to obtain for an infant which is to be nursed under the most favorable circumstances. It is true that women who are far from vigorous nurse their infants with seemingly good results, and that a frail, delicate-looking mother may have an abundant supply of good milk. These are exceptions, however, which make the principles just stated all the more true. We must have some general principles to guide us in our endeavor to perfect the nutriment of infants as a class, or we shall surely in many instances do serious harm to the individual.

Contra-Indications to Maternal Feeding.—With few exceptions, the mothers who have uncontrollable temperaments, who are unhappy, who are unwilling to nurse their infants, who are hurried in the details of their life, who are irregular in their periods of rest and in their diet and exercise, are unfit to act as the source of food-supply for their infants. Even if their milk happens to be sufficient in quantity, it will probably be so changeable in quality as to be a source of discomfort and even of danger rather than the best nutriment for their offspring. It is far better for such mothers not to attempt to nurse, but to adopt some other method of feeding. It is of still greater importance that mothers who are suffering from some chronic disease, or one which their infants may directly inherit, should give up all thoughts of nursing. Where there is no question of disease in the mother, it is our duty to investigate, and, if possible, to counteract the other contra-indications to nursing, often only caused through ignorance of what to us seem very simple truths, but which to the young mother are enveloped in mystery. There is, then, a double necessity for studying in the closest detail the conditions which constitute a normal lactation. First, that, knowing what is normal, we should at once recognize what is abnormal, and, by the intelligent use of our knowledge, render possible an apparently unsuccessful attempt to nurse. Second, that we may know exactly on what the normal and vital conditions of a successful nursing depend, in order that we may understand what we should copy in substitute feeding.

It is these normal and vital conditions which I shall endeavor to to you, and which, for the reasons just stated, you must not look trivial, for I have found them of the greatest value both in the ment of human-breast milk and in the regulation of infant feeding *maternal*, then, being the ideal method, I shall begin by showing actual illustration of this method.

NURSING MOTHER.—Here is a young mother (Case 60), pe healthy and strong, in the act of nursing an infant.

CASES 60 AND 61.

Infant 5 months old. Weight, 9800 grammes (about 21½ pounds). Birth-weight, 4500 grammes (about 10 pounds).

The infant (Case 61) was healthy at birth, and has grown continuously, wit weekly gains of about 250 grammes (about ½ pound). Its birth-weight was 4500 (about 10 pounds), and it now weighs 9800 grammes (about 21½ pounds). It is a f men of normal development produced by human milk, and is so large that it has dressed in short clothes some months earlier than is usual. You will observe that mal nutrition depends in great measure on its birth-weight rather than on any ph gain which it has made from month to month. You will understand this by ref what I have said regarding weight in my lecture on Development (Lecture IV., p where I have stated that the birth-weight is normally doubled in the first five m life. I would also call your attention to what I shall speak of more in detail late is not necessarily a milk of unusually good percentage which has produced this pr increase in weight. It is merely a good milk adapted to the especial need of this infant, and it might not at all suit a number of other equally healthy infants. as you will soon understand, merely declares that practically there is no one com of the elements in human milk which is the best for all infants, but that na des a number of combinations all equally good provided that they are suited to the dividual.

You see that the natural method of feeding is by sucking. The infant should be placed a comfortable position in its mother's arms, with its head and back supported. It should made at once to understand that it is to begin its meal as soon as the breast is offered to t, and continue, with, of course, breathing-spells, until the meal is finished. The mother hould herself preferably be sitting, as she can thus best manage and control the infant if it inclined to be restless.

Now notice more closely the method by which this infant is obtaining its food. The formation of its lips and buccal cavity are adapted to the mechanism of suction, and you see with what ease and perfect tranquillity it is receiving its food. The breast is so organized that it provides a fresh supply of food at the required intervals. It prevents fermentation of the food before it enters the infant's mouth, while at the same time the suction incites to action both the necessary digestive fluids of the infant and the function of the gland itself. The gland avoids a vacuum by collapsing as it is gradually emptied, and allows the food to flow continuously, thus obviating the tendency to exhaustion of the infant and prolongation of the nursing-time which necessarily accompanies a retarded flow of the milk. Finally, the breast is practically self-regulated as to the amount which it is required to provide according to the infant's age. A healthy infant should empty the breast with easy and uninterrupted sucking in about fifteen to twenty minutes.

NIPPLES.—In certain cases the mother's nipple is so small or depressed that it is a source of much annoyance to the infant, and at times this interferes so seriously with its obtaining the proper food-supply that its nutrition suffers, and some other method than nursing has to be substituted. It is here that the ingenuity of the physician is taxed to its utmost. Every kind of device may fail, and it is necessary patiently to try one after the other before deciding to give up the nursing. Nipple-shields should be experimented with, and will sometimes obviate the difficulty. We should, however, always impress upon the mother the fact that the value of her milk as a food may be entirely destroyed if foreign elements are allowed to enter with it into her infant's mouth. This simply means extreme cleanliness of the glass shield and rubber nipple. In a few cases where I could absolutely trust the mother on account of her being able to appreciate intelligently the details of my instructions and the danger of not carrying them out, I have allowed, for a short time, the use of rubber tubing connected with the nipple-shield in place of the direct attachment of the rubber nipple. When this is done, however, fresh tubing should be used every day, as it is extremely difficult to cleanse the interior of a rubber tube as one can the rubber nipple, which can be turned inside out and scrubbed. I would, however, decidedly state that I consider, except in these rare instances, the use of rubber tubing to be an abomination which should never be tolerated under other circumstances, and especially in feeding from the bottle, where its use is absolutely unnecessary.

Where the nipples are very tender and cause great discomfort to the mother during the nursing, their condition frequently becomes so serious an obstacle as to prevent nursing altogether. This change, however, should not be thought of for at least several days, or until it is absolutely certain that the exquisite pain is more than the mother is willing or able to endure.

It is often the case that after a little time of the greatest suffering fro tender or excoriated nipples the whole difficulty will pass away and t mother be able to nurse her infant with comfort. I know of no especi treatment which will prevent this condition of the nipples from arising, n of any way by which it can be quickly cured. Bathing with cold wat before and after the nursing, and thus keeping the tissues in a healthy co dition, appears to be as successful as the application of any medicaments.

Mastitis.—Another trouble which may arise during the nursing peri is a disturbance of the mammary gland itself, sometimes amounting mere to a stasis in its milk production, but again going on to inflammation. T latter is a serious matter, and should at once be placed in the hands of skilful surgeon. The former condition requires great care in its manag ment. Gentle massage from the periphery of the gland towards the nipp amounting in fact to merely a delicate stroking with the ends of the finge is an important part of the treatment. The breast should be withheld fr the infant for about twenty-four hours, and the milk from time to ti drawn in small quantities by means of a properly-adjusted breast-pum The breast should also be carefully supported by a swathe. If these mea ures are begun as soon as there are any indications of disturbance in t breast, these abnormal conditions soon disappear. The indications referr to consist in the appearance of hard swellings in place of the usual s elastic condition of the milk glands. These swellings may occur witho any especial pain, but on palpation they are usually tender to a greater less degree.

Breast-Pump.—In regard to the use of the breast-pump there is a gre difference of opinion, but I have very decided views on this subject, a believe that those who have opposed its use have been influenced to a gre degree by what they have seen in their hospital practice, and also by t views of others who have, in like manner, met with unfortunate results lying-in hospitals. It is well known that all inflammatory conditions abo the breast are more likely to occur in hospitals than under conditions whe the woman is less likely to be exposed to pathogenic organisms. This shou be taken into account when we are deciding whether or not to use a brea pump. In my experience, acquired in a great degree from my priva practice, where every precaution in regard to cleanliness, fresh air, a good ventilation could be obtained, I have never met with any bad resu from the use of the pump.

In regard to the relation of micrococci to inflammation of the brea according to Zweifel and Döderlein there are in mastitis two varieties organisms, the staphylococcus pyogenes aureus and the streptococcus py genes, but never the staphylococcus pyogenes albus. They admit that oth varieties may perhaps be found on closer investigation, but at the same tim they consider it striking that in all their cases there were never any local general symptoms caused by the staphylococcus pyogenes albus, althoug that they were virulent was proved by their inoculation of mice. The s not much doubt that these pathogenic organisms gain access to the gland hrough the nipple.

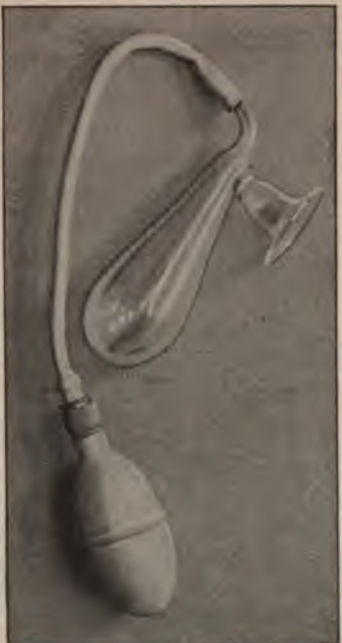
I have already said that the infant may not be able to hold the nipple with sufficient firmness on account of some abnormal condition of the nipple itself.

Under certain circumstances, even where the nipple is well formed, the infant has insufficient suction-power to obtain its food, though the food itself may be perfectly adapted to its digestion. In these cases we often find that it cannot or will not be induced to obtain its food through a shield and rubber nipple or from rubber tubing. The breast-pump may then become of great value, as in the case of an infant that was under my care during the hot weather of June, July, and August.

This infant (Case 62) was seven months old, and was dying of starvation, as I had not been able to prepare for it a food which it could digest and thrive on. (This was before milk laboratories were established.) It was totally unable to nurse, although the breast-milk was a good one and agreed with it perfectly when it was introduced into its mouth with a spoon. The milk was pumped from the breasts at regular intervals and given to it from a bottle for over three months with the greatest success, the infant thriving, and at the end of that time being in a perfectly healthy condition.

This case shows the exceptional but at times very great value of the breast-pump.

Fig. 40.



Breast-pump.

As I shall later have occasion to speak of the use of the pump in various instances, not only for relieving the breast but for obtaining milk for purposes of analysis, I will show you the form of pump which I am in the habit of using.

The apparatus should be one which can be carefully cleansed, and should, therefore, preferably be made of glass. No one special pump will, in all probability, suit every case, and it is of importance that you should use the greatest care in adapting the pump to the individual. As I have stated, however, this is the one which in most cases I have found to be suitable. When applied to the woman it should cause little or no pain or discomfort. You see that the part which is adapted to the nipple is like an ordinary nipple-shield. This is attached to a glass bulb, into which the milk falls as it is drawn from the breast. The mechanism is very simple. A vacuum can be produced in the glass bulb by means of suction through a rubber tube attached to a rubber bulb with its valve working backward. This is a far better method for producing suction than the direct application of the mouth to the end of the rubber tube, which under all circumstances should, if possible, be discountenanced.

MILK.—The product of the mammary gland of all mammals is essentially the same. It is composed of elements which in an individual milk resemble the corresponding elements in all the others. Although the attempt has long been made, and may in the future prove to be successful, to distinguish between the component parts of each element, yet at present we must, with few exceptions, accept each element as a whole and as alike both in human beings and in animals. This must practically be done until the analytical and physiological chemists provide us with much more exact data on which we can depend in elaborating our methods of infant feeding. It is the combination of the various elements of the mammary gland which makes the resulting product characteristic of the special mammal, and it is therefore best first to describe this uniform product as a whole and then to study it as it occurs in its various combinations, whether in human beings or in animals.

In addition to the general principles which I explained to you in my last lecture, a number of physiological facts regarding milk as a whole become of great interest and of the utmost importance when we attempt to modify or change the product of the mammary gland.

FORMATION.—Bunge's investigations on the comparison of tissues show that the mammary gland abstracts from the blood very nearly the amount of salts found in the tissues. According to Foster, whose remarks on this subject I quote freely, milk is the result of the activity of certain of the protoplasmic cells occurring in the epithelium of the mammary gland. So far as we know, the fat is formed in the cell through a metabolic action of the protoplasm. Microscopically, the fat can be seen to be gathered in the epithelial cell in the same way as in a fat-cell of the adipose tissue, and to be discharged into the channels of the gland either by a breaking up of the cells or by a contractile extrusion very similar to that which takes place when an amœba ejects its digested food. This observation is thoroughly supported by other facts. Thus, the quantity of fat present in the milk is directly increased by proteid food, but is not increased by fatty food; on the contrary, it is diminished. In fact, proteid foods increase and fatty foods diminish the metabolism of the body. A bitch fed on meat for a given period gave off more fat in her milk than she could possibly have taken in her food, and that, too, while she was gaining in weight, so that she could not have supplied the mammary gland with fat at the expense of fat previously existing in her body. We also have evidence that the caseinogen is, like the fat, formed in the gland itself. When milk is kept at 35° C. (95° F.) outside of the body the caseinogen is increased at the expense of the albumin. When the action of the cell is imperfect, as at the beginning and end of lactation, the albumin is in excess of the caseinogen; but so long as the cell possesses its proper activity, the formation of caseinogen becomes prominent. That the milk-sugar also is formed in and by the protoplasm of the cell is indiated by the fact that the sugar is not dependent on a carbohydrate food, nd is maintained in abundance in the milk of carnivora when these are ed exclusively on meat as free as possible from any kind of sugar or glycogen. We thus have evidence in the mammary gland of the formaion, by the direct metabolic activity of the secreting cell, of the represenatives of the three great classes of food-stuffs, proteids, fats, and carbohydrates, out of the comprehensive substance protoplasm.

NERVOUS DISTURBANCES AFFECTING THE MILK.—The secretion and ejection of milk are very evidently under the control of the nervous system, which produces marked changes in both the quantity and the quality of the mammary product in proportion to the relative nervous excitability of the special mammal. Women are especially sensitive in this respect, and when living in the midst of our modern civilization, so harmful for the production of good nursing, present an exaggerated example of disturbance of the equipoise of the mammary gland. The chemistry of the equipoise and lack of equipoise of the mammary product appears to be closely connected with its proteid element. This element is known to be a compound one and decidedly complex, but for purposes of illustration we can safely say that the word albuminoid or proteid is a general term, which includes caseinogen and albumin; also that these factors of the complete whole vary in their proportions to each other according as the mammary function is or is not in a state of equipoise. In the colostrum period, and probably in the analogous periods represented by the abnormal conditions already spoken of, the albumin is in excess in proportion to the caseinogen, while as the equipoise of the function becomes more complete the caseinogen is increased proportionately to the albumin. Probably at the end of lactation, as in the beginning, we shall find this same condition of richness of albumin and deficiency of caseinogen. This increase of the albumin at the expense of the caseinogen explains what I have previously told you concerning the excretory function of the gland at times becoming more prominent than the secretory.

These nervous disturbances, however, may also cause, as I shall describe to you later, an over-production of the total proteids, as shown by their percentages. In some cases also the fat has been found to be much reduced in its total percentage. Instances of this have arisen where, as observed by Zakowski and quoted by Jacobi, seasons of fasting with their accompanying excitement of the emotions have induced such a disturbance of the equilibrium of the milk that the fat has been found to be decreased to the low percentage of 0.88, with the result that the infant has become sick and given evidence of impaired nutrition. These same nervous influences in all probability have to a greater or less degree their analogy in the milk-product of all mammals.

CONSTITUENTS AND PROPERTIES.—Milk consists of a large amount of water and a comparatively small amount of solids. The solid constituents comprise, in varying proportions, certain *proteid* elements, *fat*, *sugar*, and *mineral matter*.

"Milk is an emulsion, the fats existing in the form of globules o varying but usually minute size. It is this condition of the fat whic gives milk its peculiar white color." (Foster.)

The *specific gravity*, *reaction*, and other *properties* can best be spoken o when describing the milk of an especial mammal.

The closely analogous conditions, however, of the earliest days of lact tion in the woman and in the cow lead me to describe in my general remark on milk the *colostrum period* of these two mammals.

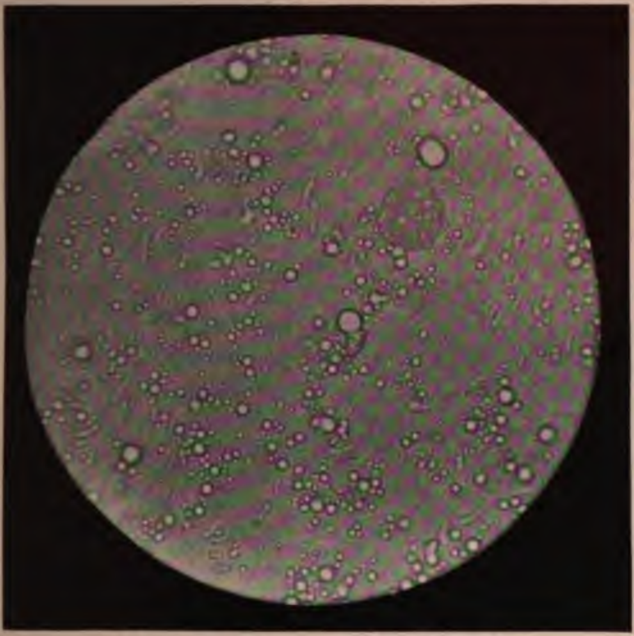
COLOSTRUM.—During the early days of lactation the mammary glan secretes a somewhat different fluid from that which is produced by it late The milk at this period is called *colostrum*, and the period is called tl *colostrum period*, on account of certain elements called *colostrum corpuscl* which are present in the milk. I have already spoken of the connectio between loss in weight of the infant (Lecture IV., page 100) and tl presence of colostrum in the milk; also of the excess of albumin over tl caseinogen in colostrum milk.

Colostrum is supposed to have a somewhat laxative effect, and in th way to aid in displacing the meconium. Whether it is of any especi advantage to the infant is a question of much doubt, for it appears to m and it will, I think, be understood by you, from what I have already tol you regarding the mammary gland, that the appearance of these colostru corpuscles is simply an indication that the equilibrium of the mamma gland has not been established, or has been disturbed, and that it is an ev dence of disease rather than of health. It may be that the not infreque disturbance of the infant's digestion, amounting at times to acute conditio of fermentation, is produced by an exaggerated abnormal condition occu ring in the colostrum period as well as by the return of the colostrum irregular periods. This may be the reason why numbers of infants are mac sick by their mothers' milk at an early period of lactation.

The analysis of colostrum milk is something which as yet has not bee thoroughly studied. Whether it will be of much importance or not is question to be determined in the future. It may, however, prove to l of considerable use to us when we attempt to prepare a substitute food fo the early days of life. It is possible that a combination of elements corr sponding in their percentages to those which are shown by the analys of the colostrum, but free from the colostrum corpuscles, may be foun to suit best the infant's digestive function at this early period of its lif I have under one of these microscopes a drop of colostrum milk (Fi 41) taken from a cow, and under the other a drop (Fig. 42) taken fro a woman.

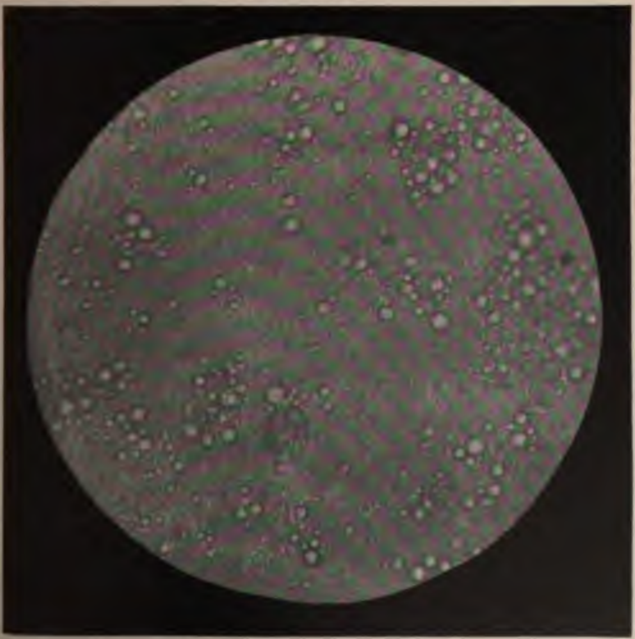
In addition to the fat-globules of various sizes which you see floating i this film of milk you will notice the large cells which occasionally appear i the field. These are the colostrum corpuscles. The one to the right abov the centre in the cow's milk appears to be about one-third larger than th one to the left and below the centre in the woman's milk.

FIG. 41.



Colostrum milk from cow. (Photo-micrograph.)

FIG. 42.



Colostrum milk from woman. (Photo-micrograph.)

An analysis (Analysis 6) made by Dr. Harrington of this cow's milk strum gave the following results:

ANALYSIS 6.

Fat	1.71
Milk-sugar	4.90
Proteids	1.72
Ash	0.79
Total solids	9.12
Water	90.88
	100.00

This table (Table 33) represents the analyses of some specimens of ıan colostrum milk, which I have also had made by Dr. Harrington:

TABLE 33. (Harrington.)

	I.	II.	III.	IV.	V.
Fat	1.40	0.68	2.40	5.73	4.40
Milk-sugar and proteids	9.44	11.53	11.15	10.69	11.27
Ash	0.17	0.31	0.25	0.16	0.21
Total solids	11.01	12.52	13.80	16.58	15.88
Water	88.99	87.48	86.20	83.42	84.12
	100.00	100.00	100.00	100.00	100.00

These analyses, while not determining minutely the percentages of the ents of colostrum milk, tend to show the great variations which occur is period and how little knowledge we have concerning it.

n speaking to you about the colostrum in my lecture on normal devel- ent (Lecture IV., page 100) I stated that the colostrum corpuscles should pear from the milk in a week or ten days after birth. They diminish lly in numbers during the second week, and if they continue into the l week, or return at any time during the lactation, they almost inva- y cause disturbance of the infant's digestion; they also become an ation that lactation should be suspended temporarily, and, if they inue, that it should be entirely given up.

n the disappearance of the colostrum corpuscles the milk should rap- acquire its normal equilibrium, and, with the exception of its well-recog- l daily variation, should show a comparative uniformity in its analysis ng the whole of the nursing period, and until the equilibrium of the nmary gland is again disturbed, as at the end of lactation.

IUMAN MILK.—I have stated the general conditions which affect nammary product of all animals. I will now describe especially what nown concerning human milk.

UANTITY.—I have already told you how the mammary gland adapts uantity to the amount needed. The question so often arises as to ther the total amount to be secreted for each feeding can in any way be eased, that it is well to mention this now and to dispose of it. Beyond general conditions affecting the mammary product of the mother, which ave spoken of, I know of no means of increasing the flow of milk. I have little confidence in galactagogues in the form of drugs or special foods, for their numbers betray their inefficiency. The milk becomes lessened in amount from many causes. Some of these are identical with those which commonly produce any disturbance of its equilibrium such as I have mentioned. Certain drugs, such as belladonna, will in some individuals cause a notable decrease in the flow of the milk, and must, therefore, be given with care during the nursing period. An active cathartic will also lessen the milk, as will also a diet composed of solid food and very little water.

QUALITY.—The quality of the mother's milk is of the utmost importance to the welfare of the infant. It is very necessary, therefore, that we should thoroughly investigate and clearly understand what the normal composition and characteristics of her milk should be. This can be done only by having analyses made by expert chemists. Even with the aid of these analyses the information which is obtained concerning the percentages of the various elements is liable to be inexact in some of them.

This is unfortunately true regarding the two elements fat and proteids, which are under any circumstances the ones most likely to vary, and we must especially allow for some slight error in the proteid percentage.

The greatest practical assistance, however, can be obtained from these analyses, as they represent the true foundation for most of our work on infant feeding. I shall not attempt to describe the method of analysis which is used, as it is too purely a chemical question to be of practical use in clinical work.

My analyses have in almost every case been made by Dr. Charles Harrington. To obtain a specimen for analysis your hands should be sterile, and the breast and nipple should be carefully washed with sterilized water, and from 20 to 30 c.c. (5 to 8 drachms) of milk drawn by the breast-pump, which, being made of glass, can also be thoroughly washed. The milk should then be poured into a sterilized bottle and tightly corked. It should immediately be taken to the chemist, and kept on ice until the examination is made.

In every case it is very important to know the exact percentage of the fat, both from its being the most variable element and from its use in the determination of the percentages of the other elements. Any means, therefore, which will procure the exact percentage of the fat should be made use of where for any reason a complete analysis cannot be procured. The most exact means for this purpose outside of the chemical laboratory is an apparatus called the *Babcock Fat Tester*, which I shall show and explain to you at the Milk Laboratory in a later lecture (Lecture IX., page 250). As this is not an expensive machine, it has seemed to me that in communities at a distance from an expert chemist, or where the people are unwilling to pay for a complete analysis, a Babcock machine could be owned jointly by a number of physicians and kept at some central place.

The smallest amount of milk required for determining the percentage of fat with the Babcock machine is 17.50 c.c.

NORMAL LACTATION.—In order to understand the many variations which are continually arising in human milk during the period of what may be considered a normal lactation, we should clearly appreciate the various conditions existing in human milk and its composition as determined by a study of a very large number of individual specimens of milk. In this way we obtain a knowledge of the composition of the average human milk.

MICROSCOPIC EXAMINATION.—The mere microscopic examination of milk beyond the determination of the presence or absence of colostrum corpuscles and foreign matters, such as pus, blood, and epithelial cells, is too uncertain and misleading to be in any way depended upon, the chemical analysis being the only practical method which can be recommended. The truth of this statement was lately impressed upon me when a physician skilled in the use of the microscope sent me a specimen of woman's milk which he stated was rich in fat, but which the analysis showed to have only a little over one and a half per cent. of this element.

The presence of an undue amount of yellow coloring matter is at times very misleading. I have also seen human milk which had a greenish color, evidently produced by some of the micro-organisms which are known to occur in cow's milk, but the nature of which is not yet fully determined and which under the microscope are not represented by anything abnormal.

CLINICAL EXAMINATION OF HUMAN MILK.—The rules by which the percentages of the other elements of the milk can be deduced when once the percentage of the fat has been obtained by the Babcock machine are the same as those which I shall presently speak of in connection with another method of clinical examination, where, however, the determination of the fat is not so accurate as that by means of the Babcock. We cannot be too particular in regard to the accuracy of the method which we employ for obtaining an analysis of the milk; yet, as the most accurate analysis can be obtained only through an expert chemist, a simple approximate clinical test is often very desirable, even though it is less accurate. Under these circumstances the method employed by Dr. L. E. Holt, of New York, will be found to be of practical use. Holt does not assert that he reaches by his method anything but a fairly accurate knowledge of the percentages of the different elements of the milk, and he recommends it for the analysis of human milk only where a better one cannot be had. His results are based upon the comparative examination by his method of a large number of specimens of milk and on the following well-known chemical facts:

(1) That the *specific gravity* of human milk varies between 1029 and 1032, the average being 1031, at 21.11° C. (70° F.). Abnormal variations occur between the limits of 1017 and 1036. An increase in the fat lowers the specific gravity; an increase in the other solids raises it.

(2) That the *salts* do not vary much in their amount in ordinary human milk. They are too insignificant in percentage to affect the specific gravity, and in the clinical examination of milk they need not be considered.

(3) That the proportion of the *sugar* is nearly constant in human milk under all circumstances. This point has been emphasized by all the chemist who have made milk analyses.

(4) That in striking contrast to this uniformity in the sugar are the wid variations met with in the *fat* and *proteids*, as is shown by the following tables:

TABLE 34.

Variations in Fat.

From 48 analyses by Leeds	. . . .	2.11 to 6.89 per cent.
" " " König	. . . .	1.71 to 7.60 " "
" 29 " " Chem. Lab. Coll. Phys. and Sur. N. Y.	. .	1.12 to 5.02 " "

TABLE 35.

Variations in Proteids.

From 43 analyses by Leeds	. . . .	0.85 to 4.86 per cent.
" " " König	. . . .	0.57 to 4.25 " "
" 29 " " Chem. Lab. Coll. Phys. and Sur. N. Y.	. .	1.10 to 3.62 " "

(5) That to determine the composition of milk we must have a knowledge of the *proportions* in which the two elements which vary most widely namely, the *proteids* and the *fat*, are present.

(6) That from the fact that the proportion of sugar is so nearly constan and that the salts are in such small amounts, we may for clinical purpose consider the *specific gravity* as *modified* solely by the *fat* and the *proteids*.

(7) That there is no known method of determining directly the percentage of the proteids in the milk by a clinical examination, and that complete chemical analysis by an expert is the only one that can be accepte as accurate. It is possible, however, from a knowledge of the specifi gravity and the percentage of the fat, to make an approximate calculatio in regard to the percentage of the proteids, at any rate sufficiently close t determine whether in a given case they are near the normal, or are in ver large or very small proportions.

METHOD OF EXAMINATION.—It is necessary first to determine th specific gravity of the milk and the percentage of the fat.

To determine the composition of the milk by Holt's method the onl instruments needed are a small hydrometer, a pipette, and a glass-stoppere cylinder graduated in one hundred parts and holding about 10 c.c.

The specimen of milk for analysis should be taken from the "middl milk," and it is important that the milk should be freshly pumped an handled as little as possible, also that the graduated glass cylinder shoul be scrupulously clean, otherwise the milk will often sour before the crean has had time to rise. This is particularly true in summer. 15 c.c. (ounce) is the amount of milk required for the test.

Specific Gravity.—The specific gravity is obtained by means of th hydrometer, for the use of which only 15 c.c. (½ ounce) of milk ar needed.

Percentage of the Fat.—The percentage of the fat is determinec by estimating the percentage of the cream, which is ascertained by the following method:

The glass-stoppered cylinder is filled with milk exactly to the upper line, which is marked 0. The pipette should be used for putting the last few drops into the cylinder, care being taken not to allow the milk to run down the inner side of the tube, since this somewhat obscures an exact reading. The cylinder is then corked and allowed to stand for twenty-four hours at a temperature of as nearly 21.11° C. (70° F.) as is practicable. A variation of a few degrees on either side of this point is unimportant. If, however, the variations are wide, the rapidity with which the cream rises is somewhat modified.

In the great majority of cases the lower line of the cream has become sharply defined at the end of twenty-four hours, and can then be recorded. If this is not the case, the milk should be allowed to stand for six hours longer before reading the percentage.

By comparing the percentage of the cream with that of the fat, as determined by a chemical analysis of the same specimen, it has been discovered that the ratio of the cream to the fat is very nearly 5 to 3, and for clinical purposes it can be so estimated.

Estimation of the Proteids.—In estimating the proteids certain suppositions must and can be fairly accepted:

(1) Supposing the proteids to remain unaltered: if the percentage of fat be low, the specific gravity will be high, but if high, the specific gravity will be low.

(2) Supposing the fat to remain unaltered: if the percentage of the proteids be high, the specific gravity will be high, but if the percentage of the proteids be low, the specific gravity will be low.

If, therefore, the fat and the specific gravity be known, the proteids may be estimated by the following rules:

(1) If the percentage of the fat be found to be high, that is, from eight to ten per cent., and the specific gravity high, that is, from 1033 to 1034, we may assume that the proteids are also of high percentage, otherwise the excessive fat would bring the specific gravity below the normal average.

(2) If the fat be found to be of low percentage, that is, from three to four per cent., and the specific gravity high, we may assume the proteids to be nearly normal, since the high specific gravity is explained by the small proportion of fat.

(3) If the percentage of fat be high and the specific gravity low, the proteids may be assumed to be normal, since the variation in the specific gravity is explained by the low percentage of fat.

(4) If the percentage of fat be low and the specific gravity low, the percentage of the proteids is also low, since otherwise the small proportion of fat would make the specific gravity above the average.

Of course it is only the wide variations in the proteids which can be recognized by these rules; but these variations are often very important.

We can then say that, knowing the specific gravity and calculating th fat as three-fifths of the known percentage of the cream, we can judg whether the proteids are nearly normal, very high, or in very small amount Holt asserts that the estimation of the composition of milk by this metho is as exact as that obtained by ordinary examinations of urine.

CHEMICAL ANALYSES.—There is no doubt of the great value of a expert chemical examination of the milk in cases where an infant is no thriving, although apparently receiving a sufficient quantity of milk fro its mother. On the other hand, you must remember that a chemical analysi will never give any information regarding the quantity of the milk, and i often happens that where such an analysis has proved the quality to b good, the infant is not thriving because the quantity of the milk is ver small. The symptoms which indicate that it is the quantity of milk whic is at fault rather than the quality are that the breasts at the nursing tim are soft, and that only a small quantity of milk can be extracted from the by the breast-pump. A period of nursing longer than the usual fifteen t twenty minutes before the child is satisfied should make us suspicious tha the milk is lacking in quantity. We can also determine the actual quantit of the milk which the child has imbibed at an especial nursing by mean of weighing, as described in a previous lecture (Lecture IV., page 79) A number of observations at different nursings in the day must, however be made before a correct conclusion can be reached by this latter procedure.

AVERAGE ANALYSIS OF HUMAN MILK.—I will now call your attention to this analysis (Analysis 7) of average human milk, which represent the work of such chemists as König, Forster, Meigs, Harrington, and others and the mammary product of a large number of women of all nationalities The figures opposite each element are the percentages which that element represents as a part of the total solids, without reference to its own composition.

ANALYSIS 7.

Average Human Milk.

Reaction		Slightly alkaline.
Specific gravity		1028–1034
Water		87–88
Total solids		13–12
Fat	3–4	
Sugar	6–7	
Proteids	1–2	
Total ash	0.1–0.2	

You can obtain from this analysis a fair knowledge of the normal composition of human milk, and you will at once notice its simplicity and its few constituents.

Reaction.—The normal reaction of human milk when freshly drawn with suitable precautions is, as a rule, alkaline; it is, however, sometimes neutral, rarely acid, and in the latter case it may be considered abnormal.

Specific Gravity.—The specific gravity varies normally to a considerable legree on account of the variations in temperature to which the milk happens to be exposed at the time when the specific gravity is taken. When, however, the milk has its average normal composition, and the temperature to which it is exposed is 15.50° C. (60° F.), its average specific gravity is 1031.

Water.—One of the most important chemical facts to be remembered for clinical purposes is the very large proportion of water which is found in normal human milk, for it teaches us that it is a highly diluted food by which the best results can be obtained in infant feeding. It also explains to us how careful we should be not to overtax the comparatively slight power for absorbing a concentrated food which exists in the early months of life.

Fat.—The fat of human milk is made up of palmitin, stearin, and olein. About two per cent. of the total fat consists of the glycerides of butyric, caproic, caprylic, and myristic acids. The production of animal heat is so very important a part of the infant's well-being that it is not surprising we should find so large a percentage of fat as well as of sugar in the food which is provided for it. The presence of fat in the milk is not only for the purpose of nutrition, but also as a means for the maintenance of bodily heat. This latter function of the fat cannot with impunity be trifled with, and is essential for that active metabolism of which I have spoken in an earlier lecture (Lecture IV., page 100). A proper amount of fat is probably of great aid in the regulation of the fæcal discharges. An amount of fat proportionate to the proteids is presumably necessary, or at least of great aid, in their proper digestion. We should naturally expect that unless the standard percentage of fat, or at least a near approach to it, existed in the mother's milk, trouble would be likely to arise with her infant, and this corresponds with my experience in cases where the special ingredient which has interfered with the success of the nursing has been the fat. I have found clinically that where the fat was much lessened the nutrition suffered, that the digestion was not good, and that there was a tendency to constipation, while where its percentage was decidedly above the standard the digestion was weakened, there was a tendency to diarrhœa, and in consequence a resulting poor nutrition.

These clinical observations at once suggest to us that in the management of infant feeding we must recognize the existence of two important conditions. One of these is the digestion of the infant, the other is its nutrition. These two requirements for a successful lactation are based on the facts that the milk may be easily digested but not nutritious, and that it may be highly nutritious but difficult to digest, so that it is the equilibrium of these two conditions which produces a perfect infantile development. It is especially important that the percentage of fat in an infant's food should be within the limits of the normal variations which are found in the milk of healthy nursing women with healthy infants. For, although it is admitted that a large percentage of surplus fat is frequently found in the fæces of

Calcium phosphate	23.87
Calcium silicate	1.27
Calcium sulphate	2.25
Calcium carbonate	2.85
Magnesium carbonate	3.77
Potassium carbonate	23.47
Potassium sulphate	8.33
Potassium chloride	12.05
Sodium chloride	21.77
Iron oxide and alumina	0.37
	100.00

In comparing the previous analyses which have been made, and wh can be found in König's Nahrungsmittel, II., 2ᵉ Auflage, with this n analysis, we must remember that the previous analyses were made so years ago. In the last few years the processes which have been emplo have been so much more exact that these results must be considered more trustworthy than those made at an earlier date. It is not remarkal therefore, that distinct differences should be found between this new anal and the analyses which have hitherto been made, and presumably this analysis is the correct one. It has been made with the greatest care, by means of the most improved technique, by two eminently compe and well-known chemists, who in their work have acted as controls each other. In this way great precision has been attained.

The residue obtained from the evaporation of about six quarts of wom milk was extracted with naphtha to remove the fat, and then ignited very low temperature so as to prevent the volatilization of the chlori The ignition was accomplished by placing the residue from the naphtha traction in a platinum dish which was supported on a platinum coil in of a larger platinum dish, the latter being heated with a free flame. E at this low temperature a partial change in the composition of the ash t place, the sulphates being reduced to sulphites, but not to sulphides, as ash on being carefully tested showed that sulphides were not present. the carbonates of calcium and all the carbonates of magnesium were red to oxides. The ash also contained seven-tenths of one per cent. of un sumed carbon.

In woman's milk of course there would be no free carbon. All calcium that did not exist as phosphate would be in the form of sulp and carbonate, not of sulphite and oxide as found in the ignited ash. magnesium would exist as carbonate, not as oxide, and the potassium as phate, carbonate, or chloride. No sulphite of potassium would be prese

The chief differences between this new analysis and all previous ones as follows:

(1) The phosphoric acid is less than half as much as previously repo

(2) The magnesium is also less than half as much.

(3) Silica and alumina are present. They have not been returned in previous analysis.

Assuming the truth of the statement that the constituents of the mineral elements of human milk are subject to great fluctuation according to age and other causes, it is right to assume that the mineral matter examined by Kinnicutt and Harrington, being the product of a large number of women, is a fair average specimen.

From what I have already said you will understand that although chemical analyses enable us to work more intelligently, yet the conclusions which we can draw from them are far from being precise, owing to the variations which may occur and to the insufficient number of reliable analyses which have so far been made. We should therefore be extremely guarded in drawing conclusions, for the present merely looking upon these analytical results as important. It is very desirable that when reliable analyses are made they should be published, and thus as our information increases we shall be enabled to arrive at results which will greatly aid us in regulating the period of lactation.

Variations in Milk.—We are led to expect that we shall find that where the milk is poor and does not agree with the infant there is an excess of proteids and a diminution of fat beyond what we have so far been able to determine as the normal average percentages of these two elements. Again, where a variation takes place in the milk it is more likely to be found in the fat and proteids, as already stated, than in the sugar or the ash. I should also advise you to have a number of analyses made, on different days and at different times, in order that the error of an especial or temporary variation may be corrected. The importance of the assistance which can be gained from these analyses is, in my opinion, very great, and many more analyses should be made than we are now in the habit of deeming necessary. The question of expense should not for a moment be considered by those who can afford to have analyses made, for not only will real benefit come to their own children through money spent in this way, but these analyses, when published and collated, will prove of great value for the proper regulation of the feeding of infants in all classes of society. An error for which we must always allow may interfere with the true analysis of the milk which the infant has actually received in its stomach at the end of the nursing, and is one which must necessarily invalidate the information which we receive from our analysis. I have already referred to this subject in speaking of the changes which arise from slight causes and influence the special specimen which is being analyzed. Thus, we should recognize that the milk varies considerably in its percentage of fat and total solids in the different periods of a nursing, and that the composition of the milk which the infant has in its stomach may differ very widely from the composition of a specimen taken directly before or after the nursing. Harrington's analyses of the three periods of a milking will illustrate the meaning of what has just been said, and although they were made from the milk of a cow, yet, knowing the closely analogous conditions existing in human and in animal milk, we shall find them equally valuable in explaining the corresponding changes met with in woman's milk. Th are represented in this table (Table 36):

TABLE 36.

	Fat.	Total Solids.	Water.	Ash.
"Fore-milk"	3.88	13.34	86.66	0.85
"Middle milk"	6.74	15.40	84.60	0.81
"Strippings"	8.12	17.13	82.87	0.82

The analyses of J. Reiset and Peligot are also of considerable interest i showing not only the increase of solids at the end of a milking, but al that this increase is mostly of the fat, and to a lesser degree of the proteid and, as I have already stated, that a short interval of nursing increases t solid constituents in proportion to the water, the reverse of this being tr where the intervals are long.

Heidenhain explains this physiological phenomenon by saying that h investigations point towards the fact that during the pauses between t milkings the cells of the glands are growing. During this time a prop tionately small amount of solids and a proportionately large amount water are secreted, while the irritation of milking causes increased activ of the milk-cells, with a corresponding increase in the solid secretion an lessening of the water. Peligot's table, giving the analysis of an ass's mi in three different portions, shows the relations of the solids both to t water and to one another:

TABLE 37. (Peligot.)

Ass's Milk.

	1st Portion.	2d Portion.	3d Portion.
Butter	0.96	1.02	1.52
Milk-sugar	6.50	6.48	6.50
Casein	1.76	1.95	2.95

His second table shows the changes of proportion according to t intervals of milking:

TABLE 38. (Peligot.)

Ass's Milk.

	Milking Intervals.		
	1½ hours.	6 hours.	24 hours.
Butter	1.55	1.40	1.28
Sugar	6.65	6.40	6.38
Casein	3.46	1.55	1.01

The next table is also interesting, and should be recorded:

TABLE 39. (Reiset.)

Cow's Milk.

Last Time since Milking.	Percentage of Solids at Beginning.	End.
12 hours	9.83	16.04
6 "	12.80	16.06
2½ "	12.84	18.08

Harrington's analyses of woman's milk showing the "strippings" of a two-hours interval and the "fore-milk" of a twelve-hours interval are also of considerable interest:

TABLE 40. (Harrington.)

	"Strippings," 2-hours Interval.	"Fore-Milk," 12-hours Interval.
Total solids	15.32	10.14
Water	84.68	89.86
	100.00	100.00

With these chemical and physiological facts before us, we are forced to acknowledge that we must be very circumspect in the conclusions which we deduce from such analyses of human milk as have been made up to the present time. I have referred sufficiently to the errors which may arise in determining the percentage of the various elements which constitute the total solids, and I will once more merely state that an error in these conclusions where a correct chemical analysis has been made is less likely to occur from the sugar and the ash than from the proteids and the fat.

Reasoning from the strong analogy which must exist between human milk and cow's milk, and being aware of the great variations which occur in the latter, we may assume that human milk is liable to vary in its composition considerably with different milkings on the same day, and also with the milkings of the same hours on different days, so that at present we are not in a position to state that our knowledge of human milk is sufficiently exact to justify an attempt to formulate a table to show the composition of woman's milk at different periods of her lactation, however valuable such information may in the future prove to be. We must also understand that human milk of normal quality, and proving to be equally nutritious to the special infants that are fed on it, may vary considerably in the percentages of all its elements, and in the combinations of these percentages. This fact is well illustrated in this table (Table 41), showing the analyses of fourteen specimens of human milk all differing in the combinations of their different elements:

TABLE 41. (Harrington.)

Human Breast-Milk Analyses.

(Mothers healthy, and infants all digesting well and gaining in weight.)

	I. Per Cent.	II. Per Cent.	III. Per Cent.	IV. Per Cent.	V. Per Cent.	VI. Per Cent.	VII. Per Cent.
Fat	5.16	4.88	4.84	4.37	4.11	3.82	3.80
Milk-sugar	5.68	6.20	6.10	6.30	5.90	5.70	6.15
Proteids	4.14	3.71	4.17	3.27	3.71	1.08	3.53
Ash	0.17	0.19	0.19	0.16	0.21	0.20	0.20
Total solids	15.15	14.98	15.30	14.10	13.93	10.80	13.68
Water	84.85	85.02	84.70	85.90	86.07	89.20	86.32
	100.00	100.00	100.00	100.00	100.00	100.00	100.00

TABLE 41.—*Continued.*

	VIII.	IX.	X.	XI.	XII.	XIII.	XIV.
	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.
Fat	3.76	3.30	3.16	2.96	2.36	2.09	2.02
Milk-sugar . . .	6.95	7.30	7.20	5.78	7.10	6.70	6.55
Proteids	2.04	3.07	1.65	1.91	2.20	1.38	2.12
Ash	0.14	0.12	0.21	0.12	0.16	0.15	0.15
Total solids . . .	12.89	13.79	12.22	10.77	11.82	10.32	10.84
Water	87.11	86.21	87.78	89.23	88.18	89.68	89.16
	100.00	100.00	100.00	100.00	100.00	100.00	100.00

All these specimens of milk were obtained from healthy mothers, and every case the infant was thriving. In a number of these cases, howeve when one of the infants which was doing well on its own mother's milk w fed with one of the other combinations, it soon became sick, and had to changed back to the one adapted to its digestion. Human milk may, the be considered to represent not an especial food but a combination of food and its fat, sugar, proteids, and ash to represent these different foods. I other words, we find by experience that the digestive capabilities of infan differ, just as do those of adults, and that nature provides a number varieties of good human milk adapted to the varying idiosyncrasies of infant

Bacteriological Examination.—Although human milk is usual considered to be sterile, except in some cases in which the woman is disease yet Cohn and Neumann have examined the milk of forty-eight healt women and have found bacteria in forty-three cases. These organis were mostly represented by the staphylococcus pyogenes albus, with a few the staphylococcus pyogenes aureus and the streptococcus pyogenes. Th found fewer bacteria when the breast had been emptied a short time pr viously, and more when there had been a stagnation of the milk in the brea More bacteria were also found in the first few drops than in the last ones, a from their experiments they concluded that the bacteria enter the nipp from without. The conclusions deduced from their experiments, as well from the experiments of others who have met with similar results, seem show practically that bacteria can enter the ducts of the nipple and penetra to a greater or less distance; also that the milk in its course from the gla to the nipple washes out the bacteria, and that we can in this way accou for the presence of these organisms in the milk which is first drawn fro the breast, and their absence from that which comes later.

Young animals at birth begin to receive their nourishment immediatel and a corresponding increase in their weight takes place from the first d of life. The human infant in like manner should begin with its nursi early, getting what it can from the breast until the full supply of milk h come. In this way it will not be so likely to have a large initial loss weight to regain, a condition by which it is often handicapped at the ve beginning of its career, when there is most danger to be apprehended from depression of its vitality. Every day, every hour, is of the utmost importan in the early days of life, and, provided it can be done without detriment to the condition of the mother, the sooner the infant is put to the breast the better it will be. Under exceptionally favorable circumstances, as I have already told you, we see the breast-fed infant steadily gaining in weight during the first year of its life (Lecture IV., page 103). Ordinarily, however, we find this uniform increase in weight, which I have just indicated to you in speaking of the infant at the breast (Case 61, page 160), to be interrupted from time to time by various causes. These may arise during the dental period, in vaccination, from some temporary trouble arising in the breast of the mother, or from a combination of circumstances which may prevent the infant from receiving the proper qualitative elements in its food, or from obtaining a sufficient quantity. This continual increase in weight is of the greatest importance in the first year, as it is the chief index by which we note the progress of nutrition in the infant and the normal condition of the milk. During the first twelve hours of life, and in most cases during the first twenty-four to thirty-six hours, owing to the inability of the mother to supply milk for her infant, scarcely any food is, as a rule, obtained. If during this period the infant is restless and evidently hungry, 5 to 10 c.c. (1 to 2 drachms) of a sugar solution may be given at intervals of two or three hours. This solution should be made by dissolving milk-sugar in sterilized water, and its strength should be from five to six per cent. If the mother's milk is delayed still longer, something additional must be given to the infant, and if the food can be obtained from a milk-laboratory, I should order the following prescription:

PRESCRIPTION 3.

Fat	1.00
Sugar	5.00
Proteids	0.75

Reaction slightly alkaline.

10 feedings, each 30 c.c. (1 ounce). To be heated for thirty minutes at 75° C. (167° F.).

Where the infant's food has to be prepared at home, these proportions of fat, sugar, and proteids can be obtained, as I shall explain to you later (Lecture X., page 279), in my lecture on the home modification of milk.

The younger the infant the greater the metabolic activity, and hence the greater need of frequent feeding, for food is required not only for repair of waste, but also for the infant's rapid proportionate growth. This, with the increased demand for additional animal heat, makes essential the regulation of the intervals of feeding according to the age.

INTERVALS OF FEEDING.—The intervals constitute a very important part of the management of breast feeding, where, as I have told you, the quantity is regulated by the breast itself. These intervals should be definitely stated to the mother at different times throughout the nursing period, and should be adhered to. I have represented in this table (Table 42) the intervals which should be recommended. You must, however, understand that these are only average rules, and that the intervals of feeding should be made to correspond to the stage of development of the individual.

TABLE 42.

The day feedings are supposed to begin with the 6 A.M. feeding and to end with the 10 P.M. feeding.

Age.	Intervals.	Number of Feedings in 24 hours.	Number of Night Feedings.
From birth to 4 weeks	2 hours	10	1
" 4 to 6 "	2 "	9	1
" 6 to 8 "	2½ "	8	1
" 2 to 4 months	2½ "	7	0
" 4 to 10 "	3 "	6	0
" 10 to 12 "	3 "	5	0

When the milk has begun to be produced in the breast, the infant should be fed once in two hours during the day and once during the night until it is six weeks old. The day feedings are usually reckoned from 6 A.M. to 10 P.M. This interval of two hours should be adhered to, allowing that exceptional circumstances may arise where the physician must judge according to the individual case, until the sixth or eighth week is reached, when the intervals may be made two and one-half hours, and the number of feedings in the twenty-four hours eight. At about the fourth month the intervals can be made three hours, and the number of feedings six. When the infant is two or three months old, the night feeding can be omitted. The number of feedings at ten months may be reduced to five. Allowing the mother to have as many hours of continuous sleep at night as possible is especially important, in order that she may not be exhausted by the lack of that regular and sufficient rest which is of the utmost necessity for the production of a normal milk.

Irregularity in nursing, too frequent nursing, and too prolonged intervals often so disturb the quality of human milk as to transform a perfectly good milk into one entirely unfitted for the infant's powers of digestion. Thus, as I have previously explained to you, too frequent nursing lessens the water and increases the total solids in human milk, making it resemble in a certain way condensed milk; while too prolonged intervals result in such a decrease of the total solids as to render an otherwise good milk too watery and unfit for purposes of nutrition, however well it may be digested. I repeat, then, that the lesson that may be drawn from these facts is that some general rule for the feeding intervals should not only be recommended but enforced. The mother should neither injure her infant's digestion by nursing it too frequently, and thus giving it a too concentrated fluid, nor, by neglecting to feed it often enough, interfere with its nutrition by giving it a food that is too diluted.

REGIMEN OF LACTATION. **Diet.**—The diet of the nursing mother should not essentially differ from what would be considered to be a healthy one for her at any time. There is no special diet which, under all circumstances, is best for all nursing women during the period of their lactation. In the early days of the puerperium there is, as a rule, more danger of overfeeding than of underfeeding the mother. The tendency, in my opinion, is to give too much meat and solid food, with the result that when the secretion of the milk is being established the total solids are increased to a degree beyond the capacity of the still undeveloped digestive function of the infant. I have usually found that infants in the early days and weeks of life thrive better on a milk that shows a high percentage of water in proportion to that of the total solids. A rule which has in my experience become almost an axiom is that the age of the individual infant is in inverse proportion to its powers of absorbing solid food, and in direct proportion to the need of a large amount of water in its food. A light and plentiful diet should therefore be given to the mother while she is confined to her bed. This diet should consist of milk, gruels, soups, vegetables, bread and butter, and after the first week a small amount of meat once during the twenty-four hours. When the mother is able to go out of the house again, and has resumed her usual habits, the quality of the diet can be very much increased, and she can have the usual variety of food represented by meats, vegetables, milk, fruits, and cereals. There are no special kinds of food which are contra-indicated, provided we keep the food within the limits of the ordinary articles which commonly represent a plain but nutritious diet. It is very important for the nursing mother to have her meals at regular intervals, and during the early part of the lactation to take food somewhat more frequently than when she is not nursing. The additional meals, as a rule, should be made up of milk or cocoa. I have not seen the advantage of adding any special beverages, such as beer, malt, or stimulants, to her diet. She should receive as much milk as is compatible with her digestion, and should drink a plentiful supply before retiring at night. I have recommended this wide range of food for the nursing mother with a purpose,—namely, that it seems necessary to counteract many erroneous ideas and false views which are held on this subject. In my experience I have frequently met with mothers who were being deprived of the very articles which would in their special case have tended to aid in the production of good milk for their infants. The food of the nursing woman is without doubt closely connected with that which she provides for her infant. I have already spoken of the possibility of the elimination of various substances by the mammary gland, and we should therefore impress upon mothers the importance of a carefully arranged diet when they are nursing. Certain vegetables, and sometimes fish, will in individual cases affect the milk and cause discomfort to the infant. We must, then, in every case, seek to determine which article of diet may cause disturbance in the special woman's milk secretion, and eliminate that article. We should, however, be very careful not to prohibit this special article of diet from the regimen of a large number of women to whom it might be of benefit rather than of harm, simply because it has affected the milk of a few women. For the average woman a plain mixed diet, with a moderate excess of fluids and proteids over what she is normally accustomed to, will, as a rule, give the best results.

Exercise.—Exercise has so constant an influence on the changes which take place in the daily secretion of the milk, that the mother should be encouraged to be out of bed and to walk about her room as soon after her confinement as is possible without injuring her physical condition. Exercise is so important for promoting the proper elaboration and equilibrium of the milk secretion during the entire period of lactation, that it should always be insisted upon, and regular hours for walking should be as definitely arranged during the day as the hours for eating. The exercise must, however, be in accordance with the strength of the special woman, for fatigue has the same deleterious influence on the production of the milk as has lack of exercise.

DISTURBED LACTATION.—The disturbances which are liable to occur in the course of lactation are frequent and varied. They should be studied carefully and recognized at once when they occur, or the continuation of the lactation may not only be interfered with but be prevented entirely. When discussing the significance of the appearance of colostrum corpuscles in human milk (Lecture VII., page 166), I dwelt so fully on the variations which are coincident with this appearance, that I shall now merely refer to them as among the possibilities of a disturbed lactation. When they are found after the first two weeks of life the milk should be looked upon with distrust, and special efforts should be made to discover their cause, and to prevent the dangers which are liable under these circumstances to arise. These dangers may be not only from combinations of the milk elements which are incompatible with the infant's digestion, but also from the disturbances which may arise from the free mammary elimination of foreign material, which I have already referred to.

Drugs.—We know that during periods of mammary disturbance there is a much greater possibility, than when the gland is in a normal condition of the direct transudation from the blood of such inorganic substances as arsenic, antimony, lead, iodide of potash, mercury, and others, taken by the mother. Well-authenticated cases come to our notice from time to time where injury has been done to the nursing infant in this way, and where even death has occurred from the elimination by the breast-milk of certain organic substances, such as colchicum and morphine.

The greatest variety of substances have been found in the milk, but no definite rule as to the amount of this elimination has yet been established, so that our knowledge of the existence of this process is valuable as a prophylactic against harm, rather than as a means of direct benefit to the infant in disease, which latter point I shall not discuss except to call attention to the fact that the medicinal treatment of infantile disease through the breast-milk is exceedingly inexact.

We must also recognize the clinical fact that this elimination may occur at any time during the nursing period in the breasts of women who, so far as we can ascertain, are in a perfectly healthy condition. Thus, every practitioner has at times doubtless observed the laxative effect on the infant of such drugs as compound liquorice powder given to the mother; and a case has lately come to my notice where an infant vomited for weeks while taking the milk from the breast of its mother, who was unusually well and strong, but who was in the habit of drinking a considerable quantity of porter daily. After the porter was omitted the vomiting ceased at once, and did not return.

These facts warn us that the use of drugs during the period of lactation should be far more limited than at other times. Saline cathartics may not only act unfavorably on the infant through the mammary excretion, but may lessen very decidedly the flow of the milk, and even stop it altogether.

MENSTRUATION.—We must next consider the question of the variation in the milk which takes place from natural causes, such as the return of menstruation. Does such a return necessarily contra-indicate the continuation of nursing? As in all questions of this kind, we cannot adopt and follow an inflexible rule, but must be guided by what seems best for the individual case. Infants are at times affected so seriously by the alteration in the constituents of the milk which occurs once in four weeks that their nutrition is markedly interfered with, and a change to a more stable food is indicated. Again, the only disturbance which may arise is a temporary and slight digestive attack for a day or two, which apparently does not materially affect the infant, and makes us hesitate to run the risk of depriving it of a food on which it thrives during twenty-six days out of twenty-eight. We must also not be too hasty in concluding from the bad symptoms in the infant that we should at once withdraw it permanently from the breast, for the catamenia may appear once, and then not again for a number of months, the infant's powers of digestion in the mean time becoming so much more fully developed that they are unaffected by the milk of the catamenial period. Even where the catamenia recur regularly, the disturbance which may have been great at one period may for many reasons fail to recur at the next; so that the question is reduced to whether the composition of the milk shows a recovery of the equilibrium of its constituents within a few days, or remains affected to such a degree as to endanger the integrity of the infant's nutrition.

My own experience is in favor of allowing the infant to continue with the breast, unless it is decidedly contra-indicated by circumstances such as have just been mentioned.

I have seldom met cases which could not without permanent injury be tided over the small amount of temporary digestive disturbance which may arise. Within a few days I have seen a case where the return of the catamenia produced no effect whatever on the infant; and this is only an instance of what in all probability often occurs where mother and infant are at the time in an otherwise normal condition. There have, as yet, been too few analyses made during the catamenial period to justify us in drawing any definite conclusions as to the chemical status of the question; but the probability is that the milk will be found to be deficient in fat and to have its proteids increased, following the general rule of disturbed mammary secretion, and that consequently it is in a condition to interfere temporarily with both digestion and nutrition.

PREGNANCY.—A much more serious question arises when the nursing mother becomes pregnant; for here the almost universal clinical experience is that the infant, for various reasons, cannot continue to be fed by its mother, it being unusual for a woman to have sufficient vitality to nourish properly her living child and growing fœtus. The danger of reflex miscarriage from the continual irritation of the mammary gland by nursing I personally have had no experience with, but this is mentioned as one of the dangers contra-indicating the continuation of nursing by a pregnant woman. We must, however, here also not judge hastily, but take all the circumstances of the case into consideration before deciding on a measure of such vital importance to both child and fœtus. If the mother remains strong and vigorous, and the analysis of her milk shows no deterioration, while the infant is a delicate one just beginning to thrive on its rightful supply of natural food, or if it is during a hot period of the year, and especially where a wet-nurse or feeding from a milk-laboratory cannot be employed, it will often be wisest to take some risk and continue the nursing for a certain time, perhaps six or eight weeks, and then, according to circumstances, gradually to substitute another food. Almost every case will differ in the questions to be decided, and must be judged on its own indications and contra-indications, always, however, recognizing the accepted rule that lactation and pregnancy are usually incompatible.

The nursing mother is inclined to believe that if she feels well and strong her milk must be good for her infant under all circumstances. She therefore frequently transgresses the rules which are necessary for keeping her milk in equilibrium, and she should be made to understand that sometimes abnormal variations are liable to arise, however good her general health may be. She is simply fulfilling a task demanded by nature from those who bear children, and her duty, when once she has undertaken to nurse, is to prevent as much as possible these variations by regulating her life to a normal standard and avoiding excitement. Both of these requisites of a normal lactation come within the province of the physician to explain as he would any other branch of rational medicine. He should impress upon her that emotional mothers do not make good nurses, and that the physiological influence of the emotions on the nervous system, with its resulting changes in the mammary secretion, has necessarily a much wider range in women who are subjected to the customs and vicissitudes of modern life than it has in those who live in a more natural way.

Having shown you in Table 41 the great variations which occur in the percentages of the elements of human milk, I will now endeavor to explain to you by means of another table (Table 43) the percentages and combinations which you will be likely to meet with in abnormal milk.

TABLE 43.

Showing typical analyses of a normal, a poor, an over-rich, and a bad human breast-milk.

	Normal Milk. (Healthy life as to exercise and food.)	Poor Milk. (Starvation.)	Over-rich Milk. (Rich feeding; lack of exercise.)	Bad Milk. (Pregnancy, Disease, etc.)
Fat	4	1.10	5.10	0.80
Sugar	7	4.00	7.50	5.00
Proteids	1.50	2.50	3.50	4.50
Ash	0.15	0.09	0.20	0.09
Total solids	12.65	7.69	16.80	10.39
Water	87.35	92.31	83.70	89.61
	100.00	100.00	100.00	100.00

The terms poor and bad milk are merely relative, and in common use do not have a definite meaning. I shall, therefore, explain the distinction which I make between them. I have adopted the terms for the purpose of simplicity and to distinguish a milk which can be restored easily to a normal condition from one where the difficulty of such restoration is very great. By a poor milk I mean one which represents a condition of lack of nourishment or starvation in the mother, but one which can easily be changed by the proper feeding of the mother. In this case the normal mechanism of the mammary gland has not been interfered with. By a bad milk I mean one which represents a profound disturbance of the mechanism of the mammary gland produced by many causes, disease, pregnancy, and especially extreme nervous conditions in the mother, and one which cannot be easily changed to a good milk.

I shall now show you a table (Table 44) in which I have condensed the many means which you will have to make use of in managing the most difficult question which we meet with in the treatment of infants.

TABLE 44.

General Principles for Guidance in managing a Disturbed Lactation.

To increase the total quantity	Increase proportionately the liquids in the mother's diet, and encourage her to believe that she will be enabled to nurse her infant.
To decrease the total quantity (Rarely necessary.)	Decrease proportionately the liquids in the mother's diet.
To increase the total solids	Shorten the nursing intervals; decrease the exercise; decrease the proportion of liquids in the mother's diet
To decrease the total solids	Prolong the nursing intervals; increase the exercise; increase the proportion of liquids in the mother's diet.
To increase the fat	Increase the proportion of meat in the diet.
To decrease the fat	Decrease the proportion of meat in the diet.
To increase the proteids (Very rarely indicated.)	Decrease the exercise.
To decrease the proteids	Increase the exercise up to the limit of fatigue for the individual.

In attempting to formulate these rules I must warn you that I am dealing with a subject of which very little is known definitely. I can, therefore at present only state my experience in a large number of cases, and giv you some general idea of how you are to recognize whether you are dealing with a bad or poor milk rather than with a normal variation of a goo milk. This knowledge, however, of the variations which take place i human milk is of the utmost clinical importance during the period of lactation, for it is the only means by which we can decide definitely and intelligently many vital questions in this period.

THE MANAGEMENT OF DISTURBED LACTATION.—Instances have continually been brought to my notice where infants have been allowed eithe to continue with their mothers' milk when they were not thriving on it simply because it was mother's milk, or, on the other hand, have bee weaned from their mothers for what would evidently have been insufficien reasons had the case been thoroughly understood. In both instances proper knowledge of what can be done with human milk—that is, with th management of its different constituents by increasing or decreasing thei relative proportions—would have been of benefit to both mother and chil and in some cases would have saved the life of the latter. This lack o knowledge, or, I should say, lack of adaptation of the knowledge which w possess of this branch of medicine, is, to say the least, reprehensible, and i other branches of our art, which are more intelligently and carefully studie would be deemed inexcusable. Physicians are continually stating to thei patients that human breast-milk is the best food for infants, and at th same time are content to ignore the very principles which would mak their statements true. We should understand that when we speak of th superiority of breast-milk as a food, we mean good average breast-milk an for the average infant.

In all these cases of disturbed lactation we must first determine wheth the symptoms in the infant are really caused by a disturbance of the mil supply. We ascertain first whether the supply of milk is sufficient in quantity by the methods which I have already described to you. We then investigate the quality of the milk. A chemical analysis shows us wheth the percentages of the different elements are (1) normal or (2) abnorma If we find them to be normal, we know that it is not the milk which disturbing the infant, and we must seek for the cause of the disturban in other sources beyond the breast. If we find the percentages to differ decidedly from those of average human milk, we must determine whether is the variation from the normal average percentage which is producing th trouble, or whether these percentages are really well adapted to the infa and the cause of the trouble is to be looked for elsewhere. This can b done only by changing the different percentages and watching the resul If we find them abnormal, we can usually determine whether it is one o several of the elements which are producing unfavorable symptoms, and w should endeavor by our treatment to change the percentages of these ele ments so as to correspond first to the normal average percentages, and then, if this is not sufficient, to reduce them to lower percentages than the average until the infant's digestive functions have recovered their equilibrium. We must not forget in applying these principles that the cause of the disturbance of the milk exists in some abnormal condition of the mother, whether physiological or pathological, and that we must first remove this cause or we shall fail to regulate the milk.

A sedentary life, with abundance of rich, mixed food, provided the woman has a strong, healthy digestion, appears to increase the total solids and to decrease the water. This increase is almost always in the fats and proteids rather than in the sugar and ash; in fact, the marked variations in human milk are almost always shown in the fat and proteids, and hence our attention must almost invariably be directed to correcting these elements. This is fortunate, as we know of no special treatment, except on very general principles, by which we can alter the proportion of sugar or salts to the other constituents. A meat, or rather a nitrogenous, diet in the woman increases the fat in her milk. Our physiological knowledge also indicates that much fat eaten by the woman tends rather to lessen the fat in her milk. Hence to increase the proportion of fat in a woman's milk we should give much meat and only a moderate amount of fat. The proteids are more difficult to deal with. They have a tendency to increase in very bad and in very rich milk. The problem which we have to solve is almost always how to decrease them, no matter what the milk is. Our knowledge, unfortunately, concerning a sure means of reducing the proteids is very limited. Practically, however, I have found that where the woman is in good health it is physical exercise which we must insist upon, preferably walking in the open air and within the limits of fatigue. A walk of from one to two miles twice daily I have found to be about what the average healthy woman in New England needs to reduce the percentage of the proteids in her milk; but the amount of exercise must be carefully regulated according to the physical capabilities of the individual.

Bearing in mind these simple rules, and having determined, by means of an analysis or analyses, the cause of the special disturbance, you will be able to regulate the nursing period in cases where a lack of this knowledge would often necessitate weaning. You may in this way also avoid serious harm to the infant.

I shall next call your attention to these illustrative tables, which still further explain the rules I have just given you. I shall presently describe in detail some of these cases and discuss their analyses, but this repetition I deem advisable, as the subject is both important and difficult. For the purpose of still greater clearness I have in each of these tables first recorded the analysis of a normal milk, and have then, in parallel columns, shown the abnormal percentages and the changes produced in them by the management of the mammæ.

TABLE 45.

(*Human Milk.*)

Showing the influence of a luxurious life on a poorly-fed but healthy wet-nurse.

	I. Normal.	II. Two days before change of food.	III. Rich food and but little exercise for a month.	IV. Food and exercise regulated.
Fat	4.00	0.72	5 44	5.50
Sugar	7.00	6.75	6.25	6.60
Proteids	1.50	2.53	4.61	2.90
Ash	0.15	0.22	0.20	0.14
Total solids	12.65	10.22	16.50	15.14
Water	87.35	89.78	83.50	84.86
	100.00	100.00	100.00	100.00

TABLE 46.

(*Human Milk.*)

Showing a bad milk and one which it was impossible to manage on account of the contin recurrence of the same cause, uncontrolled emotions.

	Normal.	Emotions causing turbance in inf digestion.
Fat	4.00	0.62
Sugar	7.00	5.80
Proteids	1.50	4.21
Ash	0.15	0.20
Total solids	12.65	10.83
Water	87.35	89.17
	100.00	100.00

TABLE 47.

(*Human Milk.*)

Showing a milk possible to manage, because the mother, though excitable, was able and willing to control her emotions.

	Normal.	Infant doing badly. Colic. Mother before treatment.	Infant doing well. Mother after treatment.	Wet-nurse pro vided but no used.
Fat	4.00	1.62	3.20	3.04
Sugar	7.00	6.10	6.40	6.60
Proteids	1.50	3.54	2.52	2.32
Ash	0.15	0.17	0.18	0.12
Total solids	12.65	11.43	12.30	12.08
Water	87.35	88.57	87.70	87.92
	100.00	100.00	100.00	100.00

In the above case the mother was very nervous and wished to nurse infant, but thought that she could not, as she had been discouraged by nurse and physician.

She was then told that she could nurse in a week, if in the mean t she took proper food and exercise and withdrew the infant from the bre This she did, and had her breasts regularly pumped, with good results.

TABLE 48.

(*Human Milk.*)

Showing the effect of the catamenia on human milk.

	Normal.	Catamenia, Second Day.	Seven Days after Catamenia.	Forty Days after Catamenia.
Fat	4.00	1.37	2.02	2.74
Sugar	7.00	6.10	6.55	6.35
Proteids	1.50	2.78	2.12	0.98
Ash	0.15	0.15	0.15	0.14
Total solids	12.65	10.40	10.84	10.21
Water	87.35	89.60	89.16	89.79
	100.00	100.00	100.00	100.00

TABLE 49.

(*Human Milk.*)

Showing a milk in which the proteids, which were disturbing the infant, could not be reduced until the mother was made to walk comfortably, and thus without fatigue.

	Normal.	Infant with colic and vomiting. Mother taking no exercise and very rich food.	Infant as before. Mother walking two miles daily, but having blisters from French shoes.	Infant doing well. Mother walking two miles. Easy shoes, no blisters.
Fat	4.00	3.05	0.65	3.34
Sugar	7.00	6.10	5.25	6.30
Proteids	1.50	3.89	3.82	2.61
Ash	0.15	0.16	0.18	0.16
Total solids	12.65	13.20	9.90	12.41
Water	87.35	86.80	90.10	87.59
	100.00	100.00	100.00	100.00

TABLE 50.

(*Human Milk.*)

Showing how a milk can be managed while the nursing is continued.

	Normal.	Infant two weeks old, with serious general nervous symptoms and pain. Mother eating much meat and taking no exercise.	Mother walking and eating less meat. Infant entirely well.	Infant four months old, with pain and diarrhœa. Mother not walking so much.	Infant doing well. Mother walking two miles daily. Milk diluted one-fifth.
Fat	4.00	3.44	2.09	3.98	3.19
Sugar	7.00	5.60	6.70	7.00	5.60
Proteids	1.50	3.96	1.38	2.22	1.78
Ash	0.15	0.20	0.15	0.19	0.16
Total solids	12.65	13.20	10.32	13.39	10.73
Water	87.35	86.80	89.68	86.61	89.27
	100.00	100.00	100 00	100.00	100.00

As is seen from the analyses in Table 50, the infant did not do well until the mother began to exercise, and at four months it was again affected by apparently the high percentage of the proteids. The infant was considerably under the weight corresponding to that of the average infant of four months. It was found to nurse twenty-five minutes at a time, a calculation from its weight before and after nursing, it was found t from 80 to 120 c.c. (20 to 30 drachms). This amount being large the probable size of its stomach demanded, the time of the nursin reduced to twenty minutes, and 20 c.c. (5 drachms) of sterilized wate given in the middle of the nursing, thus changing the percentages milk to the figures which are represented in the last column. This c tion is on the basis of 100 c.c. (25 drachms) to each nursing.

So long as this method of feeding was adhered to, the infant did It was evidently a case where the infant could not digest over tw cent. of proteids.

TABLE 51.

(*Human Milk.*)

Showing that even for a long interval the breasts may be pumped and the result b successful nursing.

	Normal.	Infant showing nervous symptoms and much uric acid. Mother taking no exercise and much rich food.	Infant showing acid and t Mother walk miles and no much meat.
Fat	4.00	5.71	2.67
Sugar	7.00	4.00	6.60
Proteids	1.50	4.29	3.18
Ash	0.15	0.19	0.17
Total solids	12.65	14.19	12.62
Water	87.35	85.81	87.38
	100.00	100.00	100.00

In this case the infant was withdrawn from the breast temporarily the breasts pumped for twenty-seven days.

When the analysis presented the figures seen in the last colum milk was treated by diluting it, as in the previous case, and the infa put back to the breast.

TABLE 52.

(*Human Milk.*)

Showing the value of retaining the breast-milk by managing even an unpromising c

	Normal.	Infant with colic and failing. Mother no exercise, nursing irregularly, irregular and improper sweet food. Nervous, worried condition.	Infant put on bottle. Breasts pumped every four hours. Moderate exercise,—one mile. Full regular diet. Tranquil.	Exercise increased to two miles. Small amount of meat.	Eating meat. cise th
Fat	4.00	0.34	3.24	2.79	4.
Sugar	7.00	5.40	5.45	5.05	6.
Proteids	1.50	3.61	3.95	3.66	3.
Ash	0.15	0.18	0.16	0.20	0
Total solids	12.65	9.53	12.80	11.70	14
Water	87.35	90.47	87.20	88.30	85.
	100.00	100.00	100.00	100.00	100.

The above represents a bad milk from the failure of the healthy mother to conform to the rules of lactation. This bad milk, represented in the second column, had to be made into a rich milk by regular feeding before any attempt could be made to alter the ratio of the constituents. The proteids were then reduced somewhat by exercise, and, after the breasts had been pumped for two weeks, the analysis showed the percentages as represented in the last column. The milk was then diluted with sterilized water by the same method as was explained in Table 50, and the infant was put to the breast and did well; in fact, was carried through an attack of retropharyngeal abscess with this breast-milk.

If you have carefully studied these tables (Tables 43, 45, 46, 47, 48, 49, 50, 51, 52) and the principles (Table 44) on which they are based, you can appreciate the importance of the interesting illustrative cases which I am about to describe to you. I have selected them from a large number of my patients because they represented so well the value of a knowledge which aids us in the management of human milk during periods of disturbed lactation.

The decrease in the total quantity of the milk is of ordinary occurrence at any time during lactation, but it is most common among civilized races at about the eighth to the tenth month. When it occurs early in the lactation it is very disheartening to the mother if she is desirous of continuing her nursing. She becomes fearful that the flow of milk may stop altogether, and the nervous influence thus brought to bear on the mammary gland tends to increase the disturbance. We should therefore encourage her to believe that the milk will return. I have just succeeded in restoring the full quantity of milk in the mammary glands of a multipara (Case 63) who was very anxious to nurse her infant, which was three weeks old and had been digesting her milk, but had never nursed vigorously, and was not gaining. The mother was much discouraged because her milk lessened in quantity so early in the lactation, and she was convinced that it would not return. She had been taking, without my knowledge, a disproportionately small amount of fluid in her diet. There was an element in this case which the intelligent nurse brought to my notice,—namely, that the infant (Case 64) was not vigorous, and when put to the breast sucked feebly and called upon the gland for very little milk. Reacting to this lack of stimulus, the gland, although in a normal condition, secreted only the small amount demanded by the infant, and the milk lessened day by day. Treatment was instituted on the supposition that the mammary gland is practically self-regulating as to the amount of food which it will elaborate at a given nursing. If it happens to be called upon to nourish twins, it will increase the amount of its supply. If the infant which is put to it has a small gastric capacity, it will produce the amount needed for that capacity. I assured the mother that the milk would return, and I treated directly the mammary gland itself. An increase was made in the amount of liquid in the mother's diet, and the breasts were, after each nursing, pumped gently, skilfully, and thoroughly. The breast-pump supplemented the feeble action of the infant, and when more work was required of the gland it began to produce more milk. T increase in the liquid diet supplied the gland with materials to work wi and its mechanism ceased to be disturbed by the nervous influence emanati from the mother. She became cheerful when she found the milk returni while the infant, now that the milk could be procured more easily, demand more, sucked more vigorously, and thus satisfied the sensitive mechanism the mammæ.

The next case (Case 65) points to the possibility of our being at tin too hasty in the decision to deprive an infant of its mother's milk.

The mother (see Table 47, page 190), a rather delicate primipara, twenty-five y of age, was delivered of a boy seven pounds in weight. Within four hours puerperal c vulsions set in, from which she recovered, but was left with albuminuria 0.25 per cent. a casts. The latter disappeared in a few days, but the albumin, although somewhat dim ished, continued; and the patient, naturally of a calm disposition, was in a highly nerv condition, fearing that she could not nurse her infant, but decidedly opposed to havin wet-nurse. The milk appeared in considerable quantity on the fifth day, but the infant not thrive, and, although it gained somewhat in weight, was very fretful, slept very litt and looked ill, so that the attending physician became alarmed, and after treating it its dyspepsia without much success until it was five weeks old, and finding that there still about 0.25 per cent. of albumin in the mother's urine, decided with me that the bre milk should be withheld until we could determine the cause of the trouble, and an analy (Analysis 9) was accordingly made, with the following result:

ANALYSIS 9.

Fat	1.62
Sugar	6.10
Proteids	3.54
Ash	0.17
Total solids	11.43
Water	88.57
	100.00

This analysis suggesting the probability that the large amount of proteids was cau the disturbance of digestion, and that the small amount of fat was not sufficient for nu tion, the attending physician was very anxious to procure a wet-nurse; but while we w endeavoring to get a proper one, we decided to empty the mother's breasts with the bre pump every day, thus relieving her from the worry of attempting to nurse her infant seeing it fail to gain. She also obtained in this way undisturbed nights and a great deal out-door life. The infant was in the mean time placed on a substitute food, which digested very well, and, as it ceased to cry, the mother's mind became tranquil, and albumin in her urine in a few days was reduced to a trace. The treatment was carried for a week, the milk continuing to flow freely, and an analysis (Analysis 10) was t made of the mother's milk and also of that of a healthy wet-nurse (Analysis 11) wh infant was thriving on its mother's milk.

	ANALYSIS 10.	ANALYSIS 11.
	Mother.	Wet-Nurse.
Fat	3.20	3.04
Sugar	6.40	6.60
Proteids	2.52	2.32
Ash	0.18	0.12
Total solids	12.30	12.08
Water	87.70	87.92
	100.00	100.00

The two milks being equally good, it was decided to allow the infant to begin to take one nursing daily from its mother, although the proteids were still about one per cent. higher than the infant seemed likely to digest; it was given to its mother, nursed well, seemed satisfied, digested its meal without trouble, and at six months is still being nursed and is thriving.

The next case (Case 66) which I shall describe to you illustrates the principle that too frequent nursing lessens the water and increases the total solids in human milk, making it resemble in a certain way condensed milk. It also illustrates what I have stated concerning the two important questions to be considered in the management of a normal lactation,—namely, that the digestion as well as the nutrition must be regarded. This case is one of the numerous instances of the same kind which have come to my notice, and also emphasizes the fact that infants are often weaned from the breast where there is not the slightest necessity for it.

The mother, a healthy primipara about twenty-two years old, had nursed her infant for six weeks, during which time the infant was fretful, suffered much from colic, and never seemed satisfied. There was, however, a continual gain in weight, although the fæcal discharges showed evidences of the food not being properly digested and were numerous and watery. By advice of the attending physician the infant was weaned. The mother came to me for advice in regard to placing her infant on a substitute food. On inquiry I found that this infant had been nursed almost continuously night and day, with intervals usually of only one hour, and it was evident that the frequent nursings had resulted in producing a concentrated milk which the infant's gastro-enteric tract was rebelling against and was not digesting, although sufficient food was being absorbed to prevent up to this time any interference with the general nutrition. This infant, then six weeks of age, was deprived of its supply of good human milk in the middle of the summer simply because the important matter of changing the intervals had not been thought of as a means of improving the milk and relieving the pain and apparent hunger. There seems to be no doubt that if the milk in this case had been properly managed it would have agreed perfectly with the infant. I would also add in connection with this case that where the digestion is not carried on properly the nutrition must soon suffer, and it is only in the early weeks of a disturbed digestion that, as a rule, we find the nutrition to be unimpaired.

The next case (Case 67) is one of a multipara who was under my care at the City Hospital, and who up to the time of her entrance had been nursing her infant, which was thriving. This patient stated that her milk had always been abundant and of good color up to the time when she was separated from her infant, which was twelve hours previously, as she had to be away from home for that time. At the end of twelve hours the breast was found to be so distended that the breast-pump had to be applied. The milk was drawn with great ease, almost flowing of itself, and in considerable quantity, but it no longer resembled the milk of the previous nursings which had been at the proper intervals. On the contrary, it was clear, with very little color, the total solids were reduced to a minimum, and it no longer would have nourished the infant.

The treatment of this case was of course to pump the breasts every three hours until the infant could again be nursed.

As an illustration of the harm which may come to an infant from the percentage of fat in its mother's milk being too high, and also of the means to employ either to increase or to decrease the fat in breast-milk, this case (Case 68) will be of interest. The mother was a healthy primipara. She had plenty of milk, but the infant suffered from colic and had very frequent watery dejections. Finding that she was eating a great deal of meat three times daily and not taking much exercise, I naturally supposed from symptoms of the infant and the diet of the mother that an over-percent of fat was one of the elements which were disturbing the lactation, a that a high percentage of proteids would also be found. The analy (Analysis 12) proved my supposition to be correct:

ANALYSIS 12.

Primipara.—Healthy; eating much meat; not taking much exercise.

Fat	4.96
Sugar	6.60
Proteids	3.29
Ash	0.17

I therefore decided to reduce the meat to a minimum, which was do and three days later an analysis gave the following figures:

ANALYSIS 13.

Eating little meat.

Fat	1.73
Sugar	5.70
Proteids	3.74
Ash	0.13

The milk was found to be lessening in quantity. The infant's dejectio were less numerous and had more consistency; but it was not gaining, a continued to have pain. In fact, the analysis showed a poor milk, or ev a bad one, as represented by the usual combination of a low percentage fat and a high percentage of proteids. The woman was consequently m to eat a moderate amount of meat, and to exercise more, and three or f days later the analysis showed an improvement in the fat:

ANALYSIS 14.

Eating moderate amount of meat; taking more exercise.

Fat	2.42
Sugar	5.50
Proteids	3.55
Ash	0.15

The infant now began to gain in weight, but continued to have c as was expected from the high percentage of proteids. The exercise still further increased, and a later analysis showed a decided lessening of proteids, as is seen in this analysis (Analysis 15):

ANALYSIS 15.

Exercise still further increased.

Fat	2.35
Sugar	6.25
Proteids	2.69
Ash	0.15

The infant began to have regular movements, of good consistency, l no longer had pain; it also gained regularly in weight, and, as you see, ks well and strong. The mother has regulated her diet, exercise, and ep in accordance with the requirements of her infant, and her milk has ain become abundant.

We shall, of course, often fail in our attempts to manage the percentage f fat in this way, but this case illustrates exactly the changes which it is sually necessary to produce in order to alter a high fat percentage. The roteids also being high, I had an over-rich milk to deal with; taking away he fat-producing element reduced the fat to a low percentage; exercise educed the high percentage of proteids, and a combination of sufficient meat and exercise finally produced a milk which could be digested.

This next case (Case 69) is an interesting one, as it illustrates a number of points in the management of lactation. A high percentage of the proteids was creating the disturbance in the infant, and it was their final reduction through treatment that permitted the lactation to go on.

The mother, a remarkably healthy and vigorous multipara, living in the country, had a plentiful supply of milk. Her diet consisted mostly of vegetables, and she did not take much exercise. The infant was not thriving, having had continued attacks of colic, with frequent vomiting, and it did not gain in weight. The analysis (Analysis 16) showed a bad milk, which was contrary to what we should usually expect to find in the milk of a mother who was in such perfect health as this one was.

ANALYSIS 16.

Fat	0.52
Sugar	6.80
Proteids	2.48
Ash	0.15
Total solids	9.95
Water	90.05
	100.00

The mother was instructed to eat meat and to walk two miles every day. One month ter, as the infant had not improved, another analysis was made (Analysis 17), which owed that the milk was in a worse rather than a better condition.

ANALYSIS 17.

Fat	0.45
Sugar	6.15
Proteids	2.47
Ash	0.16
Total solids	9.23
Water	90.77
	100.00

I found that the mother had eaten meat but once a day, and in small quantity; also that she had not walked much. I then insisted on her eating meat three times a day, and walking three miles. This she did for two weeks, when the infant was found to have gained slightly in weight, but to still have colic and vomiting. Another analysis (Analysis 18) showed an increase in the fat.

ANALYSIS 18.

Fat	1.53
Sugar	6.68
Proteids	2.48
Ash	0.16
Total solids	10.85
Water	89.15
	100.00

During the next two months the walking was continued and the meat increased quantity. The infant continued to vomit and have colic until the mother was made to r on horseback every day, when the pain ceased, and from that time the infant gained stead in weight, and was well and strong during the rest of the lactation. An analysis (Analy 19) made two and one-half months after this procedure showed that at last the proteids h been reduced to come within the limits of the infant's digestion, and that the fat, althou still having a low percentage, had been increased sufficiently for the infant's nutriti Thus a bad milk was finally changed to a good one. This infant evidently could not dig a percentage of proteids approaching 2, but fortunately could be nourished on a low p centage of fat.

ANALYSIS 19.

Fat	2.01
Sugar	6.90
Proteids	1.54
Ash	0.17
Total solids	10.62
Water	89.38
	100.00

In the next case (Case 70) I had a poor milk to deal with. The inf was four months old. It was perfectly well and was digesting well, but h not gained for three weeks. The mother was producing from her breast sufficient quantity of milk, but the analysis (Analysis 20), as you see, sho that this milk had to be modified within the breast by a regulation of diet of the mother:

ANALYSIS 20.

Fat	1.29
Sugar	6.05
Proteids	2.98
Ash	0.12
Total solids	10.89
Water	89.61
	100.00

She was consequently made to eat an increased amount of meat, and the course of a few weeks the infant was thriving and gaining in weight.

The next case (Case 71) is that of a wet-nurse whose infant was dig ing well, gaining in weight, and happened to be of about the same age that of the infant whom she was hired to nurse. In order to see if t nurse's milk would agree with the foster-infant, the nurse and her inf were brought to the house of the foster-child, and were comfortably lodg and plentifully fed. Twenty-four hours later both infants began to ha colic and green fæcal discharges. An analysis (Analysis 21) of the milk showed a high percentage of proteids:

ANALYSIS 21.

Fat	3.19
Sugar	6.40
Proteids	3.11
Ash	0.15
Total solids	12.85
Water	87.15
	100 00

The nurse was then given a lighter diet with a greater proportion of liquids, and was made to walk one mile twice daily. By weighing the infants just before and just after a nursing, it was found that they took from 90 to 120 c.c. (3 to 4 ounces) in fifteen minutes. The infants were then allowed to nurse for ten minutes. 30 c.c. (1 ounce) of sterilized water was next given to them, and they were then allowed to nurse for ten minutes longer. In this way I estimated that they were receiving in their stomachs 120 c.c. (4 ounces) of food in which the percentage of the proteids was under 2.5. The infants ceased to have colic, and the fæcal discharges became normal. The nurse's infant was then sent away. Two weeks later the foster-infant was thriving, and, as another analysis (Analysis 22) of the milk showed a sufficient reduction of the proteids, the sterilized water was omitted.

ANALYSIS 22.

Fat	2.87
Sugar	6.25
Proteids	2.90
Ash	0.15
Total solids	12.17
Water	87.83
	100.00

During the rest of the lactation the infant digested well and gained fairly in weight.

This young woman (Case 72), who has brought her infant to show you, is perfectly healthy, and is nursing her infant, which has been digesting well and steadily gaining in weight for some months. I wish you to see this infant in order that you should understand how at times an infant can thrive on what appear to be too high percentages of some of the solids in the milk. This is the analysis (Analysis 23) of her milk:

ANALYSIS 23.

Fat	4.11
Sugar	5.90
Proteids	3.71
Ash	0.21
Total solids	13.93
Water	86.07
	100.00

In contrast to this woman (Case 72) is another woman (Case 73) who has brought her infant for you to see. The infant is evidently thriving. The mother is delicate and frail, and the infant is being fed by this healthy-looking wet-nurse. In the early part of the lactation the infant did not thrive, and, as the mother was so delicate, it was not deemed advisable to attempt to improve the quality of her milk. The interesting point in connection with this case is the inability of the infant to digest a poor milk and its ability to digest perfectly well this wet-nurse's milk, which in its analysis (Analysis 24) shows a very high percentage of fat and of proteids and a low percentage of sugar:

ANALYSIS 24.

Fat	4.72
Sugar	4.55
Proteids	4.74
Ash	0.19
Total solids	14.20
Water	85.80
	100.00

This mother who has brought her infant to see me to-day represents a case (Case 74) where I entirely failed to change the percentages of the elements in the milk. She had a moderate quantity of milk, and nursed her infant for two or three months. The infant did not gain, it had colic, and at times vomited. The analysis (Analysis 25) showed that it was in the class which I have designated as "bad:"

ANALYSIS 25.

Fat	1.61
Sugar	4.67
Proteids	4.07
Ash	0.17
Total solids	10.52
Water	89.48
	100.00

An increase of meat in this mother's diet and more exercise had no effec on the percentages of the elements of her milk, and the infant was therefore weaned. Soon after beginning to take a substitute food from the Milk-Laboratory the infant ceased to have colic, gained in weight, and it is now as you see, in a healthy condition. The percentages of the elements in the substitute food which produced such an immediate change in the infant's condition were as represented in this prescription:

PRESCRIPTION 4.

Fat	3.50
Sugar	7.00
Proteids	1.00

It was merely necessary to raise the percentages of the fat and sugar, and reduce that of the proteids, in order to produce this rapid and satisfactory result.

The next analysis (Analysis 26) which I shall show you is that of a woman's milk (Case 75), which is instructive for a number of reasons:

ANALYSIS 26.

Fat	2.30
Sugar	6.65
Proteids	2.57
Ash	0.12
Total solids	11.64
Water	88.36
	100.00

You see that the percentage of fat is low, and that of the proteids is rather high. The infant (Case 76), with the exception of being somewhat constipated, was always well, gained in weight, and showed no digestive disturbance during the lactation. This was remarkable, as the mother's catamenia returned regularly during the lactation from the time that the infant was four months old. There was considerable flowing at the time of the catamenia, and the mother was habitually constipated and did not have a very good appetite. The infant did not seem to be affected by any of these conditions. The analysis of this milk was made from a specimen of the "middle milk," which was taken between the catamenial periods.

It may be of interest, in connection with what I have said concerning the variations in the milk which may arise from emotional causes and menstruation, to report the analysis of a milk of a mother and a wet-nurse where these influences appeared to produce certain chemical changes. The mother (Case 77) (Table 46, page 190), a healthy but rather delicate primipara, the period of whose pregnancy had been supervised by me with the greatest care, but whose temperament was subject to extremes of despondency and excitement, was delivered, after a short and easy labor, of a healthy boy (Case 78). She was exceedingly anxious to nurse her infant, but within a few hours after its birth she was seized with an uncontrollable fear that she would be unable to do so. In spite of all the assurances to the contrary which could be given to her, and the plentiful supply of milk which in due time came in the breasts, she remained in a very nervous, despondent condition. As the infant began to show decided signs of indigestion, I thought it best, before proceeding further, to investigate the composition of the milk. The analysis (Analysis 27) resulted as follows, and plainly showed the necessity of not persisting further, as it was evidently much altered from unavoidable nervous conditions, which seemed likely to recur through the whole of her lactation:

ANALYSIS 27.

(*Mother's Milk.*)

Fat	0.62
Sugar	5.80
Proteids	4.21
Ash	0.20
Total solids	10.83
Water	89.17
	100.00

Under these circumstances, a healthy wet-nurse (Case 79) (Table 48, page 191), whose own infant (Case 80) was strong and thriving, was employed, and the foster-infant immediately began to gain in weight and ceased to show any digestive disturbance. After a month, however, it was found not to have made its weekly gain, to be unusually restless, and to be having frequent fæcal discharges. It was then discovered that the wet-nurse was menstruating, and on the second day this analysis (Analysis 28) of her milk was made:

ANALYSIS 28.

(*Wet-Nurse.*)

Fat	1.37
Sugar	6.10
Proteids	2.78
Ash	0.15
Total solids	10.40
Water	89.60
	100.00

The catamenia lasted about four days, and did not return for some months. The infant after the first twenty-four hours showed no disturbance whatever, soon began to gain, and was not affected by the subsequent recurrence of the catamenia. This analysis (Analysis 29), made one week after the catamenia had ceased, showed a decided change for the better; that is, increased fat and decreased proteids. Forty days after the catamenia a still greater improvement was found in the milk, as was anticipated from the thriving condition of the infant. The change in the percentages is shown in this analysis (Analysis 30).

	ANALYSIS 29.	ANALYSIS 30.
	Seven Days after Catamenia.	Forty Days after Catamenia.
Fat	2.02	2.74
Sugar	6.55	6.35
Proteids	2.12	0.98
Ash	0.15	0.14
Total solids	10.84	10.21
Water	89.16	89.79
	100.00	100.00

The following case (Case 81) is of considerable interest with reference to what I have told you in regard to the incompatibility of pregnancy and lactation. Unfortunately, a full consideration of the condition of the milk cannot be presented to you, as it rapidly disappeared from the breast after the first analysis was made, and, before another specimen could be procured, had disappeared entirely.

The milk was taken from one of my patients who had been pregnant for three months and at the same time was nursing an infant (Case 82) nine months old.

ANALYSIS 31.

Fat	7.64
Solids not fat	6.04
Total solids	13.68

The infant at the breast was not thriving. It had been digesting its mother's milk perfectly and had been gaining in weight until the pregnancy had existed for some weeks. At the time the analysis was made the infant's digestion had evidently been weakened, and as a result it had ceased to thrive and was rapidly losing in weight.

This analysis will be found to illustrate several facts. In the first place, it represents a very rich food. The total solids are even greater than appear in most cows' milk, and the fat is almost double the percentage which is considered normal in both human and cows' milk.

It also shows that a food may be unusually high in the percentage of its total solids and yet not of a character suited for the nutrition of an infant. The explanation of this fact is that although for a time an infant may digest fairly well a rich food, yet that nature has provided that the percentages of the elements in its food should remain within certain limits. If these limits are transgressed, either by giving too low or too high a percentage of any of the solids in the food, the nutrition will be interfered with. In the latter case the digestive function of the infant actually becomes weakened, and the strong food soon begins to act as a foreign body. The absorption of the food is next interfered with, and the infant starves as readily on the strong food which cannot be absorbed as on the weak food in which the needed elements are lacking.

This analysis also represents a condition which, in the majority of cases of pregnancy, occurs after the first six or eight weeks,—namely, a much disturbed mammary equilibrium. The percentage of fat in proportion to that of the solids not fat is so entirely different from the percentages of the different elements in a normal milk that we may say that this milk of pregnancy represents a condition of profound disturbance.

This special analysis must not be taken as a standard one for the milk of pregnant women, for, in all probability, analyses of milk under these conditions differ very widely, yet invariably show an absence of the normal percentages.

This next case (Case 83) (Table 49, page 191) represents a milk which could have been changed with comparative ease, provided that the mother had followed the directions given to her. She was a multipara, strong and vigorous, with a good appetite and a perfect digestion, and her life was entirely free from care. She had a plentiful supply of milk, but insisted on eating much more solid food during the puerperium than was compatible with keeping the elements of her milk in proper proportions. The infant soon began to be restless, and, although it gained in weight, it vomited at times and had colic quite frequently. An analysis (Analysis 32) of the milk showed what I had expected to find,—namely, a percentage of proteids too high for the proteid digestion of the infant.

ANALYSIS 32.

Fat	3.03
Sugar	6.25
Proteids	3.51
Ash	0.12
Total solids	12.91
Water	87.09
	100.00

The mother, who was able to go out of the house, was told to walk two miles twice daily. I also ordered her diet to be regulated so that there should be a smaller proportion of solids than she was now having. Sterilized water was given to the infant in the middle of its nursing. For a few days the infant seemed to improve and was less restless, but in another week the symptoms of indigestion returned, and, suspecting that the proper proportions of the milk were again disturbed, I had another analysis (Analysis 33) made, with the following result:

ANALYSIS 33.

Fat	3.05
Sugar	6.10
Proteids	3.89
Ash	0.16
Total solids	13.20
Water	86.80
	100.00

The percentage of the proteids, as you see, was now even higher than at the time of the last analysis. The mother declared that she had been walking up to the prescribed limits, but complained that the exercise tired her very much. It was very evident that the walking did not fatigue her sufficiently to influence her milk badly. I found, however, that she was not carrying out the rules which I had laid down for her diet, and had eaten freely of many rich foods. I then insisted on her leading a more rational life if she was to continue her lactation, and she promised that she would. The infant for the next few days ceased to have colic and was apparently perfectly comfortable. At the end of another week, however, the symptoms of a disturbed digestion returned in the infant, and I had to investigate still further the cause of the mammary disturbance. The mother had been carrying out all my rules as to diet, sleep, and exercise, but I now found that for walking she had used shoes with high French heels, and that she had blisters on her feet. Another analysis (Analysis 34) of what was practically a "foremilk" showed the low percentage of fat and sugar which might be expected in a "foremilk." The percentage of proteids was very high, considering that it was a "fore-milk."

ANALYSIS 34.

Fat	0.65
Sugar	5.25
Proteids	3.82
Ash	0.18
Total solids	9.90
Water	90.10
	100.00

The mother was now made to exercise in shoes fitted to her feet and having low broad heels, and to carry out rigorously all the rules which I had given her in the early part of her lactation. From this time the unfavorable symptoms in the infant disappeared, and it gained in weight and digested its food well. One week after this change was made in her shoes the analysis (Analysis 35) of her milk showed that it was now in normal equilibrium, and that the percentages of its elements were such as to lead me to conclude that the condition of the infant's digestion had become normal.

ANALYSIS 35.

Fat	3.34
Sugar	6.30
Proteids	2.61
Ash	0.16
Total solids	12.41
Water	87.59
	100.00

A few weeks later the infant again began to show symptoms of colic and general disturbance, and although the mother said that she had not been eating any food but what I had prescribed and that she was taking a long walk every day in properly fitted shoes, I knew by the high percentage of proteids which was shown by the analysis and by the condition of the infant that she was not telling the truth. I therefore decided that in the interests of the infant it would be better to wean it, which I did at once, and gave it a substitute food with a low percentage of proteids, on which it thereafter throve.

PROLONGED LACTATION.—In what I am about to say regarding the extension of lactation beyond the normal period of twelve months I shall not include the more pronounced pathological conditions, especially of a nervous type, which occur in certain women under these circumstances. In healthy women the milk towards the end of a normal lactation has a tendency to return to the condition which we notice at the very beginning of lactation; that is, the product of the mammary gland becomes unstable and the percentages show a poor or a bad milk. In rare cases I have met with women whose milk remained of fair quality and who could continue their nursing into the second year without apparent detriment to themselves or to their infants. There is, however, no reason for thus continuing the lactation, even if the mother is healthy and the milk good, for at the end of the first year, human milk, whether good or bad, is not a food which is adapted to the corresponding stage of development of the infant's digestive organs. Unmodified cow's milk and starch in some form are much better adapted to the stage of development of the digestive organs of the second year, and should therefore at that time be substituted for human milk.

MIXED FEEDING.—It not infrequently happens to nursing women, when their general health is not in a normal condition, that the supply of milk, while good in quality, is not sufficient in quantity to satisfy the infant, and the question arises whether the mother's milk should be entirely given up, or whether it should be supplemented by other food. My experience is in favor of assisting the mother to nurse her infant during the earlier months of its life. I have found that where the substitute food is carefully regulated, this method is superior to that of withdrawing the mother's milk and feeding the infant exclusively upon a substitute food.

We have, on the one hand, a better opportunity for regulating the mother's milk, by increasing or diminishing the number of the substitute feedings, and, on the other hand, if the mother's milk agrees with her infant, an excellent opportunity for making our substitute food correspond to what nature has provided. We can regulate more intelligently the infant's feeding by this method than by any other which is known.

In arranging a mixed feeding we should in every case first have an analysis made of the mother's milk, and, if her milk has been agreeing with the infant, make the substitute food correspond to the maternal. I would also recommend the practice of having an analysis of the mother's milk made at an early period of her lactation, as soon as the mammary gland has acquired its equilibrium and when the infant is thriving. This is a very important precaution, which may be of great use to us at a later period when the mother's milk may from many circumstances be disturbed or entirely lost. When such an accident happens, we know exactly what the composition of the milk was on which the infant was thriving, and can at once arrange a proper substitute food. As an illustration of the truth of this statement, the following cases (Cases 84 and 85) are instructive:

An infant (Case 84) was thriving on the milk of a healthy wet-nurse. One day, without giving any warning, the nurse left the house and never returned. The infant had to be put on a substitute food, as another nurse could not be procured. It was left in the middle of the hot weather without the food which had been so well adapted to its digestion. Unfortunately, the precaution of having an analysis made of the wet-nurse's milk had not been taken, and it was some time before I was able to substitute a food which would agree with the infant.

The second case (Case 85) was the one which I have already mentioned in Table 50, where the mother's milk, after careful management, had become fitted for her infant, and where the infant was thriving. One day the mother received a nervous shock from seeing the arm of another of her children dislocated. Within a few hours the milk entirely disappeared from her breasts and did not return. The analysis of her milk, which had been previously made, provided me with a guide by which I could at once have a substitute food prepared which would correspond to the food which the infant had been receiving from its mother. This was done, and the infant continued to thrive, showing no bad symptoms from the change of food.

There are certain points to be considered in mixed feeding. First, if the mother's milk is agreeing with the infant, the substitute food should be of the same composition. Second, if the mother's milk is fully digested by the infant but is lacking in certain nutritive qualities, the absence of which prevents the infant's nutrition from being normal, we should, after the first week, alter the composition of the substitute food so as to make it fulfil the requirements of nutrition by increasing the percentage of that special element in the substitute which is deficient in the composition of the maternal milk.

The times at which the substitute food should be given will depend upon the number of feedings which are found to be necessary in addition to the maternal feedings, and we should carry out the same principles in this mixed feeding that I have laid down for the general management of human breast-milk. If the mother's milk is lacking in quantity we should make the intervals between her nursings longer, and introduce one or two substitute feedings according as the age of the child requires shorter or longer intervals. If, on the contrary, the mother's milk is abundant, but either too strong or too weak, we should make the intervals of her nursings correspondingly long or short. In this way, with an accurate knowledge of the percentages which exist in the mother's milk, and with our power to change these percentages in substitute feeding, we can usually in a week or ten days regulate the substitute feeding of the infant to such a degree that the mother's milk will also agree with the infant, and the infant will thrive again.

WEANING.—There is no doubt that in a considerable number of cases occurring in the practice of physicians among civilized nations the mother's milk appears to be entirely unfit for her offspring, and it becomes a question whether the infant shall be withdrawn from its mother's breast temporarily or entirely. In such an emergency the careful and repeated analysis of the milk will enable us to determine this question wisely.

I am convinced that a large number of infants are deprived of their natural food and weaned on insufficient grounds. We thus assist to keep up the resulting high mortality figures, and I believe that these figures will be sensibly reduced when, in consequence of our taking a more enlightened view of the subject, we increase the number of infants who are fed during the first three or four months of life upon a suitable breast-milk.

A particular reason among many for waiting at least three or four months before weaning is presented by the fact that the stomach, after growing rapidly, has by the fourth or fifth month become a more perfect receptacle both as to size and to function.

A number of nursing women find that at variable periods in the course of their lactation their milk begins to fail, and they are forced first to lessen the number of their nursings and then to wean entirely. The time, then, when the infant should be weaned almost always settles itself, without our intervention, at varying periods. The period of lactation, and the one which might be called physiologically normal, can, when the breast-milk remains of good quality and quantity, be carried through the first year with benefit. We have certain guides which aid us in determining the proper time for beginning to wean. Physiologically, we know that certain functions, such as that which converts starch into glucose, are but slightly developed in the early months of life, and that they are only gradually established during the first year, and not, as a rule, perfected and in a condition in which we can call upon them with impunity until the last two or three months of that year. A sign which aids us in judging the progress of this development of the functions is the appearance of the teeth, calling our attention to the fact that nature is preparing the infant to digest and assimilate a form of food different from that which it has thus far received by sucking. The presence of six or eight incisors corresponds usually in the normally developed infant to the full development of the pancreatic secretion.

A most valuable index which assures us that we need not be anxious to change the infant's food during the first year is the continuous increase in its weight, which, with a general healthy condition, results from a normal lactation. We must allow, however, for certain variations which in special cases are as important as is the rule to terminate the lactation at a definite period. The period of lactation may be curtailed or lengthened by a month or two according to the season of the year, the development of the teeth, or the condition of the child from illness or convalescence. Under such circumstances it may be wiser to feed the infant from the breast during the heated portions of the year, and to wean it in cool weather, before or after the hot season, according to the individual case. An interdental period is also preferable to a dental period, on account of the possible disturbances which may arise in the latter and interfere with the proper actions of the new functions to which I have referred. In these exceptional circumstances, where there is any uncertainty as to the character of the milk which the infant is taking, a chemical analysis should be made at once, and repeated several times at intervals of a few days. These latter months, though not so difficult to manage intelligently as the early period of the infant's life, are much more likely to need careful supervision than the middle period, which, from its usually uninterrupted tranquillity, has been called the period of normal nutrition.

Where on account of an insufficient supply of milk in the mother the infant has for some time become accustomed to several meals of a substitute food daily, the matter of weaning becomes a very simple one, for we know that we have a food which will agree with it; but where we have to begin to wean directly and to adapt a food to the infant's digestive capabilities, as in cases of sudden failure of the milk or of sickness in the mother, this procedure becomes much more intricate, and is at times fraught with considerable danger. It is in these cases that an analysis of the milk made when the mother was in good condition often proves to be of great assistance.

The method of weaning which I have adopted, and have found to be the safest and best, is the one which I have been enabled to use since having a milk-laboratory at my command. My rule is, provided that the infant is thriving or digesting its mother's milk well, to order from the laboratory a substitute food the percentages of the elements of which are very similar to what the infant has been taking from its mother. After a few days, if this food is agreeing with the infant, I begin to change the percentages of the different elements, with the object of gradually combining these percentages in such a way as to correspond to the percentages of the elements of unmodified cow's milk. This is easily and precisely accomplished. For instance, supposing that the infant is receiving from its mother a milk in which the percentage of the fat is 4, of the sugar 6.50, and of the proteids 2, I begin by giving the same percentage of fat (4), a lessened percentage of sugar (5.50), and an increased percentage of proteids (2.25). After a few days, if this milk is digested well by the infant, I make the fat 4, the sugar 4.50, and the proteids 3. In a few more days, if this food is digested well, I give plain cow's milk heated to 75° C. (167° F.), with lime water sufficient to make it slightly alkaline. If this still agrees with the infant, I soon change to cow's milk unheated and unmodified.

Unless under very exceptional circumstances, sudden weaning is to be deprecated, though of course we must admit that it is sometimes done with impunity. The safest method, so long as we cannot judge beforehand which infants will be likely to be unfavorably affected by sudden weaning, is to take plenty of time and gradually ascertain by frequent changes, such as I have described, the food best adapted to the case. The infant should be gradually accustomed to this food, omitting the breast-feedings one by one, until finally we are sure that we have a substitute food on which it will thrive. At the tenth or eleventh month, provided that the weaning of the infant is deemed desirable at so early a period, and after having accustomed it to taking plain cow's milk, starch in some form can also be given. It will be necessary to determine how much of this new element may be introduced into the infant's diet, carefully adapting the amount to its amylolytic function, which varies in different infants, and which has but lately arrived at its full development. When these changes have been accomplished, the breast can with safety be entirely withdrawn.

The danger of injudicious weaning was strongly impressed upon me in a case which I watched for several days through the courtesy of Dr. Sinclair, of Boston, and which it seems well to put on record.

A delicate infant (Case 86), backward in its development, digesting well, and a little over one year old, was, without Dr. Sinclair's advice, suddenly deprived of the plentiful supply of breast-milk of its healthy mother and fed on oatmeal gruel. Vomiting and prostration immediately began, and continued until the oatmeal was omitted and the breast-feeding resumed, when the infant began to thrive again. Three weeks later the mother, through ignorance, suddenly and without any preparation fed it again on oatmeal gruel. On the following two days the infant vomited incessantly and was much prostrated. Several changes were then made in its food, but the symptoms grew worse, and the now thoroughly terrified mother again put the infant to her breast, with, however, this time a disastrous result, as her milk from nervous influences was so changed in its quality that it acted like a poison on the infant, who fell into a condition of collapse. Dr. Sinclair was sent for, and a few hours later I saw the case. A wet-nurse with a healthy infant four months old was immediately procured, and after several days of complete prostration the foster-infant began to revive, and later was gradually weaned without trouble. It may be well to add, for the encouragement of physicians who have cases of this kind to deal with, that after the mother's milk had poisoned the infant, and when I first saw it, the skin was gray and cold, the fontanelle sunken, and the eyes fixed, yet recovery took place. Under the same circumstances equal success in the treatment would probably be obtained by writing for a milk prescription to contain fat 2.50, sugar 5, proteids 1. This, of course, would be an exceedingly weak food for an infant twelve months old, but it would be the safest combination to begin with, and could be increased in strength as the infant recovered.

II. **DIRECT SUBSTITUTE FEEDING.**—WOMEN.—Where for any reason it is impossible or inadvisable for the mother to nurse her infant, some other food must be substituted for the maternal. The milk of another woman approaches the mother's in its characteristics most closely, and should be obtained unless contra-indicated.

It is generally supposed that the mother's milk, as a rule, is more likely to be suited to her infant's digestion than the milk of another woman; but we have as yet too few cases where direct investigation by means of chemical analysis of the two kinds of milk has been made to lay down actually as a fact what we can merely grant as a supposition, that an idiosyncrasy in the mother's milk will find an analogue in her infant's digestive powers. The reverse of this proposition has also been held to be true, that at times some idiosyncrasy in the mother's milk will make it radically unfit for her infant. The probability, however, is that analyses will show either that these varieties of milk are poor ones, or that the infants have unusually weak digestive powers.

The fact that every mother cannot provide as good a milk for her inf as can be supplied by another woman finds its analogy in the inability Jersey cows to rear their own calves.

In connection with what I have said about an infant sometimes havi an idiosyncrasy of digestion corresponding to some unusual percentage its mother's milk, this case (Case 87) will be of considerable interest:

The mother, a primipara, was healthy, but of a highly nervous temperament. infant was thriving, but, as a measure of precaution in case of mammary disturbance a later period of the lactation, I had an analysis (Analysis 86) made of the milk, with following result:

ANALYSIS 86.

Fat	5.16
Sugar	5.68
Proteids	4.14
Ash	0.17
Total solids	15.15
Water	84.85
	100.00

The report made by Dr. Harrington in connection with this analysis was, "The preci tated curd is quite similar in its appearance to that obtained in the analysis of cow's mil

I advised the mother on general principles to take more exercise, and ten days la another analysis (Analysis 87) of the milk was made.

ANALYSIS 87.

Fat	4.88
Sugar	6.20
Proteids	3.71
Ash	0.19
Total solids	14.98
Water	85.02
	100.00

The second analysis was so similar to the previous one that, in conjunction with perfect digestion and health of the infant, I concluded that this infant had an idiosync of digestion which enabled it to thrive on what would in most cases cause extreme distu ance. This view of the case proved to be correct, as the infant, which was under my for a number of months, continued to thrive. If you will compare this analysis with of the milk of the wet-nurse (Table 45, Analysis III., page 190) which I have previo described to you, where the high percentage of proteids caused vomiting of thick curd the infant, you will be impressed with the striking similarity of the two milks. There i doubt that in the majority of cases a milk such as is represented by these two ana would be totally unfit, and would not only cause marked indigestion but often more ser results, such as convulsions.

The following case (Case 88) presents an illustration of the reverse the supposition that the mother's milk will suit her infant's digestion be than the milk of a wet-nurse:

This infant (Case 89) was being nursed by its mother and showed continual disturb of its digestion. At times it would be constipated, and again it would have attacks of with watery discharges. The colic was the most prominent symptom, and the child, tho looking fairly well, was not gaining in weight. An analysis of the mother's milk sho that the percentage of fat was from 2 to 3, the sugar was of about the normal percentage, and the proteids varied from 3 to 3.50 per cent. The mother was of an extremely nervous temperament and was unwilling to carry out the rules for the management of her milk, which were absolutely necessary in order to reduce the high percentage of proteids, which evidently caused the disturbance. I therefore procured a wet-nurse, the analysis of whose milk was as follows:

ANALYSIS 38.

Fat	2.96
Sugar	5.78
Proteids	1.91
Ash	0.12
Total solids	10.77
Water	89.23
	100.00

The infant on taking this new milk ceased to have colic, but was more constipated and did not gain in weight. I therefore decided that it would be wise to increase the percentage of the fat in the nurse's milk. This was done by giving her considerably more meat to eat and making her take moderate exercise. The infant within a week began to gain in weight and to sleep well, the bowels ceased to be constipated and were moved naturally every day. There was also a plentiful supply of milk. Another analysis of the milk was then made, with the following result:

ANALYSIS 39.

Fat	3.31
Sugar	6.45
Proteids	2.36
Ash	0.16
Total solids	12.28
Water	87.72
	100 00

This last analysis is of great significance. The increase in the percentage of the fat evidently regulated the fæcal movements. The total solids increased from 10.77 to 12.28, and the plentiful supply of milk made the infant gain, especially as it now was digesting perfectly. You will observe that it could digest a milk with a percentage of proteids below 2.00, while it was a percentage of 3 in the mother's milk which prevented her from carrying on her lactation.

In this case it will be seen that the milk of another woman was far preferable to that of the mother, and that the idiosyncrasy of a high percentage of proteids in the mother's milk did not find its counterpart in an idiosyncrasy in the proteid digestion of her infant.

WET-NURSES.—The general question as to whether a wet-nurse shall be employed is one which is of serious import, and must in each instance be decided by giving full weight to all of the many circumstances which are involved in the case. Foster-feeding, where all the conditions are good, is superior to substitute feeding. The reverse of this statement, however, must always be kept in view, that a poor nurse, whether from temperament, or age, or general health, or the quality of her milk, had better be set aside where the conditions are favorable for a successful substitute feeding. It is perhaps better that the nurse's milk should correspond in age somewhat nearly to that of the infant she is to suckle, but a difference of some months in age may not be a contra-indication, as we are not yet in a position to say definitely that the milk differs sufficiently in different months to make this a reason of importance in choosing a nurse. A feeble child will nurse more easily and probably have better care from a multipara than from a primipara. The preferable age of the nurse is between twenty and thirty years. Her other requisites are a condition of good health and a quiet temperament. It will save much trouble and often obviate the frequent necessity for changing if before her engagement we have made a chemical analysis of her milk; in fact, all the points which have been already referred to for a successful maternal nursing are of equal significance in the case of a wet-nurse.

The general health of the wet-nurse should be carefully investigated, as women suffering from constitutional syphilis or any chronic disease are manifestly unfit for nursing. At the same time we should be careful, unless decided symptoms of disease are present, not to set aside the milk of a delicate-looking woman until it has been analyzed. The wet-nurse (Case 89) whose milk proved to suit the infant better than did its mother's was a frail, delicate-looking woman, but healthy. The mother, on the othe hand, was a large, strong-looking woman, but of a very nervous tempera ment. The rapid progress which is being made in the detection of th bacillus tuberculosis, not only in the sputum but also in the milk and i other secretions, may in the future be of much practical importance in th determination as to whether a woman should nurse an infant or not, but th present state of our knowledge is only sufficiently advanced for us to stat that this bacillus has been found in the secretion of the mammary gland.

Diet.—The same general principles that I have given in speaking o the diet of the mother should be applied to that of the wet-nurse. W should be extremely careful not to change suddenly the customary diet o a healthy nursing woman on purely theoretical grounds. For many yea the mistake was made of keeping women on too low a diet in the early perio of lactation, with the consequent delay of the establishment of a sufficientl nutritious milk-supply, and a corresponding initial loss of weight in the infants. Where, however, we are especially likely to err is in permittin a healthy, hard-working wet-nurse, accustomed to a somewhat coarse b nutritious diet, to adopt totally different habits of exercise and a diet t which she is unaccustomed, rather than to have her continue her usual mo of life. This sudden change of habits frequently results in loss of healt to the nurse, with its accompanying deterioration in the quality of her mil or at least a change in its quality so as to make it an unfit food for h foster-child. A notable instance (Case 90) of too radical a change of habi was brought to my notice by Dr. Swift, of Boston.

A wet-nurse had been procured for an infant (Case 90) ten days old. An analys (Analysis I.) of her milk, two days before she began to nurse, is seen in the followin table (Table 58). Her milk was digested well for two or three weeks, during whic time she was fed on an abundance of good food and rich milk. The infant then began t omit thick curds identical in appearance and toughness with the curds of cow's milk. nother analysis was made (Table 53, Analysis II.), which showed the amount of total solids be increased in a most marked degree, the percentage of proteids corresponding far more early to that of cow's milk than to that of woman's milk. The nurse was then given lainer food and skimmed milk, and the infant ceased to vomit. The infant and nurse connued well and strong during the whole year, the infant making a weekly gain in weight.

I have here an analysis (Table 53, Analysis III.) of this same nurse's nilk, made in the twelfth month of her lactation:

TABLE 53.

(*Wet-Nurse.*)

	Analysis I. Two days before change of food.	Analysis II. Rich food for a month.	Analysis III. Food regulated and milk agreeing with infant.
Fat	0.72	5.44	5.50
Sugar	6.75	6.25	6.60
Proteids	2.53	4.61	2.90
Ash	0.22	0.20	0.14
Total solids	10.22	16.50	15.14
Water	89.78	83.50	84.86
	100.00	100.00	100.00

ANIMALS.—I shall merely allude to the other method of direct substitute feeding by means of animals. In parts of France, notably in Brittany, infants are put directly to the cow's teats, and sometimes with good results. I know of one family of eight children all of whom were nursed by the family cow, and all of whom grew up healthy and strong. Yet the undesirability of feeding human beings directly from the udders of animals is so manifest that this method need not be discussed.

I shall at my next lecture deal with the third division of the First Nutritive Period, which I have designated "Indirect Substitute Feeding."

LECTURE VIII.

THE FIRST NUTRITIVE PERIOD.—(Continued.)

III. Indirect Substitute Feeding.

To-day, gentlemen, I have asked you to meet me here at the farm connected with the Milk-Laboratory, in order that you should study practically what will be of great use to you in your future careers. I would impress upon your minds that in this subject of *indirect substitute feeding* we have many links of a long chain, all of which should be as nearly perfect as we can make them if we expect to obtain a satisfactory result.

CHOICE OF FOOD.—I have laid great stress upon the importance of feeding infants during the early months of life by means of human milk We know, however, that in civilized communities the necessity will often arise for supplying the infant with food not from the human breast. In al probability the employment of substitute feeding will increase rather than decrease as our civilization advances. With this prospect before us, and appreciating the difficulties which in a large number of cases are liable t arise when we attempt to adapt a substitute food to the wants of an infant it manifestly becomes a duty to endeavor to reduce the high mortality figure resulting from artificial feeding. With this purpose in view, we should carefully investigate different methods of feeding and adopt some more uniform plan for starting human beings in life; for diversity and not uniformity i now the rule. While inherited diseases contribute a certain proportion of th deaths which occur in infants, yet diversity of method in feeding is the mos prolific source of disease in early infancy. The group of symptoms which for want of a better name is designated as difficult digestion occurs mos frequently in the three periods when the infant's digestion is likely to b tampered with,—namely, in the early weeks of life, when experiments ar being made to determine what food will be best to start with; next, when in addition to the irritation arising from the beginning of dentition, new articles of diet are added to the original food; and, thirdly, at the time of weaning, when there is often a sudden and entire change in the character of the food. The proper management of the first of these periods is of th greatest importance, because it is the time when, as before stated, the stomach is in its most active period of growth, and when the function of digestion i being established, and, following the rule of functional establishment, is i a state of unstable equilibrium.

We should recognize the fact that the problem of substitute feeding i not a simple one. We cannot reiterate too often that the question which commonly is supposed to be a simple one, and the one which in the grea

ajority of cases is alone considered,—namely, “Which food shall we give
the infant?”—is a misleading and insufficient one. The problem is a
ombination of factors of which the kind of food is only one, and I per-
onally have long been convinced that the neglect to investigate thoroughly
nd carry out in detail the combination of these by no means insignificant
eneral factors has had much to do with our failures with substitute feeding
n the past. It would seem, also, that the present is a most opportune time
or raising a note of warning against allowing our enthusiasm over any one
pecial theory to warp our better judgment. There will surely be a reac-
ion which will relegate to its proper place every theory built upon single
actors of the problem before us, and which is actually doing harm by keep-
ng in the background other theories which, each in its own sphere, as a
ignificant part of a complete whole, may be of very great importance in
he successful solution of the general problem. An error of oversight of
ne-eighth in a mathematical problem is not so great as one of one-fourth,
ut nevertheless the correcting of the greater error will not prevent an over-
ight of the smaller from completely destroying a correct result. Until
ately it has been the quality of the food which has been monopolizing to
oo great a degree the attention of the medical profession. To-day it is
terilization which in feeding has become prominent. Already one of the
atest German writers on substitute feeding has stated that the physiology
nd pathology of infantile digestion depend not on the chemical but on the
iological character of the food. If we are not on our guard, this exagger-
tion of each single factor will prevail, and by its influence will blind us to
nuch good work which in other directions has already been done, and which
ve cannot afford to ignore. Not that I would for a moment be understood
o underrate the value of feeding an infant on a sterile food, for it has for
ears proved of very great benefit in my practice and that of others, but I
redict that by just so much as we enhance the value of this one important
art of the whole at the expense of others, just so much farther shall we
e from an intelligent comprehension of the whole subject.

To feed an infant one month old with six ounces of acid cow's milk every
our hours, no matter how thoroughly such a mixture has been sterilized,
ould be a radical offence against well-known anatomical and physiological
aws. It therefore seems to me that time will be well spent in the discussion
f the subject of substitute feeding, if we investigate and endeavor to copy,
ach in its turn, the various devices which nature makes use of, for we must
dmit that we are not in a position to improve on nature's method.

It is certainly wiser and more economical not to spare expense and
trouble in arranging the infant's diet, for, as I have explained, the period of
active growth of an organ is the time when its function is readily weakened,
and, when once weakened, the digestive function is a prolific source of
annoyance and expense in childhood and adolescence. Cheap foods and
cheap methods of feeding, unless they are the best that can be procured,
should not be tolerated in the early feeding of infants. We often, however,

see a food recommended for a young infant because it is cheap and easily prepared, in spite of the fact that its well-known lack of nutritive ingredients would with adults stamp it as unfit for use.

In discussing the treatment of disease we advocate what is best, without reference to what it costs, and then, in the special case where expense is an element which has to be taken into consideration, we endeavor to adapt our treatment to these considerations, and approach as nearly as possible to our first standard. In like manner I believe that we are doing wrong to the public if we allow ourselves to be handicapped in so difficult a question as infant feeding by the cry of expense. Infant feeding is an expense which is vital to the welfare of the human race, and we can, without being accused of extravagance, safely relegate to the province of the manufacturers of patent foods the recommending to the public of foods which if judged by the amount that is offered in bulk are cheap, but which when judged by their nutritive properties are extremely expensive.

Our scientific knowledge and clinical investigations have not yet enabled us to follow nature exactly, and we therefore have not yet obtained an ideal method of substitute feeding. We must, nevertheless, go as far as the present state of our knowledge will allow, thus gaining a little ground every year; and we must be especially careful not to be led astray by the fictitiously brilliant results which are reported from time to time in favor of certain foods. Instances are continually occurring where one food will fail and another, when substituted for it, will succeed, and yet these successes are merely temporary, and the mortality resulting from the use of various infant foods always remains far above that from the employment of human breast-milk.

SOURCE OF FOOD.—Having decided to substitute some food in place of woman's milk for the infant, we must decide from what source the elements of this food shall come. The food which approaches most nearly in every respect the product of the human mamma is that produced by the mammæ of other animals. The reason for this is that the food which all mammals provide for their offspring is an animal one, and consists of the same elements, although the mammary product of different animals varies in the percentage of these elements.

Assuming, then, that average human breast-milk is the safest standard for us to copy, we are impressed with the fact that although a vegetable diet would often seem far the easiest method of procuring nourishment for young infants, yet nature has persisted in providing an animal one. We should therefore be very careful not to introduce into our substitute diet a vegetable element, which, as judged by our standard, must be a foreign element. Milk is the food which our reason tells us should be given to the young infant, and a milk which will approach as nearly as possible to the average human milk. That of various animals has from time to time been recommended as the best substitute for human milk, the recommendation being based on their analyses approaching more or less nearly the composion of human milk. The milk, however, of all animals has to be modified o correspond to human milk; and when we begin to modify, it is as easy o change the proportions of the different constituents to a great degree as o a small. The fact that the milk of any particular animal approaches in ts analysis nearly to that of the human breast is not of much significance, ther considerations being far more important; and it is most important of ll that we should use one which can be obtained easily by the people at arge. This at once settles the question that it is the milk of the cow to which we must turn our attention. Cow's milk may differ in its composi-ion from human milk to a greater degree than does the milk of the ass or he mare, whose milk approaches, so far as is shown by analyses, most nearly f that of all animals to human milk; but this in all probability is for the very reason that cow's milk is so universally used as a food for human beings of all ages.

If the ass and the mare should be employed for dairy purposes to the ame extent that the cow has been, there is every reason to suppose that their milk might change in its composition and their comparatively undeveloped mammary glands increase in size, just as has been the case with the cow, an nimal which for thousands of years has been used for the production of milk, and which probably did not in the beginning give such an over-production of the mammary secretion as is the case now. In fact, on the monuments in Egypt, where formerly there was either no trade in milk or very ittle, we find represented cows with only slightly developed udders, while he generative organs of the male animals are clearly depicted, a fact of some ignificance when we remember the well-known tendency of the Egyptians o realistic representations. It is, then, from the public demand, and by breeding, that cows have been made to produce so much more milk than is necessary for the support of their young. Not only quantitative but quali-ative differences exist in animals according to the development of their mammary glands; and, as Martiny has shown in his collection of statistics on this subject, the condition which determines the quantity and the quality of the milk depends on the development of the organ which produces it. The question of substitute feeding, then, is reduced practically to some modification of cow's milk, for this is the milk which is procured most easily everywhere, and, as the milk of all animals must be modified for the human infant, it is as easy to deal with cow's milk as with any other.

A further exemplification that cow's milk is practically the universal source of the substitute food-supply for infants in most civilized communities is the fact that the various foods, patent or not, all depend for their basis on cow's milk, and that without this addition of milk they would show but an insignificant percentage of many of the most important ingredients of the food. Logically we should not speak of the various foods as such, but merely as adjuvants to cow's milk. If this is thoroughly understood, much misapprehension regarding the apparently successful results of innumerable foods will be done away with.

One of the principal reasons for using cow's milk in preference to all others is that the cow has been kept under more strict control than any other animal has ever been.

As I shall in a later lecture (Lecture X., page 278), when speaking of home modification, have to refer to the necessity of using milk from common cows on any farm, it will be well for you to know what the average analysis (Analysis 40) is of milk taken from large numbers of common cows all over the world. This average analysis represents the work of well-known chemists, such as König, Forster, and others.

ANALYSIS 40.

Average Cow's Milk.

Reaction	Slightly acid.
Specific gravity	1029–1033
Water	86–87
Total solids	14–13
Fat	4.00
Sugar	4.50
Proteids	4.00
Total ash	0.70
Chlorine	13.45
Sulphur	0.41
Phosphoric acid	27.98
Iron oxide and alumina	0.44
Lime	23.17
Magnesia	2.63
Potassium	53.00
Sodium	4.49

The differences between the constituents of the ash of human milk and of that of cow's milk are as follows: in cow's milk there are more lime, magnesia, potassium, much more phosphoric acid, and less chlorine and sulphur.

THE COW.—Having chosen the cow for our primal milk-supply, we must consider whether any special breed is better adapted than others for accomplishing our purpose. To do this we should first examine chemically and microscopically the elements of the milk of those breeds which can be employed best throughout the civilized world. It has been found that the finer breeds of cows from the Channel Islands are more liable, when transported from their home to countries where the climate is more severe, to contract diseases, such as tuberculosis, than are the animals represented by the Durham, Devon, Ayrshire, and Holstein breeds. The characteristic analysis of the milk of the finer breeds, such as Jersey and Guernsey, is represented in this table (Table 54) in comparison with that of the milk of other breeds; the difference being mostly in the percentage of fat and slightly in the proteids. It may be well to state here that the percentage of proteids in the milk of pure Holsteins is also a little higher.

TABLE 54.

Cow's Milk Analyses.

	Jersey, Guernsey.	Durham, Ayrshire, Devon, Holstein.
Fat	5.50	4.00
Sugar	4.50	4.50
Proteids	4.25	4.00
Ash	0.65	0.65
Total solids	14.90	13.15
Water	85 10	86.85
	100.00	100.00

It is for future research to determine whether there is a qualitative as well as a quantitative difference between the fat secreted in the milk of the Channel Island and that of the more common breeds, but at present it would seem wiser, in choosing our medium for modification, to select the milk of the hardy breeds of cows.

A cow whose milk is to be used for purposes of infant feeding should be properly housed and well cared for, as the domestic cow is an animal peculiarly sensitive to her surroundings, and her product is correspondingly liable to be thrown out of equilibrium. The milk product of a herd of healthy cows is much less liable to the variations so injurious to the infant's digestion than is the milk of any one cow. It is especially to be noticed how much easier it is by proper care to control exaggerated nervous influences upon the cow's product than upon the woman's. This at once suggests to us the question, where and how shall cows be taken care of?

The ordinary cow is allowed to range over wide pastures which are sometimes over-flushed with herbage and sometimes parched by drought, and which nearly always contain noxious weeds, which she seems eagerly to seek. Again, she is forced to drink from stagnant pools and polluted streams, and at other times suffers for want of water for many hours together. She is also frequently exposed to storms. Cows cared for in this way are not those which provide the best milk for substitute feeding. These are the adverse conditions which surround the ordinary cow during the summer. In the winter she is crowded in the stifling atmosphere of a close barn with the manure of the whole winter kept underneath the floor on which she stands. Her head is usually confined in a narrow stall. The fodder intended for the winter's supply is kept above her head, and is continuously contaminated by the foul odors of the barn. She is turned out to the watering trough at periodical intervals. Thus she cannot be said to be cared for in a manner conducive to the equable function of her mammary gland.

For cows to be used for the purpose of infant feeding a barn is needed where each cow shall have at least fifteen hundred cubic feet of fresh air. The food should be kept where it cannot be contaminated. The manure should be as carefully removed from the barn as if it were a human dwelling. The cow should have freedom for her head and limbs in wide stalls all the year round. Large, dry, sunny exercise-yards should be provided for her. Her food should always be brought to her and selected with great care. Pure water should be provided, and suitable cups or troughs containing running water should be in her stall. The bedding should be fresh and free from mould or from any soil productive of bacterial growth. This can be accomplished best by means of sand or dry soil constantly changed at least twice a day. Methods should be used to get rid of all the usual foul odors and free ammonia so commonly produced in barns. Cows should be carefully guarded against fright, the worrying of dogs, and unusual excitements of all kinds, which cause serious disturbance of the lacteal functions of domesticated cows, in contradistinction to those of cows in a more natural condition, as for instance the cows in a semi-wild state on the plains of Montana, Texas, Australia, and the Pampas of South America. Excitement does not apparently injure the lactation of these cows, while it inevitably throws out of equilibrium the milk of the well-cared-for dairy cow. If the same care should be applied to regulating the woman's life as is employed here in this barn with these cows, we should encounter fewer difficulties in human breast-feeding.

The feeding of the cows of this farm has for its object the production of an even, nutritious, digestible milk and the careful avoidance of over-stimulation of the lacteal secretion. For this purpose a somewhat wider ration than that employed for the production of milk to be used in butter-making, but somewhat narrower than that employed for the production of beef, has been found to be the best adapted. For example, a ration for the production of butter fat up to the limit of the cow's capacity would be in accordance with the ratio of Wolfe so often employed,—namely, one nitrogenous part to four and a half non-nitrogenous. The ration for the production of beef in its most economical manner would be that used by English feeders as prescribed by Lawes,—namely, a proportion of one nitrogenous to eight non-nitrogenous parts. The ratio which has been demonstrated to produce the best milk for infant feeding is the mean between these two,—namely, one nitrogenous part to five and a half or six non-nitrogenous parts. A constant use of this ratio in the combinations of many fodders and grains appears to have produced a reasonably large supply of milk with fair richness, but without over-stimulation such as would be shown by a disturbance of function. Nitrogenous foods for cows are the leguminous groups of grasses and plants, such as the clovers, lucern, beans and peas, vetches, and other plants of like kind. Besides these fodders we have for nitrogenous foods suitable for producing milk for substitute infant feeding, such grains as wheat-bran, oil-meal in small quantities, and pea- and bean-meal. Of the non-nitrogenous fodders the principal ones are maize-stover, the hays from timothy, red top, orchard grass, Johnson grass, rye grasses, the bents, Kentucky blue grass, June grass, and oat straw. Most of the grasses in a green state afford a fairly balanced medium ration for substitute feeding. Of the non-nitrogenous grains the most suitable is maize-meal. We also have oatmeal and barley-meal, which contain less of the non-nitrogenous elements than the above, but still must be classed with them. The exact chemical analysis of any one ration used for feeding cows for our purpose must be carefully considered in accordance with the ratio of the digestible nutrients of the food, and this must of course be arranged practically from the recognized food tables. A great variety of food is necessary in feeding cows, but in the transition from green foods to dry, or the reverse, much care is needed to graduate the change, as disturbance in the equilibrium of the mammary gland is rapidly followed by injurious effects on the consumer. In past times, before I could rely as I do now on this carefully-managed change of rations, the spring of the year with its flush pasturage and the fresh grass following the autumn rains were fruitful sources of infantile digestive disturbance in my nursery practice.

You will now appreciate how important are all these links in the chain which constitutes a successful substitute feeding. The cows must be kept clean by grooming and the necessary washing, the precaution always being taken to rub the moistened parts dry. The milkers should be dressed in clean white suits and caps. Their hands and arms should be thoroughly scrubbed before milking. The hands in milking should be kept dry. The milk should be drawn with some force, simulating the action of the calf, and at each milking every drop of milk should be drawn out. The milk should be drawn into glass-lined pails and carried immediately from the barn to the milk-house, which should be a sufficient distance from the barn to be free from odors. No means yet known to science can prevent some few bacteria coming into the milk during the milking-time, though it is possible to reduce the number so greatly as to make the milk practically sterile for the purpose of infant feeding, particularly if the second half of the product of the udder alone is used and milked into sterile tubes. The first half probably contains many bacteria, which, entering from without, have reached the lower portion of the teat.

BIOLOGY OF THE MILK.—The experiments on the biology of the milk of this special herd which I am showing you have been made by Professor Ernst and Dr. Jackson, and the results are shown in this table (Table 55). The specimens examined were taken from the mixed milk of the entire milk of the herd.

TABLE 55.

Bacteriological examination of milk from the entire herd milking showed six hours after the milking sixty-eight thousand colonies.

Specimen.	Heated to		Minutes.	Developed Bacteria.
Whole milk	75° C.	(167° F.)	10 and 20	0
Modified milk	75° C.	(167° F.)	10 and 20	0
Whole milk and modified milk .	65.55° C.	(150° F.)	10 and 20	Numerous.

In striking contrast with these results obtained by experimenting with the entire milking are some special experiments made on this same milk by Dr. Austin Peters and Dr. A. K. Stone, at Mr. Gordon's suggestion, for the purpose of deciding whether it was possible to obtain a practically sterile milk at any part of the milking. The manner of performing the experiments was as follows:

Dr. Peters was dressed in a freshly-boiled white suit and cap, and had his hands and arms thoroughly washed with a 1 to 1000 bichloride of mercury solution. The cow's udder, teats, flanks, sides, groins, and abdomen were washed with the same solution, and dried with a freshly-boiled cloth. The milking was then done by Dr. Peters into bottles which had been carefully sterilized at the bacteriological laboratory, with the following result.

Of the four cows milked for this experiment and selected without special choice, the bottle marked 1 in each of the following sets of figures in this table (Table 56) represents the milk of the first half of the milking and drawn by the hand of the milker directly into the sterile bottles. Number 2 in each set of figures represents milk drawn through a sterile canula directly into the bottle, while numbers 3 and 4, respectively, represent milk drawn by hand after more than one-half of the udder had been emptied. A bacteriological examination of the milk in these bottles, by Dr. A. K. Stone, gave the following results:

TABLE 56.

	Colonies.	Colonies.	Colonies.	Colonies.
1	141	167	19	58
2	0	0	1	2
3	0	6	0	0
4	0	0	1	2

The results of Dr. Stone's examination showed, first, that the milk obtained from the first half of the milking contained a comparatively large number of micrococci and fine bacilli of the same general appearance respectively; second, that the milk drawn through the sterile canula was practically sterile, and that the milk drawn in the second half of the milking by hand was so uniformly sterile as to awaken the suspicion that the isolated colonies might have been the result of the manipulation between the "cow and the plate."

These experiments at once provide us with a means of procuring a milk practically sterile but not sterilized. This experiment also seems to prove that the bacteria which are found in cow's milk do not necessarily come from external sources, whether they be of the cow herself or of her surroundings, but may also come from some part of the milk tract between the udder and the end of the teat. These conclusions, it may be said, are made with reference to healthy cows.

Infectious mammitis, to some extent, seems clearly to be carried by the hands of the milkers from cow to cow. This also points to the fact that bacteria may find their way to the ducts through the teats.

These experiments are of great practical importance when it is considered that while under certain circumstances it is impossible to obtain the advantages of such a farm as this and the modification of milk by means of aboratory processes, yet it may be of great necessity to the infant on account f sickness to be fed with a sterile fresh milk not sterilized. This could, f course, be accomplished on any farm with any cow by means of ordinary are in the milking, and by such rules as were carried out by Dr. Peters. The major part of the bacteria present in the milk are such as cause the usual acid fermentation which we recognize in the common souring of milk, but there are many species of bacteria which ought to be prevented from gaining access to the milk, arising from mouldy hay, straw, or fodder, partially decayed roots, and the natural decay of the wood-work of the barn and adjoining buildings. These latter varieties, which are found to be especially inimical to the preparation of substitute foods, cause in some cases the alkaline fermentation and other abnormal conditions of milk. Every barn apparently has its own set of bacteria, and the flora in America do not exactly resemble the analogous European species which have so often been described.

REACTION OF COW'S MILK.—It seems to be true that milk drawn from cows fed on the better grasses in a half-ripe condition is nearly or quite alkaline, while the milk from stall-fed cows, where dry fodder and grain only are used, is inclined to be acid.

It will perhaps be interesting to you, inasmuch as grass feeding is not always practicable, to hear what has been done to produce a normal cow's milk which is alkaline and thus corresponds to normal human milk.

The importance of the subject lies in the well-recognized fact that the infant's digestive functions have been from time immemorial better adapted to the digestion of an alkaline or a neutral fluid than of an acid one. Whether the moderately alkaline reaction of human milk is an important factor in the problem of infant feeding is a question which future investigation alone can completely prove, but with our present knowledge we are not prepared to dispense with even the least important of the many factors which make up this problem. At any rate, we should be very suspicious of a breast-milk which shows an acid reaction. In the preparation of an infant's food from cow's milk, according to the latest experiments by means of modification, the best results have been obtained by making the reaction of this food correspond to that of normal human milk. This, up to the present time, has been done best by the addition of an alkali, which is the only foreign element that it has been found necessary to employ.

My attention was first drawn to the possibility of obtaining an alkaline cow's milk corresponding in its reaction to that of human milk by Mr. G. E. Gordon, who, by his extended and intelligent investigation of this subject carried on for so many years, has given such a stimulus to these questions of clinical interest. Many years ago it was noticed that cows fed on certain pastures, such as occurred in Kentucky, represented by the Kentucky blue grass, and also in many other parts of the West, produced at the height of the season of such grass a product which was alkaline rather than acid, and which remained alkaline for a number of hours after milking. It is also of course well known that milk in general, wherever it is produced throughout the world, has an acid, or at least an amphoteric, reaction. This information at once incited the investigation of the food values which existed in these peculiar pastures. A careful analysis showed that the nitrogenous elements of this grass bore a certain proportion to its non-nitrogenous ones,—namely, about 1 to 4.5. We should naturally suppose that if we combined nitrogenous and non-nitrogenous foods in the proportion of 1 to 4.5 the product of cows fed upon this combination would resemble closely the product of cows fed upon the pasture grasses already mentioned. This to some extent has proved to be true, but not so completely as is to be desired for the precision needed in infant feeding. It is therefore interesting to record that the experiment of supplying the non-nitrogenous proportion of the food with sugar-beets (ten pounds to each cow daily) of the highest saccharinity has accomplished unlooked-for results. The cows which were experimented with in obtaining these results were under observation for three months, and were cared for in the same barn and under the same general conditions. Two-thirds of this herd were fed on hay and grain combined in the ratio of 1 nitrogenous to 4.7 non-nitrogenous parts. The remaining third of the herd was also fed according to the same ratio, but this ration, so far as the non-nitrogenous elements were concerned, was made up partly of Austrian sugar-beets grown for this purpose. No beets were given to the first two-thirds of the herd just spoken of. During the three months when the experiments were being made, the reaction shown by the milk to common litmus paper was constantly as follows: the milk of the cows fed partially on the beets exhibited a neutral or feebly alkaline reaction, while that of the cows that received no beets showed a somewhat acid reaction.

A still more delicate test of the reaction of the milk of the entire herd was made by Dr. Austin Peters, of Boston. Hay and grain without beets, as previously stated, had been the food of two-thirds of the herd, and ten pounds of beets to each cow daily had been fed to the remaining third.

The results of the testing of the alkalinity of this milk at the various stages of the experiment were as follows. The milk of the cows which had been fed with beets, when tested directly by Dr. Austin Peters as it was milked into the pails and where it had a temperature of 33.88° C. (93° F.), invariably gave the following reactions:

Blue litmus paper gave no change whatever.
Red litmus paper was turned slightly blue.
Cochineal and ammonia paper turned still bluer.

The mixed milk of the whole herd in the vat and at a temperature of 5.55° C. (42° F.) was then tested by Dr. Peters, with the following results:

Blue litmus paper showed no change.
Red litmus paper was turned slightly blue.
Cochineal and ammonia paper was turned still bluer.

Finally the mixed milk of the whole herd, after being carried twelve miles to the Laboratory, was tested by Mr. Gordon with cochineal and ammonia paper; the paper was found to turn just as blue as when the milk was tested in the vat at the farm.

These experiments are of great interest as showing that not only can the product of the cow, so far as its reaction is concerned, be made to correspond to that of human beings by means of perfectly natural feeding and under perfectly normal conditions, but that this alkaline modification can be produced to such a degree that one-third of the milk is sufficient to destroy by its alkalinity the acidity of the remaining two-thirds.

THE MILK-HOUSE.—After the cows are milked, the milk is carried quickly from the cow to the milk-house, which in this instance is over a hundred yards from the barn and is completely isolated from all other buildings. To prevent the milkers from going into the milk-room, the milk is poured by means of a block-tin pipe through the wall of the milk-room into a large ice-lined block-tin tank, which is also the mixer for the milk of the entire herd. In the space of four minutes, by means of an ice-jacket, the milk is cooled from 33.88° C. (93° F.) to below 4.44° C. (40° F.). This is to rapidly remove the heat, which is conducive to bacterial growth. The milk passes through eight thicknesses of sterilized gauze on its way to the tank.

The milk-room is practically clean from a bacteriological stand-point, for the walls and floor are kept wet with clean water, and all dust is excluded. The milk is drawn into these jars (Fig. 50, page 246), in which it is to be transported. The jars are then sealed, packed in ice, and in a few hours delivered at the place where the milk is to be used for substitute feeding.

After this treatment of the milk I have had repeated bacteriological examinations made on its arrival at the Laboratory, with the uniform result that it has proved to be comparatively sterile, and at times it has contained either no colonies of bacteria or only one or two.

No antiseptic can, without danger to the infant, be used about the cow, while all the mechanical devices heretofore tried to take the place of manual milking have inevitably tended to impair the lacteal function of the udder.

CHARACTERISTICS OF COWS WHICH PRODUCE MILK SUITABLE FOR INFANT FEEDING.—Some of the marks which distinguish the breeds best adapted for infant feeding are:

I. Constitutional vigor.
II. Adaptability to acclimatization.
III. Notable ability to raise their young.
IV. Freedom from intense inbreeding.
V. A distinctly emulsified fat in the milk.
VI. A preponderance in the fats of the fixed over the volatile glycerides.

You must understand that the volatile glycerides do not exist in the mammæ, but are formed in the milk soon after the milking, and that in some breeds this occurs more quickly than in others, such as those from the Channel Islands.

By means of these distinguishing marks we can eliminate from the cows which we wish to use for infant feeding such breeds as the Jersey, Guernsey, and any others in which intense inbreeding has been carried on and in which acclimatization has not been perfected, leaving for our purposes such breeds as Mr. Gordon has here to show you,—namely, the Durham, Devon, Holstein-Friesian, Ayrshire, Bretonne, and Brown Swiss. These you will understand are types of the breed, though not in all instances pure bred. These breeds, of course, do not represent all of those available for substitute feeding, for we may mention many others equally good each in its country. For example, the Kerry of Ireland, the Red Polled of England, the Dutch Belted and the Flemish, also the Flamande and the Cotentine of France, the Norman breed of Normandy, and, besides the Brown Swiss just spoken of, and which you will presently see, the Simmenthal, sometimes called Bernese, of Switzerland, also the Chianina of Italy, and the Allgauer of Germany. I say very little about the native cow of this country, the "Red Cow," because through many generations of neglect and exposure in winter she has undoubtedly acquired an impaired digestion and does not respond readily to appropriate changes of food.

Mr. Gordon will now show you the types of those breeds which represent best in his herd the requirements of substitute feeding.

The first cow (Fig. 43) represents the best type of the milking Durham or Shorthorn. She has great constitutional vigor, great capacity for food, a perfect digestion, is of a placid temperament, not easily frightened, and yields a large quantity of rich milk, the analysis of which is as follows:

ANALYSIS 41.

Fat	4.04
Sugar	4.34
Proteids	4.17
Ash	0.73
Total solids	13.28
Water	86.72
	100.00

The physical characteristics of the Durham are variety in color, a white nose (this especial Durham is a strawberry roan and white), large size, rather small head, large udder, and a placid, intelligent, and rather refined appearance.

The next cow (Fig. 44), the Devon, has the same general characteristics as the Durham, combined with great gentleness and docility. The color is, as you see, almost uniformly red, with the nose generally white. They are of medium size and have medium-sized udders. They are very gentle and very vigorous. They come from an old south-of-England established breed, and have been known for centuries. They have never been intensely inbred or pampered. They have a fair capacity for food, are not easily frightened, and their digestion is good. They give a moderate quantity of milk of medium quality, the analysis of which is as follows:

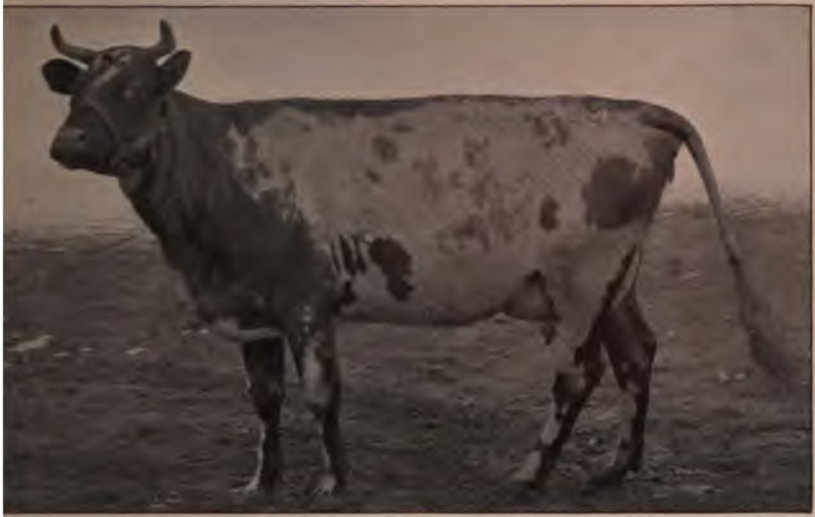


FIG. 43.—Durham. (Shorthorn.)

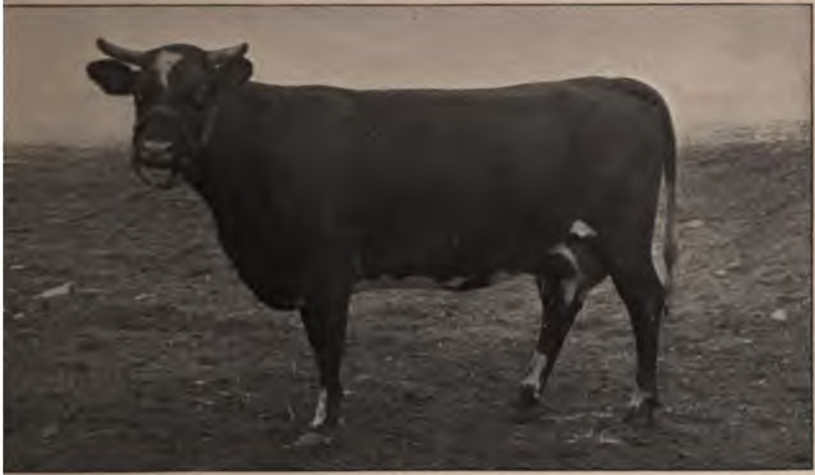


FIG. 44.—Devon.

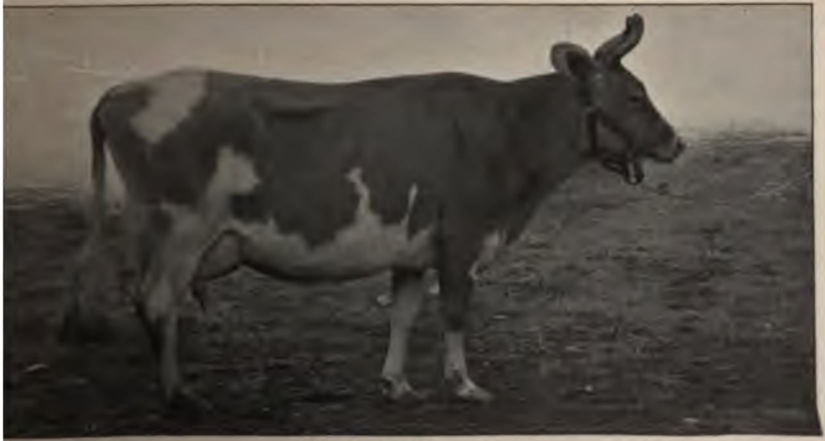


FIG. 45.—Ayrshire.

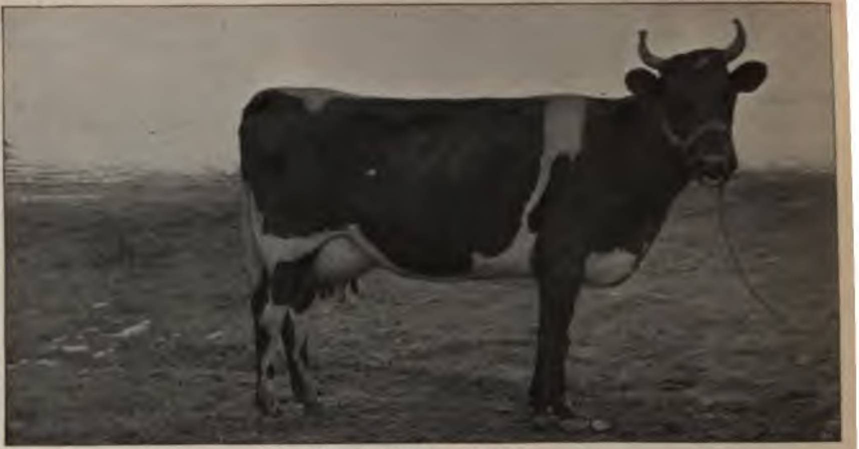


FIG. 46.—Holstein-Friesian.

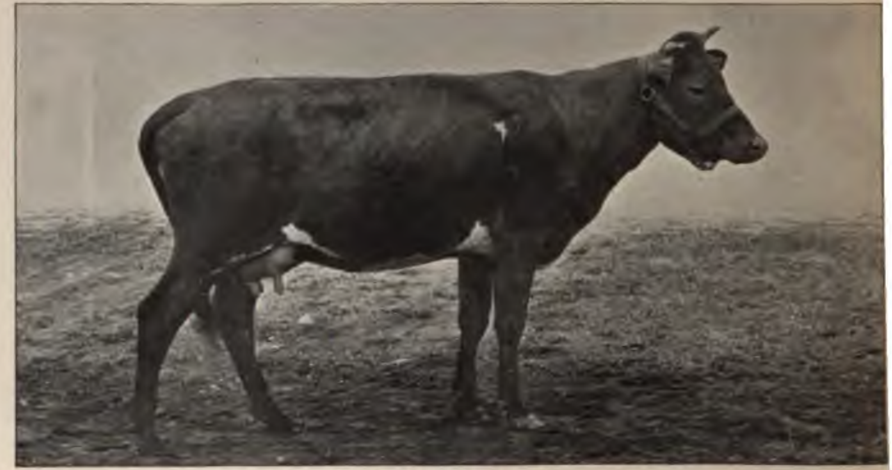


FIG. 47.—Brown Swiss grade.

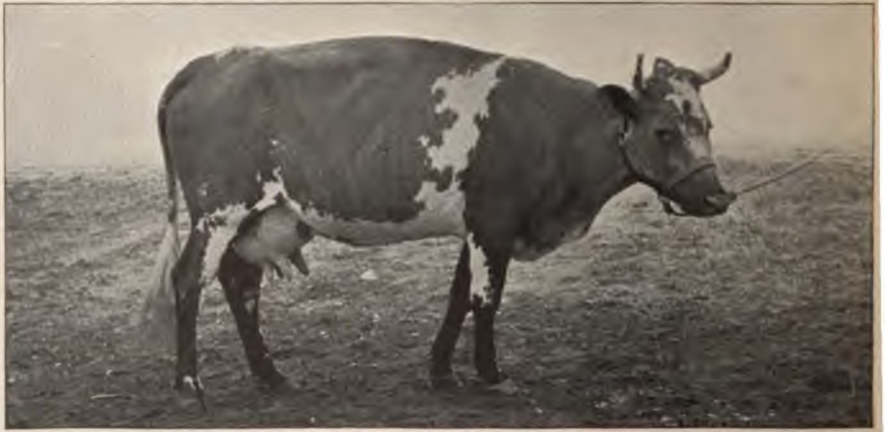


FIG. 48.—Bretonne. (Naturally straight back, arched from cold.)

ANALYSIS 42.

Fat	4.09
Sugar	4.32
Proteids	4.04
Ash	0.76
Total solids	13.21
Water	86.79
	100.00

The next cow (Fig. 45) is an Ayrshire, descended from a celebrated race in the south of Scotland dating back many centuries. Their constitutional vigor is great. They have great capacity for food, a good digestion, a temperament rather nervous, arising, probably, from an out-cross with the wild cattle of Chillingham. They are not so hardy as the Durham, but are very free from disease. The prevailing color is brownish red with white spots or flecks, though many of the best Ayrshires incline to a pure white or to a dark brown without white. This one is brown and white and is of medium size. Their horns turn upward and backward. They have large udders, and yield a large supply of milk, with the following analysis:

ANALYSIS 43.

Fat	3.89
Sugar	4.41
Proteids	4.01
Ash	0.73
Total solids	13.04
Water	86.96
	100.00

The next cow (Fig. 46), which is of the thorough dairy type, is called the Holstein-Friesian. This cow represents the most perfect milking animal known, having every characteristic of a cow suitable for our purpose, but her milk is so light in its total solids that it is not so profitable as the other breeds. These cows are usually black and white in color, with black noses. The fat-globules of their milk are very small and evenly distributed, and the emulsion is perfect. These cows are usually large, weighing about 54.5 kilogrammes (about 1200 pounds). This special cow is now two years old and is not full grown. They are very domestic and gentle. They have large udders, and yield a larger quantity of milk than any other known breed, although the analysis shows it to be poorer in quality:

ANALYSIS 44.

Fat	2.88
Sugar	4.33
Proteids	3.99
Ash	0.74
Total solids	11.94
Water	88.06
	100.00

The fifth cow (Fig. 47) is a Brown Swiss grade. The Swiss element is derived from the hardy race of the Alpine pastures. They are very vigorous, stand cold well, are docile and not easily frightened. They are rather under medium size, and are generally brown in color. The nose is black, with a mealy ring around it. They have a slightly dished face, and the udder is of medium size. They are very healthy, and yield a fair supply of milk of about the richness of the Devon, the analysis of which is as *follows:*

ANALYSIS 45.

Fat	4.00
Sugar	4.30
Proteids	4.00
Ash	0.76
Total solids	13.06
Water	86.94
	100.00

Finally, here is a little Bretonne cow (Fig. 48), known all over Europe as the "cow for the family." Cows of this breed have all the characteristics of the good domestic cow which I have already mentioned. They are blue-black or black and white in color, and have black noses, which are sometimes mottled and are rarely white. A distinguishing mark is that the mucous membrane of the mouth is always white, while that of some other breeds is black or gray and white. They are small, but have large udders, which produce a medium amount of milk, large, however, in proportion to their size. This special cow is cold from standing, and this is the reason that her back is arched.

I have mentioned the natural constitutional vigor of these cows, because certain breeds of cows in some localities do not appear to be able to resist the attacks of common diseases, such as tuberculosis. A notable illustration of this is represented by the Jerseys in America.

It is very important that certain precautions should be taken to prevent the use of cows which are affected with tuberculosis. It is probable that three per cent. of the cows whose milk is used for food are tuberculous. Where tuberculosis is developed to such a degree in the cow as to be dangerous to the consumer of the milk, the disease can usually be detected by a skilful veterinarian by means of the physical examination which is employed in cows. But, as it is a disputed question at present as to when the milk of a tuberculous cow becomes affected, it is wiser to adopt all measures of precaution known to science. Of these measures the one which is most efficacious in detecting even the incipient stages of tuberculosis is that which is used here on this farm.

The cows employed for the production of the primal milk-supply for the Milk-Laboratory have been subjected to the test for the diagnosis of tuberculosis. This test is known as the "tuberculin test." The method of making this test is as follows:

At about 9 o'clock P.M. the temperature of the cows is taken per rectum with an ordinary clinical thermometer. The temperature in healthy cows may vary from 37.7° C. to 39.7° C. (100° to 103½° F.), according to age, the weather, the condition of pregnancy, or the period of the day. As soon as the temperature of the individual cows is recorded, each one receives a subcutaneous injection of from 2 to 3 c.c. of a ten per cent. solution (1 c.c. of Koch's tuberculin to 9 c.c. of a one-half per cent. solution of carbolic acid in sterilized water), the proportion being adapted to the weight and vigor of the especial cow. This fluid, for convenience and uniformity, is introduced in the upper part of the right shoulder. After an interval of ight hours—that is, at 5 A.M.—the temperature is again taken per rectum, nd this procedure is repeated at intervals of three hours until 2 P.M.

At 5 A.M. the temperature should in healthy cows be slightly lower than hat found on the previous evening. Subsequently the temperature should ot rise above that of the first record at 9 P.M. No rise in temperature ccurs in a cow which is free from any tubercular affection. Where the emperature rises to 41.1° to 42.2° C. (106° to 108° F.), it indicates disease nd marks the cow as tuberculous, though even a lower reading sometimes arks the presence of the disease in cows whose normal temperature was ow.

No water should be given to the cow during the period of the experiment, because it is found that the temperature, as soon as the water reaches he stomach, is lowered to or nearly to normal, according to the amount and emperature of the water.

This test is a very delicate one, and records the presence or absence of the slightest tuberculous infection, even if the disease has not previously affected the cow in any way which can be detected by an ordinary physical examination.

At the point of inoculation there are marked tenderness and heat in cows that are tuberculous for many hours after the conclusion of the test, while in cows that are healthy the skin is not irritated by the use of the syringe.

I have now explained to you what I consider to be a very important part in accomplishing a successful substitute feeding. I shall at my next lecture describe the characteristics of the milk which is brought from the herd to the Laboratory, where it is modified.

LECTURE IX.

III. INDIRECT SUBSTITUTE FEEDING.—(Continued.)

GENERAL REMARKS ON SUBSTITUTE FEEDING—COMPARISON OF WOMAN'S AND COW'S MILK—MILK-LABORATORIES.

IN my last lecture I explained to you at the farm the methods employed for obtaining a primal milk-supply especially adapted to infant feeding, and the types of cows which experience has proved to be the best for this purpose. You will now understand that where human milk that is suited to the individual infant cannot be obtained, or if obtained cannot be regulated by modification, it is desirable to substitute for it the combination of elements which such a human milk represents. To accomplish this we must have materials which, while closely resembling the elements of normal human milk, are easily obtained.

Physiological experiments on the mammary gland show that the albumin of the milk is not directly an exudation from the lymph-vessels supplying the mammary gland, but that it is actually modified in the gland itself. We thus see that the mammary gland, besides being an elaborator for infant nutrition, is also a modifier. This suggests to us that the modification of milk is not contrary to nature's method of preparing food for infants. Following, therefore, nature closely, we have learned that the proper modification of absolutely pure and fresh milk is the vital principle which should underlie our efforts to perfect a substitute food. I have already shown you the best method of obtaining a stable and perfectly pure cow's milk. When this milk is obtained, how shall it best be modified?

In addition to the general principles which I have enunciated concerning maternal feeding, and which apply equally to substitute feeding, there are certain principles connected especially with substitute feeding to which I desire to call your attention before taking you to the Milk-Laboratory, in order that you may use the Laboratory to the best advantage.

The infant at the breast receives for its nutriment a fluid which is fresh, sterile, neutral, or faintly alkaline, which has a temperature of 36.7°–37.8° C. (98°–100° F.), furnished in an amount proportionate to the age and size of the consumer. It is this fluid which we have to copy in every possible detail when we undertake to prepare a substitute food. We should also consider as foreign matter, to be carefully avoided, any element which we know is not to be found in the milk we are copying. Thus, and thus only, can we arrive at the proper solution of this intricate question of substitute feeding.

The analyses of human milk, which I have shown you in a previous lecture (Lecture VII., page 179), teach us that there is a great capacity in different infants to assimilate a variety of proportions of the same nutritive elements. In all probability the infant needs a variety in its food to somewhat the same extent as does the adult. In order, therefore, to copy nature closely, we must have some means of preparing a food not only for the many but for the individual, and when introducing new methods for preparing a substitute food we must recognize the necessity for providing for many prescription possibilities. In this busy age of scientific rational medicine physicians all over the world demand, first, means of saving time, and second, exact methods of work, which in themselves soon become time-savers. In every branch of our art the tendency is growing year by year to systematize the detailed and laborious work of the individual for the common practical use of the profession at large. I have long felt that in some way the subject of substitute feeding should be reduced to a more exact system, and that an effort should be made to rescue this important branch of pediatrics from the pretensions of the owners of proprietary foods and the hands of ignorant nurses. With this end in view, I have given my professional assistance to the establishment of a system of milk-laboratories where the materials used shall be clean, sterile, and exact in their percentages. These laboratories have been placed under the control of educated, intelligent men in whom we have the same confidence that we have conceded to the pharmacist, and we can write directions for infants' foods and send them to these laboratories just as, in the treatment of disease, we write our prescriptions for the division of one drug or the combination of several. As the pharmacist has nothing to do with the various methods of treating disease, so the milk-modifier is simply required to carry out the directions and ideas of the physician. No special school of medicine need be represented. No special method of feeding need be undertaken. An opportunity has, however, for the first time in the history of medicine, been presented for the physician to carry out his own methods, and these methods for the first time to be judged on a fair basis. In this way only can each clinical observer, when lacking in success, be sure that it is the fault of the food he is giving, and not because the food has varied from what he supposed he had ordered.

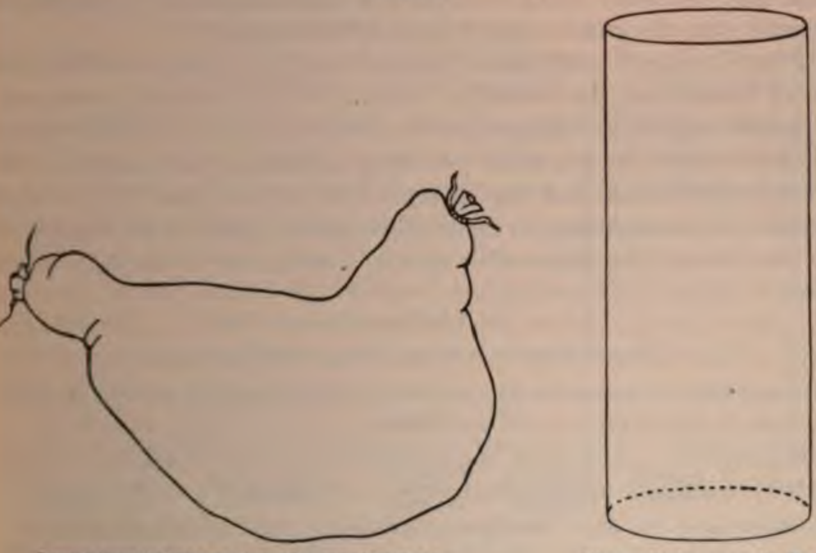
I have come to the conclusion that even slight changes in the percentages of the three important elements of milk of which we have most accurate knowledge—namely, the fat, the sugar, and the proteids—are of real value in the management of the digestion and nutrition of the infant, and that these changes are often necessary day by day as well as month by month. With this fact impressed upon us, we can well see that no one mixture will in all cases prove successful, but that a great variety in the percentages of the different elements of the milk will be needed in substitute feeding just as they already exist in maternal feeding. This explains the diversity of results obtained in the past with the same food by different practitioners.

The means for prescribing a diversity in the elements of milk, according to the idiosyncrasy of the digestion we are dealing with, is supplied by a milk-laboratory equipped with special machinery and controlled by educated milk-modifiers. From what I have previously said, you will understand that purity of the original material is the first object to be attained. This milk should be obtained from cows bred, fed, and cared for in the manner which was described in the last lecture, and, in order to insure absolute uniformity in the methods which I then explained to you, untiring vigilance must be used in the supervision of the farm, cows, and milk-house, and in the transportation of the milk from the farm to the laboratory. It is also necessary that the cows should be under the medical supervision of a skilled veterinary surgeon. These are all questions which to my mind have been definitely decided, but which now need time and attention devoted to them to insure their being systematically carried out. As in all other advances which are made in practical medicine, so also in this one it is well to adopt at once a high standard of work and to demand everything that can in any way tend to perfection. We may not always be successful in carrying out all the details, but until we are so perfection will not be arrived at. Bear in mind, then, the chain of facts which I have endeavored to simplify and explain to you, and understand that each link of that chain is of vital importance, because, if broken, the value of the whole chain may be lost. One end of this chain is at the milk-farm. We have followed it from the stall to the milk-house, and from the milk-house to the laboratory, and we must now so manage the continuation of this chain that it shall come unbroken and intact to the infant consumer.

Apparatus for Feeding.—Human ingenuity has not yet been able to devise anything which approaches the perfection of nature's apparatus for feeding, and the best that we can do to offset this complex mechanism is to adopt that which is exactly the reverse,—namely, an apparatus of absolute simplicity,—and thus combat the tendency to fermentation by preventing, through perfect cleanliness, the apparatus from becoming a source of fermentation. To accomplish this object the receptacle from which the infant is to be fed should be made of glass, in the form which will enable it to be most easily cleansed, and, as in the future the question of transportation will undoubtedly be a grave one, the receptacle should be such that it can be adapted to transit and not easily broken. For this purpose, what are practically test-tubes fulfil these indications best. These tubes have open mouths larger than those usually provided in the ordinary nursing-bottle, and, having no angles, are readily cleansed. The artificial receptacle is not self-regulating, and hence we must determine the amount of food in bulk which nature provides for the average infant at different ages, and from these average figures deduce the proper amount for the especial infant. The feeding-tubes are graduated for the more important periods of growth, for the purpose of continually impressing upon the mother and nurse what the physician often has the opportunity of telling them only at the beginning of the nursing period,—namely, that the error is in giving too much food rather than too ittle, an error, also, which naturally results when, as is commonly the case, he usual eight-ounce nursing-bottle is provided as the receptacle at the very eginning of infantile life.

I have found that I can easily convince most mothers of the mistaken eal of nurses who advocate giving the young infant large amounts of food, by showing them the size of the infant's stomach at birth and comparing this small tube which corresponds to the stomach's capacity with an eight-ounce nursing-bottle.

Fig. 49.



Stomach from infant five days old; capacity 25 c.c. (Natural size.)

Glass cylinder, capacity 25 c.c. (Natural size.)

I shall presently show you these tubes at the Laboratory, and I speak of them here merely to impress upon you the great importance of carefully attending to the smallest details in substitute feeding.

Nipples.—A nipple made of fine soft rubber adapted to the especial infant as to its size and the holes for the milk is substituted for the maternal nipple. These rubber nipples should be large enough to be turned inside out and carefully cleansed after each feeding. They should be boiled after being used, and kept in cold water with a little soda in it. They should be renewed frequently, the oftener the better: preferably a new one should replace the old one three times a week. It will be found that the rubber nipple has to be adapted to the taste of the especial infant, and that it often has to be changed as to its size, texture, and holes before the infant is satisfied with it and sucks satisfactorily from it.

Intervals of Feeding.—I have already shown you in this table (Table 42, page 182) the intervals of feeding which should be, as a rule, adhered to in maternal nursing. These intervals should also be adopted in substitute feeding, but the amount of food to be given now becomes a prominent feature in the division of the total amount of food which it is proper to give in the twenty-four hours, according to the age and development of the individual infant.

Amount at each Feeding.—The infant's weight and its gastric capacity quite frequently do not correspond. Yet there seems to be no doubt that the weight is a condition to which marked consideration should be given when we are attempting to determine so difficult a question as the proper amount of food to be given at each meal in the early months of life. The amount to be given at each feeding must be carefully regulated according to the gastric capacity, and I have stated in a previous lecture (Lecture IV., page 80) what the gastric capacity is at different ages.

I have arranged some tables (Tables 57 and 58) to show how the intervals of feeding and the amount of food to be given should correspond to the gastric capacity at different periods of the first year. I think that they will prove useful to you when you have to decide on the amount of food which it will be safe and wise to begin with in your cases. It is so important to avoid stretching so easily distensible an organ as the stomach that it is wiser to give too little rather than too much food in the early days of life.

TABLE 57.

General Rules for Feeding during the First Year.

The day feedings are supposed to begin with the 6 A.M. feeding and to end with the 10 P.M. feeding.

Age.	Intervals, hours.	Number of Feedings in 24 hours.	Number of Night Feedings.	Amount at each Feeding.		Total Amount in 24 hours.	
				Cubic Centimetres.	Ounces.	Cubic Centimetres.	Ounces.
1 week . .	2	10	1	30	1	300	10
2 weeks .	2	10	1	45	1½	450	15
4 weeks .	2	9	1	75	2½	675	22½
6 weeks .	2½	8	1	90	3	720	24
8 weeks .	2½	8	1	100	3¼	840	28
3 months .	2½	7	0	120	4	840	28
4 months .	2½	7	0	135	4½	945	31½
5 months .	3	6	0	165	5½	990	33
6 months .	3	6	0	175	5¾	1035	34½
7 months .	3	6	0	190	6¼	1125	37½
8 months .	3	6	0	210	7	1260	42
9 months .	3	6	0	210	7	1260	42
10 months .	3	5	0	255	8½	1275	42½
11 months .	3	5	0	265	8¾	1312	43¾
12 months .	3	5	0	270	9	1350	45

The first month being the most critical period for the infant's nutrition, as it is the time when the equilibrium of its metabolism is being established and its chance for life is least, especial interest should be attached to the series of careful investigations made at the Children's Hospital in St. Petersburg by Ssnitkin to determine the amount of food which should be given in the first thirty days of life. As the result of these investigations he deduces the rule, "the greater the weight the greater the gastric capacity." Ssnitkin's general results (Table 58) show that *one one-hundredth of the initial weight* *should be taken as the figure with which to begin the computation, and to this should be added one gramme for each day of life.*

TABLE 58.

Illustration of Ssnitkin's Rule to aid in adjusting the Food to especially difficult Cases in the first Thirty Days.

	Amount at each Feeding.		
Initial Weight.	Early Days.	At 15 Days.	At 30 Days.
3000 grammes . . .	30 grammes. (About 1 ounce.)	30 + 15 = 45 grammes. (About 1½ ounces.)	30 + 30 = 60 grammes. (About 2 ounces.)
4500 grammes . . .	45 grammes. (About 1½ ounces.)	45 + 15 = 60 grammes. (About 2 ounces.)	45 + 30 = 75 grammes. (About 2½ ounces.)
6000 grammes . . .	60 grammes. (About 2 ounces.)	60 + 15 = 75 grammes. (About 2½ ounces.)	60 + 30 = 90 grammes. (About 3 ounces.)

It is wiser always to accomplish first the proper digestion of the food, even if there is no gain in weight, and then, when once the infant is digesting well, to increase the amount of the percentages of the different elements. At times when the infant is digesting well, and even gaining, it will suddenly cry so hard and with such evident hunger that an immediate increase in the amount of its food is not only indicated but demanded, no matter what its age or weight. In these cases the stomach has probably grown rapidly and out of its normal proportion to the age and size of the child, and a larger supply of food is what is needed.

Our clinical experience proves to us that the average infant in the early months of its life does not digest unmodified cow's milk. The exceptional instances where it is tolerated have their counterparts in the success of many other foods diverse in their composition, and only serve to prove that the human digestion can at times be tampered with without much apparent injury, and to emphasize the general rule that the chemistry of the food which will produce the best average result should be the chemistry of human milk. Cow's milk, therefore, should be carefully compared with the standard human milk in order that we should know how nearly it resembles it. This table (Table 59) is a comparison of the average human milk and the average cow's milk, the figures representing the later and more reliable analyses:

TABLE 59.

	Woman's Milk directly from the Breast.	Cow's Milk as ordinarily received about 24 hours old.
Reaction	Slightly alkaline.	Slightly acid.
Water	87–88	86–87
Total solids	13–12	14–13
Fat	4.00	4.00
Milk-sugar	7.00	4.50
Proteids	1.50	4.00
Coagulable proteids . .	Small proportionately.	Large proportionately.
Coagulation of proteids by acetic acid	Not perceptible in test-tube.	Marked in test-tube; greatest with pure milk; less with milk diluted with water, and when 1 to 5 is not perceptible.
Ash	0.20	0.7

From this comparison we at once see that human milk and cow's milk differ as markedly from each other in their chemistry as they do in their clinical results as foods; and, as practically we must use cow's milk in substitute feeding, our wisest course is to modify it until we have approached the chemistry of human milk as closely as possible.

Before speaking of the various modifications of cow's milk which it is necessary to make in order that it may correspond to human milk, it will be well to say a few words about its properties as represented in the table (Table 59, page 235).

Reaction.—The reaction is stated to be slightly acid; and this is the case whether it has stood twenty-four hours with ordinary care or whether it is tested directly from the udder. This I have determined by direct experiment: so that practically the same amount of modification will be correct for the first twenty-four or thirty-six hours, so far as the reaction is concerned.

As it is wise in preparing a mixture for substitute feeding to make such a mixture approach as closely as possible in both taste and reaction to woman's milk, Harrington's experiments made at my request (Table 60) with lime water and ordinary cow's milk twenty-four hours old are important. Lime water was the alkali used in these experiments because it is the most simple adjuvant which we can use for making cow's milk alkaline, the amount of lime contained in it being so small that its addition in even considerable quantity does not materially alter the amount of the total mineral matter. As small an amount as one-sixteenth part, when added to ordinary milk, will render it alkaline, so that for making an acid milk correspond in its reaction to woman's milk, lime water is of great value, as it apparently does not produce any other changes in the milk. In addition to this, the taste of a mixture which is made from ordinary cow's milk, so as to correspond to the composition of woman's milk, is strikingly like that of woman's milk if it contain one-sixteenth part of lime water.

Harrington has made an estimate by actual experiment of the amount of lime water which is needed to produce an alkalinity in a mixture such as I have just mentioned which would correspond to the alkalinity of human milk. This table (Table 60) shows the results of his experiments.

TABLE 60.

Amount of Lime Water in Mixture.	Reaction.
25 per cent.	Strongly alkaline.
12.5 per cent.	Still strongly alkaline.
6.25 per cent.	Slightly but distinctly alkaline, and corresponding to woman's milk.

It must be remembered that these proportions of lime water are those required for ordinary milk twenty-four hours old, a much smaller proportion being needed to produce the same results when the milk is treated with the care which I showed you was employed at the farm connected with the Milk Laboratory.

WATER.—There is about one per cent. less of *water* in cow's milk than in human milk. Chemical analyses invariably show so large an amount of water in human milk that it is evident that the infant is intended to take, and can best assimilate, a very dilute food. We must bear this fact in mind in preparing a substitute food, as the precaution of supplying a thoroughly diluted food is of extreme importance in managing the infant's feeding both in health and in disease.

TOTAL SOLIDS.—There is about one per cent. more of *total solids* in cow's milk than in human milk. These solids in the milk are held partly in solution, partly in semi-solution, and partly in suspension.

FAT.—The percentage of *fat* in the average cow's milk and in the average human milk is the same. The glycerides of the fatty acids composing the fat in both cow's milk and human milk have been determined, yet our chemical and clinical knowledge of the nutritive value and digestibility of these, separately or collectively, has not arrived at a point where we can practically make use of this knowledge, and we therefore direct our attention to regulating in a milk modification the percentage of the fat as a whole.

Under this microscope you will see (Photo-micrograph, Fig. 61, page 259) a thin layer of milk which is represented by a transparent medium permeated with small globules of fat. This fat is simply held in suspension, which enables us to separate it easily by mechanical means. It is, in fact, in a condition which marks the milk as an emulsion.

SUGAR.—The *sugar* which is present in the milk of all mammals is of the variety called *milk-sugar*, or *lactose*. It is a simple and uniform element to deal with. Its percentage in cow's milk is 4.5, and in woman's milk 7. It is held in solution in the milk.

Regarding the kind of sugar which should be used in making up a substitute food, we have certain questions to consider which would seem to be important. Cane-sugar has been, and still is, a favorite form with which to regulate this part of the solid constituents of the food. The reasons given for using it have been its preservative qualities, as seen in the manufacture of condensed milk, and the theory that it is not liable to set up excessive so-called lactic acid fermentation, with its consequent disturbance of digestion, as has been supposed to be the case with milk-sugar. Cane-sugar in a concentrated form, as it is found in condensed milk, seems to act as a preservative. But when it is diluted, as in its administration to the infant, cane-sugar ferments very readily, and in this respect has no advantage over milk-sugar. Reasoning from analogy, we should say that as milk-sugar is the only form of sugar found in the milk of mammals, it is there for some good purpose, and that it is needed for the accomplishment of some process which takes place after the food has been swallowed. Both cane-sugar and milk-sugar are converted into glucose in the intestine. There seems, however, to be some difference in the degree to which they can be used for purposes of nutrition before they are converted into glucose. So far as is known, whether in plants or in animals, cane-sugar is merely a reserve, and cannot be used directly for nutrition. Milk-sugar, on the other hand, is probably not merely a reserve, but may possibly be utilized in the economy also for nutrition. Thus, Bernard has shown that seven grains of milk-sugar dissolved in an ounce of water could be injected under the skin of a rabbit without the subsequent appearance of sugar in the urine, while under the same conditions and in the same amount cane-sugar was found to be eliminated as foreign matter by the kidneys.

Milk-sugar undergoes no direct alcoholic fermentation, but it changes readily to lactic (possibly acetic) acid in the presence of nitrogenous ferments, while cane-sugar easily undergoes alcoholic fermentation, but changes to lactic acid less readily than milk-sugar. Cane-sugar, moreover, takes on the butyric acid fermentation more readily than does milk-sugar. The *bacillus lactis aërogenes* (Escherich) is present in normal digestion, and acts on the milk-sugar to produce an organic acid which drives out the more noxious forms of bacteria, which by their presence would interfere with normal digestion. When milk-sugar is converted into glucose, we physiologically have a gradual conversion into lactic acid, which may aid in the digestion of the albuminoids, thus giving us a very valuable addition to the means at our command for rendering modified cow's milk digestible.

Jeffries says, in reference to the different actions of the various kinds of sugar in the digestive tract, that it is important to note that starch, dextrin, inulin, cane-sugar, and dextrose afford material for the butyric acid fermentation, while milk-sugar does this only after completed hydration.

Escherich, in speaking of Brieger's bacillus, says, "Milk is coagulated with sour reaction first after several days (eight to ten) at the body temperature. With exclusion of air this bacillus cannot grow either in milk o milk-sugar solution, but will in grape-sugar."

We thus see that the milk-sugar offers less danger of the butyric aci ferment, which we know makes much trouble at times in the body, an that under certain conditions of the intestine it should be exempt from th assaults of Brieger's bacillus.

When we consider that by means of heat we can practically put an en- to the lactic acid fermentation, which may have begun to act upon the mil before it enters the stomach, it would seem that we are justified, on bot physiological and bacteriological grounds, in using the same animal sug in substitute feeding that is found in the infant's natural food, instead o introducing a vegetable sugar, which in milk is a foreign element.

The dangers from lactic acid are, at any rate, much exaggerated b writers on this subject.

PROTEIDS.—The proteids of normal human milk have quite a wid range in their variation; still, it is now well recognized that their averag normal percentage is very much below that of cow's milk. Assuming tha the percentage of proteids in human milk is 1.5, or between 1 and 2, it ca be stated that the relation of the percentage of the proteids in cow's mil and in human milk is as 4 to 1.5.

The proteids represent the nitrogenous elements of milk. They are artly in solution and partly in suspension, as is seen on filtering through orcelain, where nearly all the caseinogen is left behind with the fat, while a nall portion of the caseinogen and the other proteids is easily recognized the serum.

Of the total nitrogenous constituents of milk which are classed under e general term proteids, and of which the caseinogen and albumin are arts, the coagulable proteids in cow's milk are proportionately larger in mount than in human milk, so that under the same conditions a larger curd ill be formed with the former than with the latter.

Coagulation of Proteids.—In conjunction with Dr. Harrington and r. Townsend I have made some careful experiments as to the relative oagulability by acids of woman's milk, cow's milk, and cow's milk diluted with lime water and barley water in various proportions. The coagulation by rennet was not found to be a satisfactory or reliable test. The experiments were performed in the following way. Equal volumes of the fluids tested were placed in a number of test-tubes. Ten drops of acetic acid were then added to each test-tube. Each test-tube was then inverted slowly three times, so as to insure thorough, equal, and uniform mixing in all.

This table (Table 61) shows the results of these experiments, which may prove to be of considerable value:

TABLE 61.

Coagulability of Milk by Acetic Acid.

Test-tubes.	Mixture.	Coagulum.
1.	Woman's milk	No curd perceptible to the eye.
2.	Cow's milk, raw	Large curds.
3.	Cow's milk, boiled	Same as 2.
4.	Cow's milk heated by steam to 100° C. (212° F.)	Same as 2.
5.	Cow's milk . . . . . 2 parts. Water . . . . 1 part.	Finer than 2.
6.	Cow's milk . . . . . 2 parts. Lime water . . . . . 1 part.	Same as 5.
7.	Cow's milk . . . . . 2 parts. Water . . . . 1½ parts.	Slightly finer than 5 and 6.
8.	Cow's milk . . . . . 2 parts. Barley water . . . . . 1 part.	Same as 7.
9.	Cow's milk . . . . . 1 part. Water . . . . 4 parts.	Finer than 7 and 8.
10.	A mixture the composition of which was Fat . . . . 4.0 Sugar . . . 7.0 Proteids . . 1.5 Ash . . . . 0.2 Reaction . . Slightly alkaline (6.25 per cent. of lime water.) Total solids. 12–13	A very fine curd, finer than 9.
11.	Cow's milk . . . . . 1 part. Water . . . . 5 parts.	Same as 1: no curd perceptible to the eye.

When a few drops of mercuric nitrate solution were added to woman's milk and to cow's milk which had been diluted 1 to 5, as is represented in test-tube 11 in the table (Table 61), a fine coagulum was produced in the woman's milk, and a still finer one in the cow's milk.

There was found to be practically no difference as to the rapidity of the coagulation of the different mixtures whether the milk was not heated or was heated to 100° C. (212° F.).

Cow's milk taken directly from the udder was found to coagulate in just as large curds as when twenty-four hours old. It was found that there was practically no difference in the coagulation of raw, boiled, or steamed milk; also that the size of the curd depends on the dilution of the proteids, rather than on any especial property belonging to the substance with which the dilution is made. With lime water the result was the same as with water in equal amount, and barley water shows only a fractional difference from the results obtained with plain water.

ATTENUANTS.—In order to ascertain if the statement is correct which is so often made, that "attenuants act mechanically by getting between the particles of coagulum during coagulation and thus preventing their running together and forming a large compact mass," I have experimented as follows with various substances containing different percentages of starch:

In each of six test-tubes of equal calibre, and containing 5 c.c. of hot water, 10 c.c. of milk were placed. In test-tubes 1, 2, 3, 4, 5, and 6 were added equal portions respectively of some starchy foods, cracker-crumbs, and bread-crumbs. The proteids were then coagulated as before with acetic acid, and the results were as seen in this table (Table 62):

TABLE 62.

Test-tube.	Mixture.	Coagulum.
1.	Hot water and milk	Finest curd of all.
2.	Hot water and milk and starchy food	Not so fine as 1.
3.	Hot water and milk and starchy food	About like 2.
4.	Hot water and milk and starchy food	Not so fine as 2 or 3.
5.	Hot water and milk and cracker-crumbs	Not so fine as 4.
6.	Hot water and milk and bread-crumbs	Not so fine as 5.

There is no doubt that where no attenuant was added the curd looked decidedly finer, while where attenuants were used there was not a great deal of difference in the result obtained with the substances employed, except the possibly rather larger curd according as the attenuant contained a larger percentage of starch.

We may conclude, then, until something more definite is known concerning this rather theoretical method of treating the curd, that dilution with plain water is the most practical and efficient means at our command.

As the predigestion of the proteids is frequently recommended by physicians when the infant's digestion is normal as well as when it is weakened, it is well to say a few words about this predigestion in connection with substitute feeding.

Peptonized milk is cow's milk with its proteids partially or entirely preigested by means of the extract of pancreas and soda. There is no doubt hat the proteids of cow's milk are at times a source of trouble to the nfant's digestion, and that under certain circumstances they can with great enefit be treated by predigesting them for a time, and allowing a stomach vhich otherwise digests well to rest and recover its entire digestive power. It is of use also where a decided idiosyncrasy of the individual precludes he digestion of these constituents of the milk. In many cases the indigesion is attributed to a lack of power to digest proteids at all, while in fact the stomach is simply rebelling against an amount of proteids above the standard percentage, or against some other constituent. It would seem that, for the average infant, this predigesting of the proteids is contrary to nature's teaching. There are certain natural functions which should be allowed to act as they do on human milk, and it seems irrational and contrary to the laws of physiology not to encourage all the functions to act naturally, instead of forestalling their action and allowing them to fall into disuse and thus to be weakened. The infant's stomach is intended to digest proteids, and not to have the proteids digested for it. Clinically, also, the use of peptonized milk supports this view, for, so far as I know, no very brilliant results have been obtained from its use, except where the infant's digestion has been in an abnormal condition and one which has called for some decided relief from the proteid elements of milk. Peptonized milk, therefore, as a food for young infants is one which consists of too large an amount of digested proteids, too little sugar, and a very large over-proportion of mineral matter.

Ash.—The constituents of the ash of cow's milk have been analyzed with comparative care and success. I have already, in speaking of the differences which exist between cow's milk and woman's milk, stated the differences which exist in their constituents and the elemental percentages of those constituents. This question of the percentage of the ash practically does not enter into the modification of milk at the laboratory, as our knowledge has not yet advanced to that point where we can make use of what we know of these differences.

There are a few other questions concerning the composition of cow's milk in relation to its proper modification for substitute feeding which it will be well to speak of here.

Cow's milk, besides the elements which I have just spoken of and which I have represented in this table (Table 59, page 235), is supposed to contain a small portion of fibrinogen held in suspension. I have adopted the terms fibrinogen and caseinogen as recommended by Haliburton. They represent their respective elements as they actually exist in the milk before any change has taken place in them. After the milk has been drawn from the udder we have certain elements which we call *casein*, resulting from the *caseinogen*, and *fibrin*, resulting from the *fibrinogen*.

Cow's milk is also supposed to contain urea and citric acid.

In substitute feeding, the addition to modified cow's milk of some substance, such as starch in various forms, is so frequently recommended that I think it will be well to state my opinion of this practice.

This brings us to the consideration whether starch should be made a part of an infant's food. Physiologically, we know that during the first ten or twelve months of life the function of converting starch into sugar is in the process of development. It is true that a partial conversion of the starch can be performed at quite an early age, and, in exceptional cases, to a much greater extent than by the average infant. It is rational to suppose that when a function is being developed it should not be taxed with a trial of the use which will later be demanded of it. That is, a function develops more perfectly if its power is not exerted too early. With these facts before us, and simply recognizing that the best known food for infants, woman's milk, does not, under any circumstances, contain starch, I believe that starch should not form a part of the infant's food in the early months of its life.

The question whether milk should be boiled or steamed is one which is not of much significance, and can be settled according to the fancy of the individual practitioner, a greater or less destruction of the bacteria contained in the milk taking place according to the degree of heat to which it is submitted. My own experiments in comparing steamed with boiled milk show that the odor and taste of boiled milk are present when milk is steamed, but to a much less degree than in boiled milk; also that while a thick scum is formed on milk boiled for twenty minutes, which is tenacious and does not disappear on shaking, only a very thin scum forms on milk steamed for twenty minutes, and that this is not tenacious and almost entirely disappears on shaking.

BACTERIOLOGY.—A few matters concerning the bacteriology of cow's milk can best be considered in connection with the subject of substitute feeding. Respecting this question Dr. J. A. Jeffries very aptly remarks "that it is a curious fact that, while older people are chiefly fed on sterilized food,—that is, cooked food,—infants are fed on food peculiarly adapted by its composition and fluid state to offer a home for bacteria." In some experiments made by Jeffries agar-agar cultures were made before and after the different fluids were sterilized, and the colonies of bacteria were counted. His results coincide with those of previous experimenters,—namely, that steaming for fifteen minutes is sufficient to kill the developed bacteria, while a second steaming is necessary for complete sterilization. Out of one hundred and twenty lots of milk steamed but once, all but four or five showed distinct signs of change within a month, while the majority of those steamed twice did not change at all.

Jeffries's experiments also show that spores develop slowly, and, indeed, rarely form, in milk, which, as he says, is an excellent medium for growth, while spore-formation among bacteria, like seeding among higher plants, is a phenomenon of impaired growth. He also explains the preservation of some of the milk steamed but once by the absence of any enduring spores from the start. In an article of very great interest and value to the practising physician "On the Bacteria of the Alimentary Canal," Jeffries has reviewed, at my request, the work done by the various bacteriologists:

"Miller, De Barry, and Escherich have shown that living bacteria are to be found in the stomachs of men and animals, and the former author has also clearly proved that bacteria can pass through the stomach into the intestines and live for a considerable time. . . . Of the morphology and biology of the forms found in the stomach little is known. The field is a new one, and the species have not been sufficiently described to enable others to recognize them with certainty. Miller has found five kinds which give off carbonic dioxide and hydrogen gas, lactic, acetic, and butyric acids being formed. . . . Of the flora of the intestines much more is known than of that of the stomach. The researches of Brieger, Vignal, Stahl, and Escherich have now proved that a large number of species may occur in the fæces. Brieger isolated two new kinds: one a micrococcus, which turns grape- or cane-sugar into ethylalcohol, with a trace of acetic acid; the other the well-known Brieger's bacillus. This species occurs in the fæces in vast numbers, ferments sugar, and decomposes albumins. Vignal isolated ten species from the fæces, six of these also being found in the mouth. Of these some produced acid fermentations and gas, but unfortunately they were not sufficiently studied to show their effects on digestion. . . . Escherich studied especially the fæces of infants, and found a large number of kinds of bacilli, among them a small bacillus capable of converting milk-sugar into lactic acid, carbonic dioxide and hydrogen gas being evolved, either in the presence or absence of air, a facultative anaërobic species, his *bacillus lactis aërogenes*. Escherich established, by the examination of a large series of cases, the fact that the kinds occurring in the fæces vary with the food,—that is, the intestinal contents. . . . Starting at birth with the sterile meconium, consisting of mucus, epithelium, and the like, infection by the mouth and rectum quickly occurs, and in a short time almost any form may be found, but chiefly such putrefying forms as *proteus vulgaris*.

"With the suckling of the infant and the substitution of the refuse of the milk and secretion of the digestive tract for the meconium, a sharp transition occurs. Instead of the generally distributed forms causing decomposition, only two kinds are regularly found, *bacillus lactis aërogenes* and Brieger's bacillus; the first chiefly in the upper parts of the intestine, the second in the lower parts. Passing on to the period of mixed diet, quite a number of forms appear, among them the *streptococcus coli gracilis*, the *putrefying green fluorescing*, a *tetrad coccus*, and *several kinds of yeast*. This brings us to the pith of the subject: Why are the flora so limited in the milk-eating infants and so diverse in others? What drives the forms found in the meconium out? That they can live there is clear, as shown by their presence the day before. Again, what prevents forms so common with meat diet from gaining a footing? It is not the milk alone, for milk is an almost universal food for bacteria, and all the kinds found in the intestines thrive in it.

"According to Escherich, the *bacillus lactis aërogenes* and the milk diet keep out the other forms.

"Formerly," continues Jeffries, "even before the action of ferments and putrefactive processes were clearly understood, the significance of this question was seen. The chyme is a mass admirably adapted for putrefaction or fermentation, yet ordinarily but little of either occurs. It is an alkaline or, as in the milk-fed, acid mixture rich in albumins, fats, and the starch group, amply provided with water and warmth. Such a mixture outside the body at an equal temperature would quickly decompose. It was generally held that some preservative action was exerted by the digestive juices: Bidder's and Schmidt's dogs with biliary fistulæ were supposed to explain the whole. These dogs, deprived of their bile, became emaciated, and suffered from diarrhœa and decomposition of the intestinal contents. Thus it seemed clear that in the absence of the bile decomposition occurred,—that is, that the bile was a powerful germicide or germ-inhibitor. During the last few years, however, different results have been obtained in cases of biliary fistula. Rohmann's dogs did not suffer from diarrhœa or putrefaction in the intestines, hence it is clear that the bile is not the cause of prevention. The diarrhœa, if present, is due to the large amount of fat passed on to the lower intestines.

"Maly and Emich ascribed value to the bile acids, especially the taurocholic, basing their results on crude methods; and Lindenberger, really leaving the subject, attributed the action to the organic acids in combination with the bile.

"All this argument and belief in the decided germicidal action of the bile occurred in the face of the well-known fact that bile itself will decompose.

"From a bacteriological stand-point, Miller has shown that a ten per cent. solution of bile, if anything, favors growth. Macfadyen has studied bile, bile salts, and bile acids in varying strengths. The only positive results were got with the acids; these arrested the development of bacteria if sufficiently strong, especially taurocholic acid. Neither acid had much effect, and least of all on the forms causing putrefaction. *Proteus vulgaris* was only arrested by a strength of from one to two per cent. The pathogenic forms were arrested by a much smaller quantity, from one to one-half per mille.

"It is thus clear that other causes must be sought for. One of these is to be found in the lack of oxygen in the intestines, as pointed out by Escherich and strangely forgotten by others. There is certainly very little free oxygen in the chyme, if any; not only is it scarce in the food at the start, but is taken up by the chemical changes during digestion, and also by the intestines. This clearly must be a potent factor, for the majority of bacteria require a fair supply. Accordingly, many bacteria are found in the fæces which will grow in the air, as shortly stated by Macfadyen, and the mass of those isolated in the air are able to grow without it.

nerich, though he points out the absence of oxygen, does not seem
full value, or rather forgets the subject in treating of the action of
acid bacillus. As before stated, this form is regularly found in
mbers in the upper part of the intestines of milk-fed children.
onverts a considerable part of the milk-sugar into lactic acid, and
ents the other forms from growing,—most forms being susceptible
d reaction, and especially to the organic acids. The action of sali-
d is known to all, and recent experiments, of which Macfadyen's
) are the best, show acetic, butyric, and lactic acids to be efficient
ibitors in strengths of from one to one-half mille according to the

milk-fed infants another point is the comparative inability of bac-
ttack casein, so that the bacteria are literally starved.

may therefore conclude that the bile acids, lack of oxygen, lack of
albumins, and the presence of organic acids are the causes of immu-
n the putrefying and fermenting kinds of bacteria to which we are
Certain forms are probably limited by the lack of water,—that
fluid state,—doing poorly if unable to swim freely about. It must
ever, be supposed that bacteria are scarce in the intestines; on the
, they form a large part of the dry substance of the fæces.

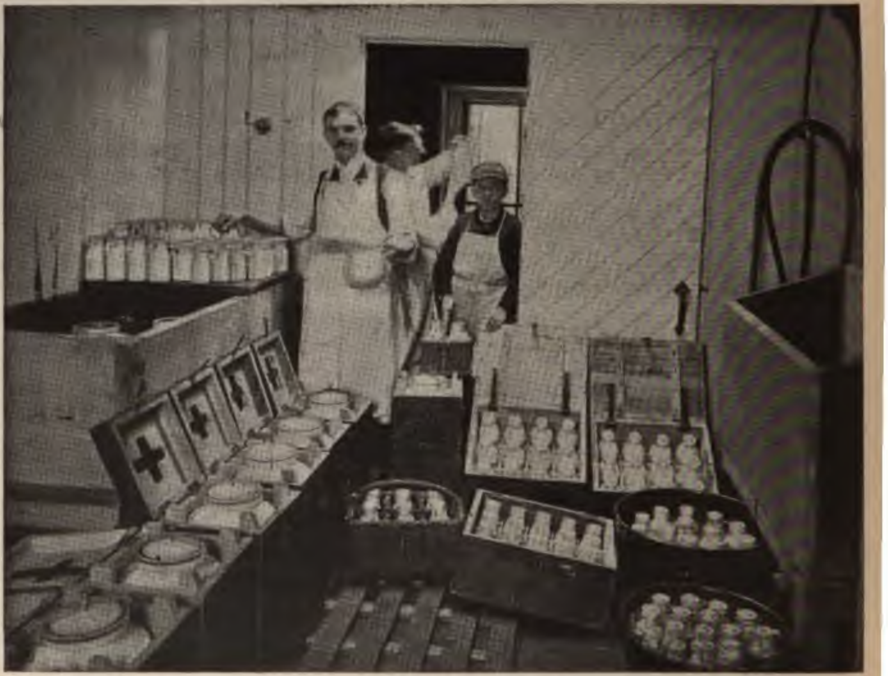
e ferments act by the production of various acids, chiefly derived
milk-sugar. In small amounts, as in the case of the *bacillus lactis*
s, the acid seems to be of benefit, and certainly does no harm, as it
y occurs in healthy breast-fed infants. In large amounts, however,
tend to over-acidify the contents of the intestines and interfere with
n of the digestive fluids."

K-LABORATORY.—I shall refer again to this analysis (Analysis
218) of the average milk of herds of cows when I am explaining
hod by which those who are too far away from medical centres to

laboratory should be situated in a healthy locality. It should be as free as possible from contaminating influences, should be kept absolutely clean, and every aseptic precaution against the harboring or development of pathogenic organisms should be taken.

From the moment that the milk is delivered from the farm at a temperature of about 4.4° C. (40° F.) it should be watched over and cared for with scientific accuracy during the whole process of the modification which it undergoes in the laboratory. The milk-rooms should be cool and free from dust, and isolated, so far as possible, from other parts of the laboratory.

FIG. 50.



Milk-room.

There should also be an entirely separate room where the returned packages and all articles received from the homes of the consumers should be directly brought from the street or wagons, and where these articles can be immediately sterilized in apparatus reserved for this purpose.

The modifying materials used in the laboratory should be carefully kept for use in glass vessels, and at a temperature of about 4.4° C. (40° F.), to prevent the growth of bacteria. The reason for this is that milk modified from materials free from bacteria is better for the infant than milk in which the bacteria have been destroyed by heat. Therefore the utmost care is necessary in all parts of the process and in every department of the laboratory.

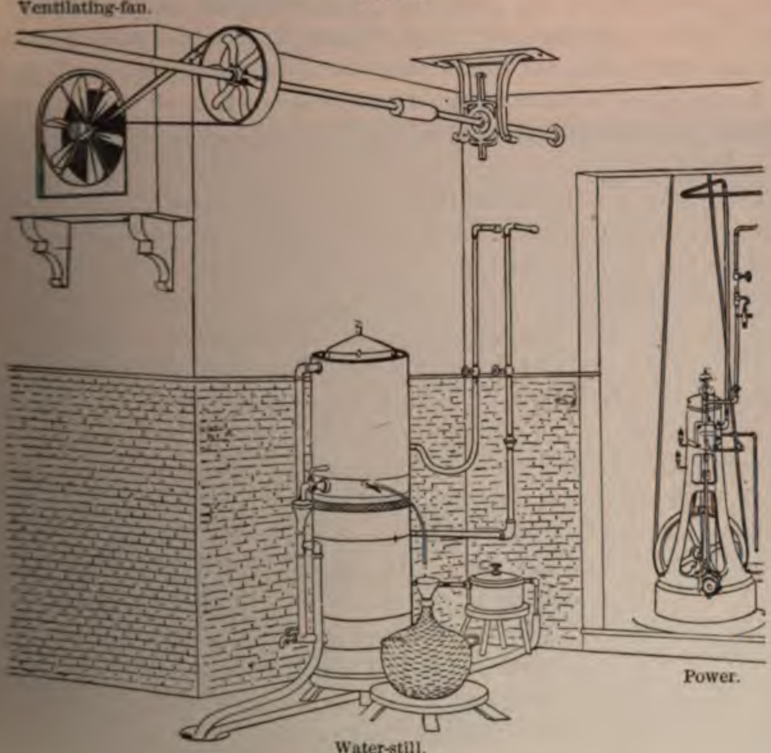
A special room should be provided for the milk-modifiers who are to put up the mixtures required by each prescription.

There should also be a room where the milk is separated by means of machinery and where it can be tested and steamed.

The office at the laboratory should be entirely separate from these work-rooms, so that customers coming to leave their orders should not go near the materials used for modification and thus possibly contaminate them.

It is necessary, also, that all odors should be excluded from the work-rooms, as milk absorbs odors very quickly.

Fig. 51.


Ventilating-fan.
Power.
Water-still.

Separating-room.

It is hardly necessary to say that the employees of a laboratory, whether they be in the office or in the work-rooms, should be intelligent and interested in their work.

I have explained to you in a general way the chief requirements of a milk-laboratory. I will now take you to the Laboratory and explain to you on the spot the various details which must be understood by the physician so that he can intelligently order what is best fitted for the infant under his charge.

Milk-Room.—We are now in the milk-room (Fig. 50, page 246), where the milk is received on its arrival from the farm.

The milk from the farm is delivered here in the milk-room within a few hours from the time of the milking. You saw how it was aerated at the farm and cooled to about 6.66° C. (44° F.), and you now see that on its arrival at the milk-room its temperature is found to have been held by means of ice during the transportation below 7.22° C. (45° F.). You see how it has been transported in these boxes and how the man in charge of the room has had it placed in the tanks of ice-water.

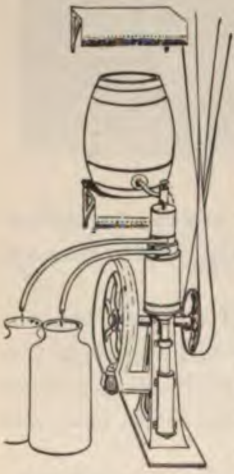
This milk, as a result of the especial manner in which the cows have been fed and cared for and the selection of them according to the proper breed, may be said to have an almost uniform percentage of its elements. Even at those times of the year when the percentages of the different elements of milk commonly vary from changes in the pasturage and in the habits and surroundings of the animals, the milk of these cows, which have their food supplied to them in stated rations at one time of the year as well as another, is not subject to the elemental variations which occur in the milk of ordinary cows.

Having seen here in the milk-room the methods by which the milk is treated and is kept uncontaminated, we will visit the separating-room, where the milk is taken to prepare it for the modifying clerk.

SEPARATING-ROOM.—This room (Fig. 51, page 247) is arranged and cared for in very much the same way as is the milk-house at the farm. The walls are of white tile, and the ceilings are of material which can be washed and scrubbed. The floor is of asphalt, impenetrable to water, and is kept thoroughly moistened and free from every kind of dirt and dust.

Ventilator.—In addition to the precautions against pathogenic germs, which I have already explained to you, the air of the separating-room is kept fresh and pure by means of this *ventilator* which you see in the corner of the room. It consists of a large steel fan, which revolves at the rate of two thousand times a minute, and by the force of its current carries away any flies or particles of dust which may come within its reach.

FIG. 52.



Centrifugal separator.

Separator.—Here is a piece of machinery of great delicacy, called the *Centrifugal Separator.*

This separator is made to revolve six thousand eight hundred times in a minute, and works with such searching effect on the milk that only a small percentage (0.13) of fat remains in the separated milk.

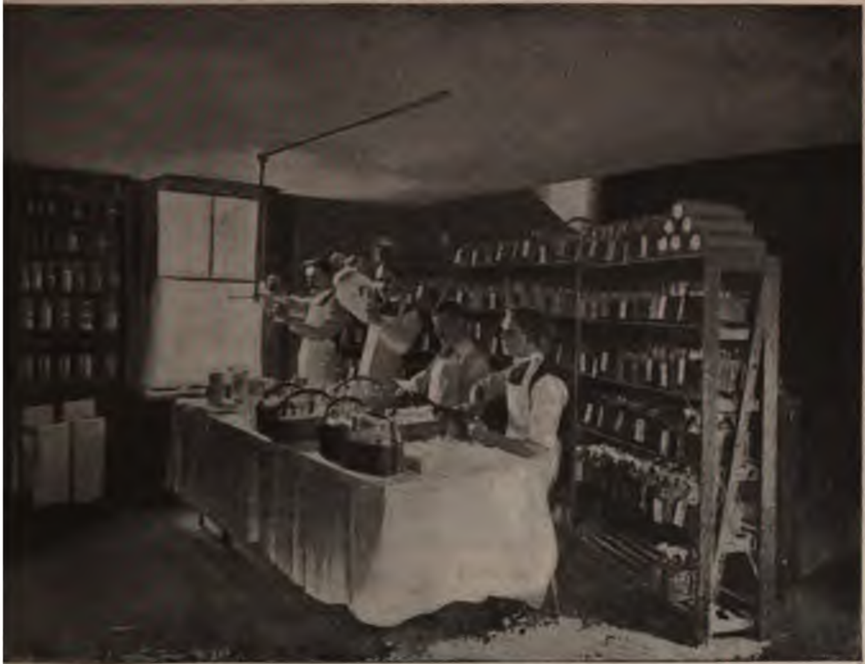
The utility of the separator, however, does not consist wholly in its absolute withdrawal of the fat from the milk and in providing cream as fresh as to time as is the separated whole milk: it accomplishes two other very important results. First, by its great centrifugal force it separates from the cream and the separated milk any dirt or foreign matter of any kind which necessarily gets into every milk, and thus provides at once a practically clean milk, a most important result from a bacteriological point of view. Secondly, the resulting cream has an almost table percentage of fat,—sixteen per cent.,—the importance, of course, of his being in its stability, and not in its special percentage.

Still.—We also have in this room (separating-room, p. 247) a still for reshly preparing each day distilled water.

In this next room you will see the steam-power (represented to right of eparating-room, p. 247) which runs the ventilating-fan, the separator, the ater-still, and the sterilizer which I shall presently describe to you.

Modifying-Room.—We are now in the modifying-room, where the ilk is tested, where the materials for preparing the food are brought from e different rooms when needed, and where the modification of the milk is mpleted.

Fig. 53.



Modifying-room.

Babcock Milk-Tester.—To be doubly sure that the chemistry of the ilk is what we suppose it to be from the uniform nature of the primal ilk-supply, we take advantage of the knowledge which we have concernng the changes most likely to take place in certain elements of the milk.

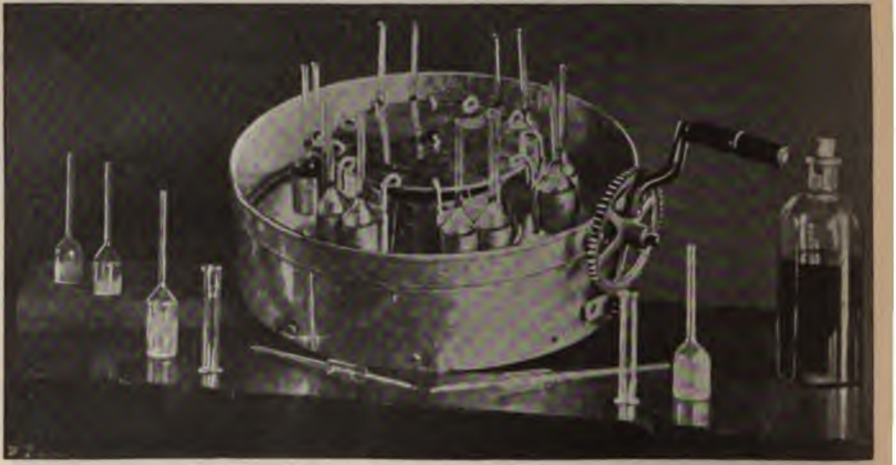
The percentage of the proteids, of the sugar, and of the mineral matter n the milk of a herd of this kind, where uniformity in the feeding is the ule, is not apt to be appreciably affected. But the percentage of the fat in ndividual cows differs from day to day, and thus slightly affects the perentage of the fat in the milk of the herd.

The fat, then, being the element by which we know whether each milkng gives a uniform product, we test this element by means of what is called he "Babcock Milk-Tester." I have here on this table the Babcock machine, and I will have the percentage of the fat in a specimen of this morning's milk tested for you.

The peculiar feature of this method of ascertaining the percentage of fat in milk, as described in the Wisconsin Experiment Station, Bulletin No. 24, July, 1890, consists in placing these test-bottles containing the acidified milk in a centrifugal machine, by the rapid revolution of which the fat is made to separate quickly and completely. The milk is acidified in order that the proteids, casein and fibrin, may be changed to soluble acid albumins, which offer less resistance to the rising and aggregation of the fat-globules.

Approximately equal volumes of milk and commercial sulphuric acid of 1.82 specific gravity are mixed in a test-bottle with a long graduated neck. This pipette, delivering about 17.5 c.c. of milk, and this measuring cylinder for the acid, are used. The acid is in this large bottle to the right of the machine. The bottles are whirled for several minutes at a temperature of 93° C. (200° F.) in a horizontal wheel making from seven to eight hundred revolutions per minute. This wheel is surrounded by a copper jacket, which may be filled with hot water for heating during the test. The separation of fat by gravity alone is not complete even when the bottles are left standing for several hours. By centrifuge, however, a perfect separation is accomplished in a few minutes. If whirled at once, no heat need be applied, as that caused by the strong acid and milk is sufficient. After whirling, the bottles are filled to the neck with hot water, returned to the machine, and whirled for one or two minutes longer, after which they are filled with hot water to about this seven per cent. mark, and the machine is again turned for a short time, the temperature being kept up by means of a lamp or by filling the jacket with hot water. The fat separates and its percentage is noted while still liquid, preferably at about 65° C. (150° F.), the reading

FIG. 54.



Babcock milk-tester.

giving the percentage of fat directly without calculation and being easily taken to 0.1 per cent.

This daily testing of the fat enables the modifier to preserve the accuracy of his material, and to correct any variation in the percentage of the cream as it comes from the separator.

The milk this morning shows four per cent. of fat, and therefore we conclude that we are dealing with the usual uniform milk expected to come from the farm. The average and almost stable analysis of this milk throughout the year shows a percentage of fat of 4.00, and is the basis on which the office clerk makes the calculation by which the percentage of the fat called for in the various prescriptions is exactly obtained. Knowing the exact percentages of this milk, the office clerk can, by a simple mathematical formula, give the required directions on the modifying clerk's formula for obtaining whatever percentages of the other elements the physician may call for.

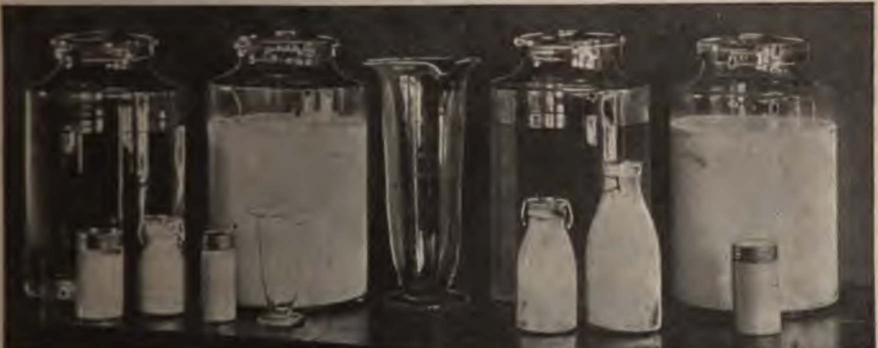
Here are the figures (Analysis 46) which have been found to result from many analyses of the milk of the herd which you saw at the farm:

ANALYSIS 46.

Fat	4.00
Sugar	4.30
Proteids	4.00
Ash	0.65
Total solids	12.95
Water	87.05
	100.00

I have also had placed here on another table for your inspection the modifying materials used for making up the prescriptions.

FIG. 55.



Modifying materials.

In this large glass jar on the left side of the table is the stable cream obtained from the separator, which is used in obtaining the prescribed percentage of fat. On the right side of the table is another large glass jar which contains the separated milk, also of stable percentage, obtained from the separator, and which is used for obtaining the different percentages of the proteids as called for in the prescription.

We must, of course, allow that the *cream* as well as the separated *milk* contains its own definite percentages of sugar, proteids, and mineral matter. This analysis (Analysis 47) shows the percentages of the fat, sugar, and proteids in this cream and separated milk :

ANALYSIS 47.

	Fat.	Sugar.	Proteids.
Cream	16.00	4.00	3.60
Separated milk	0.18	4.40	4.00

To provide the means for adjusting the percentages of the *sugar* which are called for, a carefully prepared twenty per cent. solution of milk-sugar and distilled water is used, and is kept in this large glass jar which stands beside the cream-jar. The reaction of the food is adjusted by means of the *lime water* which you see in this large glass jar beside the separated milk, and which is freshly prepared every day.

The other jars on the table contain specimens of cream of different percentages, and preparations of *oats*, *barley*, and *wheat*, which are freshly prepared at the Laboratory each day, and which can be used for infants when they are old enough to have starch added to their food.

With these modifying materials the modifying clerks combine each infant's food according to the prescription before them, and pour it into the glass tubes from which the infant is to nurse. These tubes, which you see standing in their baskets on the modifying clerks' table, have been especially devised as the most practical for general use, are adapted both for transportation and for use as nursing-bottles, and are easily cleansed.

There are two sets of clerks. (See page 249.) One set is engaged in modifying the milk according to the prescriptions. As soon as the tubes are filled by the modifying clerks they are passed on to the stoppling clerks, who immediately seal them with aseptic non-absorbent cotton especially prepared for this purpose, and place them in these baskets adapted as to their compartments to the number of feedings ordered for the special infant. Here are some baskets which hold eight, some which hold ten, and some which hold four tubes. The tubes are kept on tube-racks within easy reach of the modifying clerks. Each basket has its own label attached to it, with the address of the person to whom it is to be sent.

The rule of absolute cleanliness is carried out in every possible detail, from the table on which the materials are combined to the dress and hands of the clerks.

When the milk has been separated, recombined according to the prescriptions, stoppled, and placed in the respective baskets, the baskets are taken from the modifying-room to the separating-room.

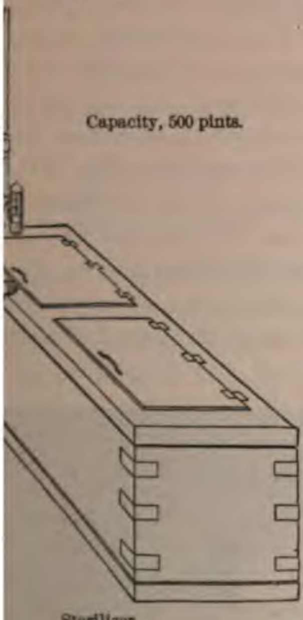
STERILIZER.—We will now return to the separating-room (page 247)

he baskets placed in this large sterilizer (Fig. 56), which has a
f 240 kilogrammes (500 pints).

erilizer is so arranged that the steam which passes through it can
ted so as to produce any degree of heat required up to 100° C.
. This is accomplished by a regulator attached to the steam-pipe,
u see, the man in charge of the heating of the food, by keeping his
he regulator and his eye on the thermometer which is fitted to the
can subject the baskets and the tubes in them to whatever degree
ordered, and of course for the length of time required.

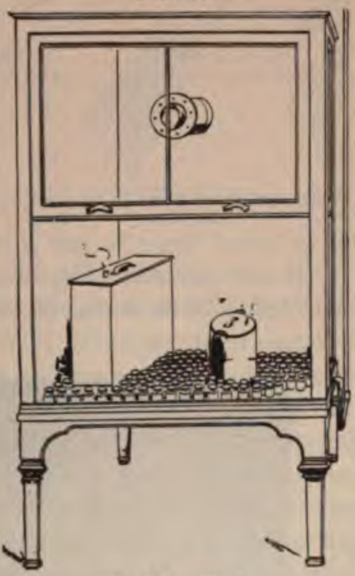
the food has been heated, the baskets are taken out of the steril-
placed in the cooling-tank, where the temperature of the food is
13.3° C. (38° F.).

FIG. 56.



Sterilizer.

FIG. 57.



Special sterilizer.

baskets are then placed in the delivery-wagon, which quickly
hem to their various destinations.

a special prescription at an unusual time of the day is called for,
d in this hood and special sterilizer (Fig. 57).

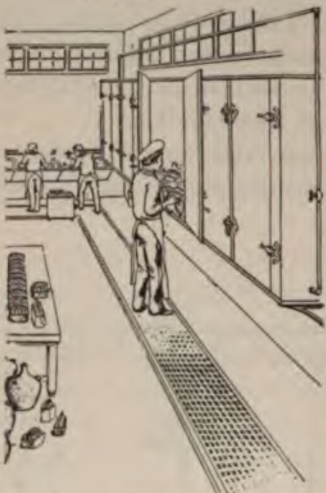
the baskets are delivered at the homes of the consumers, the
nd tubes of the previous day are returned to the Laboratory.
y reach the Laboratory they are taken directly from the street to
room (Fig. 58), which is entirely shut off, as I have before told
the rest of the Laboratory.

-ROOM.—Here in the wash-room (Fig. 58), in order to carry out
the aseptic precautions, the baskets and everything which has
rned to the Laboratory are placed in this special sterilizer belonging

to the wash-room. You observe that the baskets and tubes are just being taken out of the sterilizer. The bottles, after being sterilized, are thoroughly washed in these tubs, which are especially adapted for this purpose, in a solution of soda and water. All the tags and stoppers are destroyed after sterilization. The baskets are of woven willow, and are easily kept sterile.

FIG. 58.

Aseptic precautions.



Wash-room and sterilizer for returned materials.

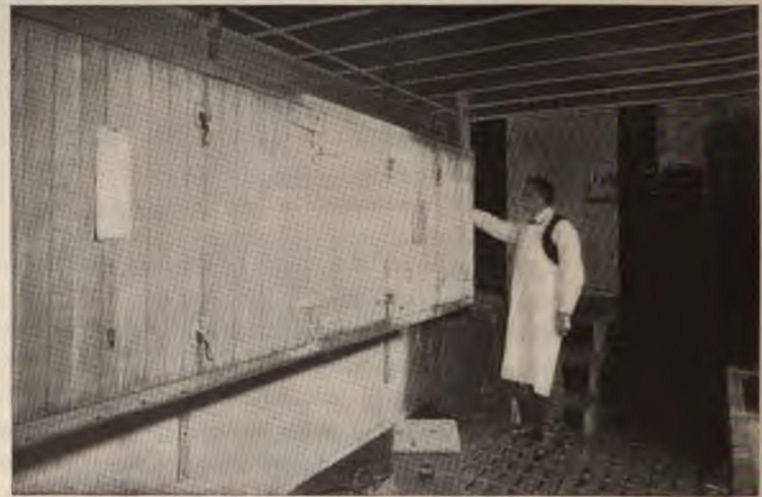
In this way, always guarding against possible infection of all kinds, the Laboratory enables us to make use of the chemical and bacteriological knowledge which we have acquired in connection with the feeding of infants, and fulfils the requirements of that system of substitute feeding which up to the present time has proved to be the best.

You will now have an opportunity of seeing the returned baskets and tubes actually steamed in the sterilizer (Fig. 59).

The doors of the sterilizer are tightly clamped, and Mr. Gordon has just ordered the steam to be turned into it.

MODIFICATION.—We will suppose that you wish to prescribe some modified milk for an infant four months old, with normal digestion and of normal weight and general development. The regular prescription-blank issued by the Laboratory can be used if you have one, but, of course, a milk-prescription can be written as you would write a prescription for a

FIG. 59.



Sterilizer, containing returned baskets and tubes.

Here is one of the prescription-blanks that I am in the habit of
this special Laboratory, and which I will fill with some supposed
s.

PRESCRIPTION BLANK.

	Per Cent.		
. . . .	4 00	Reaction	Slightly alkaline.
ar	7 00	Number of Feedings	7
. . . .	1 50	Amount at each Feeding . .	135 c.c. (℥ 4½)
atter		Heated for	20 minutes.
er		Heated at	75° C. 167° F.
rections.		Remarks.	
		Infant's Age?	4 months.
For Whom Ordered.		Infant's Weight?	14 pounds.
		Signature.	M.D.

ll direct the percentage of fat to be 4, that of sugar 7, that of the
1.5. I shall order the reaction to be slightly alkaline.

gard to the question of the reaction, it can be left to the milk-
as we leave to him the carrying out of other directions contained
escription. If the milk brought to the Laboratory on the special
we are sending our prescription has been produced from cows
have previously described, on sugar-beets, the milk may be already
ly alkaline for an infant's digestion when normal. If, on the con-
milk has its usual acid or amphoteric reaction, the milk-modifier
e it slightly alkaline, in accordance with our prescription and
as the milk of the special day has a greater or less acid reaction.
purpose lime water should be used, as being the best material
ast likely to do harm. If, however, the infant's digestion is not
nd we wish to prescribe a precise amount of lime water, we can do
iting for whatever percentage we choose, as we do for the other
of the milk. In modifying the milk which comes from the farm
l with this Laboratory, as a rule, one-twentieth part of lime water
cent.) is sufficient to make the reaction correspond to that of nor-
an milk. By referring to this table (Table 60) you will see what
ntage of lime water should be in order to obtain a greater or less
alkalinity. The hydrate of lime is said to be soluble to the ex-
part in 778 parts of water at a temperature of 15.5° C. (60° F.).
uld make one ounce of lime water to contain rather more than
rain) of CaO_2H_2 (hydrate of lime).

I shall write for seven feedings, and make the amount at each feeding 135 c.c. (4½ ounces).

I showed you at a previous lecture (Lecture VIII., p. 221) that the milk from the farm connected with the Laboratory has proved to be comparatively free from bacteria, and that it would probably be unnecessary to destroy the few bacteria which exist in it if the infant could be immediately fed here in the Laboratory. As this is not possible, and as the milk has to be transported from the Laboratory to the homes of the consumers at various distances, I have found it better to heat the milk to 75° C. (167° F.). This temperature, as I have already explained to you, is sufficient to kill those developed bacteria which would be of any harm to the digestion of the infant, and at the same time is below 77.2° C. (171° F.), the point at which coagulation of the proteids is supposed to take place. We thus obtain a practically pure fresh milk, uncooked and sterile. We therefore write in our prescription 75° C. (167° F.). If, however, the milk is to be sent a long distance, if the weather is hot, or if the milk-supply has to last more than twenty-four hours, a higher degree of heating can be used, according to the wish of the prescriber. Thus, 100° C. (212° F.) is a temperature used for these purposes at the Laboratory. Where, again, we wish the milk to be absolutely sterilized, as may be the case when we are preparing it for an ocean voyage or for a trip across the continent, not only a high degree of heat, 100° C. (212° F.), but two or three heatings, with intervals of some hours, are necessary for this complete sterilization, and this can be called for in our prescription. The length of time during which the milk should be heated, as a rule, can be left to the judgment of the superintendent. I have already shown you in this table (Table 55) that ten minutes is often sufficient to kill the developed bacteria and to make this especial milk practically sterile. Experience, however, has proved that during transportation the milk is often exposed to temperatures conducive to the further development of bacteria, and that practically the bacteriological results which we obtain in the Laboratory do not entirely hold when the milk is exposed to these varied conditions of transit. As a rule, therefore, from twenty to thirty minutes is the proper time to heat mixtures of modified milk sent from the Laboratory.

I shall also, for record in the Laboratory and for reference later, state on the prescription the infant's age and weight.

Finally I shall date the prescription, write on it the address where the food is to be delivered, and sign it.

This prescription is now handed to the clerk in the office. The clerk copies it into this book, which records each day's feeding of each individual infant, and then translates the physician's prescription into such form as can be readily understood by the modifying clerks. Of course this form may vary in different parts of the world, according as the metric or the apothecary system is in use. In the work of this especial Laboratory, although the prescriptions are written by the physicians in the metric system, it has ound more convenient, when delivered to the patrons of the Labora- o have the amounts expressed in ounces and drachms. The office after translating the metric percentages into ounces and drachms, it on to a blank of this form, which is called the modifying clerk's ption:

MODIFYING CLERK'S PRESCRIPTION.

f Infant
Infant . . . . 4 mos. wks. days.
of Infant . . . . 14 lbs. oz.

..............at..............o'clock.

ysician's *Prescription.*		*Clerk's Formula.*	
	Per Cent.		Oz. Dr.
. . . .	4 : 00	Modifying Cream	7 : 7
ugar	7 : 00	Modifying Milk	4 : 7
s	1 : 50	Sugar Solution	8 : 3
l Matter		Lime Water	1 : 5
Vater		Water	8 : 6
		Total	31 : 4

Feedings 7
t at each Feeding . . . . 4½ ounces.
l at . . . . 167° F.
n Sterilizer . . . . 20 minutes.

ks.

Put up by
Date..............
Month..............

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31

..............................189......by..............................

his prescription is then placed in the hands of the modifying clerk, ombines the different elements of the prescription by means of the ntal materials which have been brought into the modifying-room from rent part of the Laboratory, and which I have already described.

have requested physicians to write their prescriptions within certain as to the percentages of the fat, sugar, and proteids, and to allow the al matter for the present to regulate itself. The limits which up to resent time the Laboratory has found it necessary to place on the pre- ions for the milk-modifiers, and within which the modifying clerk pposed to put up the prescriptions, are as is shown in this table e 63):

TABLE 63.

Fat	from 0.03 to 36.00
Sugar	from 0.87 to 20.00
Proteids	from 0.22 to 4.00

There is not much doubt that in the future more and more exact results will be obtained, representing definite percentages of still wider limits. The results obtained from combining the modifying materials used by the modifying clerks have so often been proved to be practically correct, that we can assume that when we write a prescription we shall obtain in return a product which in its various elements comes within a fraction of one per cent.

I have arranged in this table (Table 64) figures which will aid you in writing for such percentages of the fat, sugar, and proteids as can be obtained at the Laboratory:

TABLE 64.

Practical Limits of Milk-Modification which can be accomplished in the Laboratory.

I.

Low Fats.

Fat	0.03	0.04	0.08	0.12–16
Sugar	2.00	3.00	4–5.00	6.00–7.00
Proteids	0.75	1.00	2.00	3.00–4.00

II.

Low Sugars.

Sugar	0.87	1.40	2.12	3.50–4.30
Fat	2 00	3.00	3.50	4.00
Proteids	0.75	1.00	2.00	3.00–4.00

III.

Low Proteids.

Proteids	0.22	0.34	0.45	0.53
Fat	2.00	3.00	4.00	4.50
Sugar	2.00	3.00	4 00–5.00	6.00–7.00

FIG. 60.

You see that in I. I have taken the lowest percentage of fat which can practically be used at the Laboratory and have combined it with various percentages of sugar and of proteids. In II., in like manner, I have taken the lowest percentages of the sugar which can be combined with these various percentages of fat and proteids. Finally, in III. I have made the same calculations for the proteids.

Other materials can also be obtained at the Laboratory on the physician's prescription for older infants and children, notably preparations of oats, barley, and wheat, which you see this young woman (Fig. 60) preparing in a special apparatus devised for steaming these cereals.

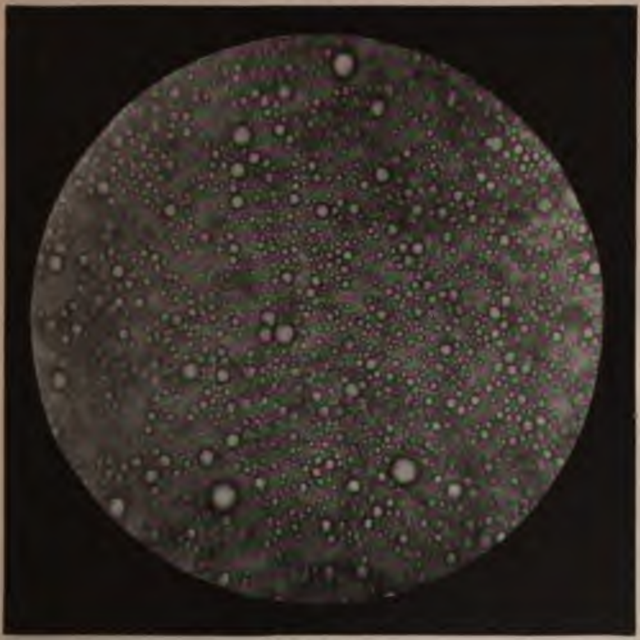
When a physician orders cereals to be prepared at the Laboratory, he is by this apparatus to obtain exact preparations as to the percentages onstituents of any cereal foods. This is accomplished by employing lysis of the special cereal ordered, and, with distilled water as a , regulating the time the heating shall be maintained with "live around the porcelain crocks.

e question having arisen whether the emulsion of milk which is used dification is interfered with or destroyed by modification, I have ed it in the following way :

der each of these four microscopes there is a drop of milk strongly fied.

nder the first microscope (Fig. 61) you will see a drop of milk as it from the cow, unmodified and unchanged, except as it might have been d by transportation from the farm.

FIG. 61.



Cow's milk.

he analysis (Analysis 48) of the milk from which this drop was taken ollows :

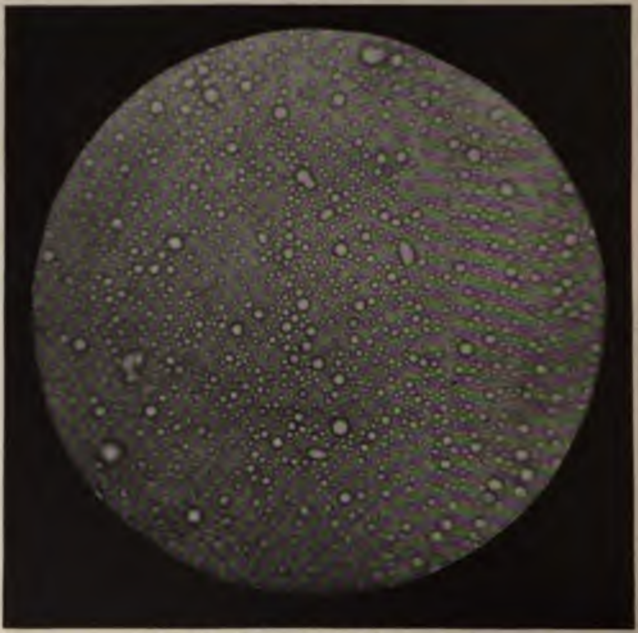
ANALYSIS 48.

Cow's Milk.

at	4.04
ugar	4.55
roteids	4.15
sh	0.71
otal solids	13.45
ater	86.55
	100.00

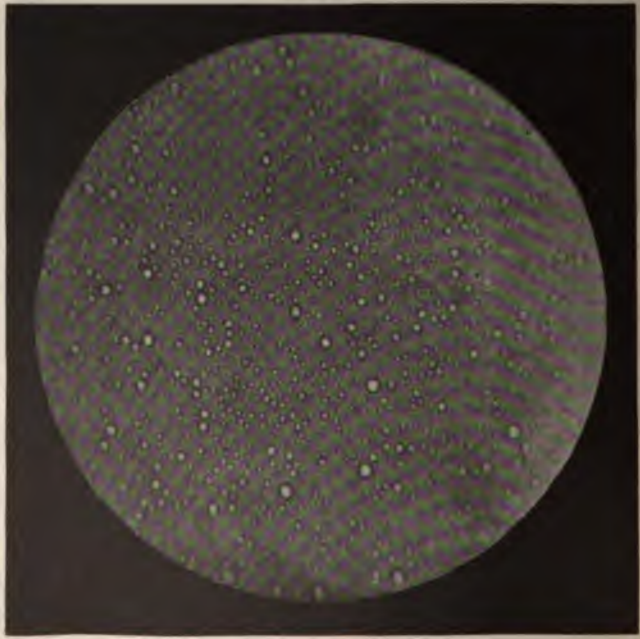
Under this second microscope (Fig. 62) is a drop of a mixture whic has been so treated as to represent the same analysis (Analysis 48) as tha

FIG. 62.



Cow's milk separated and recomposed.

FIG. 63.



Human milk.

original whole milk, and which in fact is the original whole milk as Fig. 61, which has been separated and recomposed.

u will notice that the emulsion of the recomposed milk is quite as that of the original whole milk from which it was separated.

der this third microscope (Fig. 63) I have placed a specimen of milk.

analysis (Analysis 49) of this milk was sent to me in order that I have a food modified to correspond to it, to be used for the purpose ixed feeding:

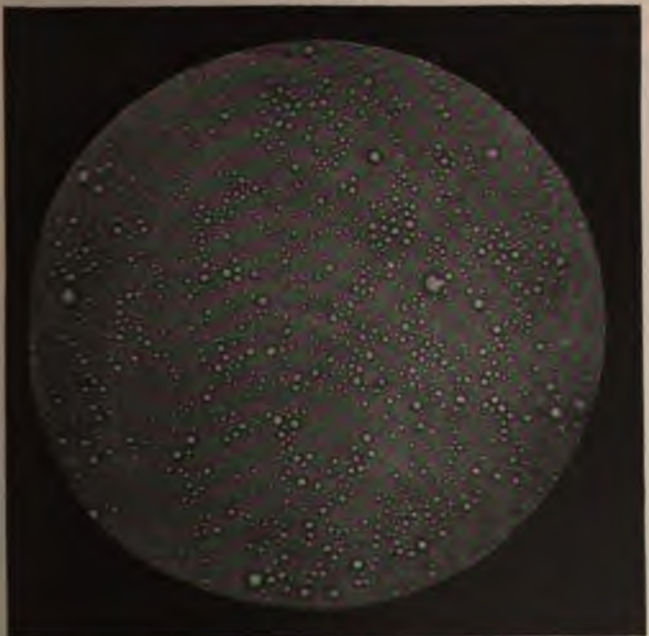
ANALYSIS 49.

Human Milk.

at	2.67
ugar	6.37
roteids	2.69
sh	0.15
otal solids	11.88
Vater	88.12
	100.00

der this fourth microscope (Fig. 64) is a specimen of cow's milk modi- correspond to the human milk which is under the third microscope 63).

FIG. 64.

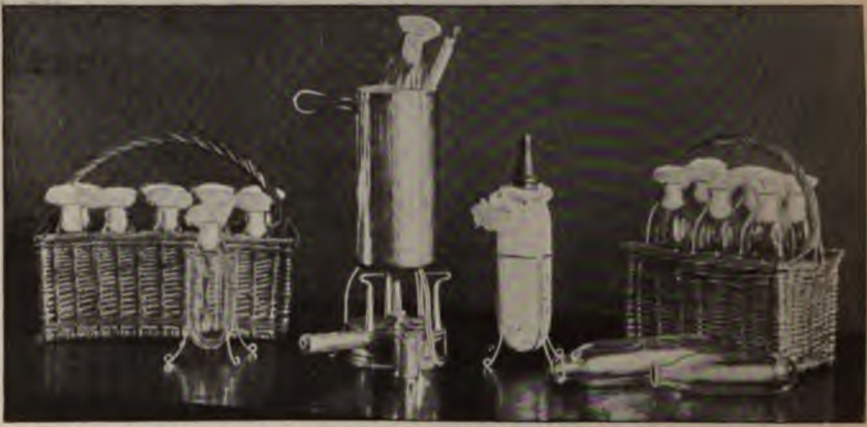


Modified milk.

ou see that the emulsion corresponds almost exactly: so that there is estion that it is not injurious, so far as the emulsion is concerned, to ate the elements of milk and then recombine them.

I shall now take you back to the office and show you the various forms of apparatus which are provided for feeding the infant in its home. I have had some of them placed on this table (Fig. 65).

FIG. 65.



In the left of the picture is a basket holding eight tubes of a capacity of six ounces each. In front of this basket is a four-ounce tube in a wire stand. In the middle of the picture is a tin apparatus for warming the milk at the time of feeding. An alcohol lamp is shown beneath the warmer, and a tube of milk and a thermometer for testing the temperature of the milk are in the tin-warmer. Next to and to the right of the tin-warmer is a tube with a capacity of eight ounces. It is enclosed in a white worsted cozy, has the rubber nipple in place, and is supported in a wire stand. In the right of the picture is a basket containing six tubes with a capacity of eight ounces each. In front of this basket are an eight-ounce tube and a four-ounce tube.

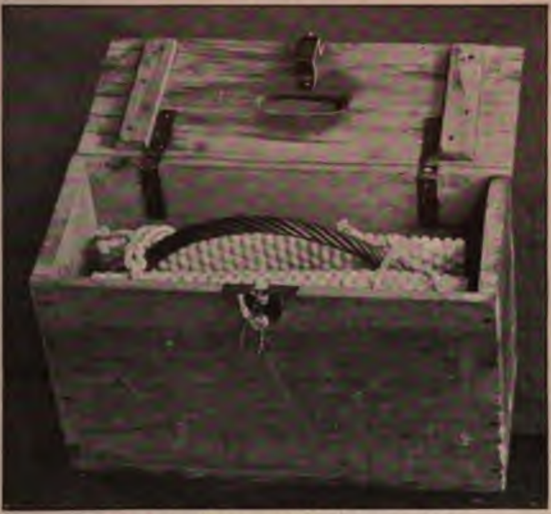
This apparatus is very simple and practical for transportation. A wicker basket, divided into a number of compartments corresponding to the number of feedings which are to be sent to the infant, has been found to be the most practical. These baskets with their tubes can be placed, as you saw, directly in the sterilizer, and are not harmed by the heat to which it is necessary to expose the food.

This tin receptacle can be placed above an alcohol lamp; the water in it is to be on a level with the height of the milk which is contained in the tube, and the tube is submerged in the water. It has been found necessary to take the temperature of the food by means of a thermometer placed directly in the tube. No rule can be laid down by which the temperature of the water-bath determines that of the milk, unless the tubes are of uniform thickness and the milk uniform in quantity and temperature when placed in the bath. The thermometer must be washed in sterilized water with the greatest care, both before and after it is used. The food when given to the infant should have a temperature of from 36.6° to 37.7° C. (98°–100° F.).

As in direct feeding from the breast the food which the infant receives has the same temperature at the end of the feeding as at the beginning, we should copy this provision of nature and not allow the temperature of the food to vary during the time it is being taken. To accomplish this end, this white worsted cozy can be used. The cozy is warmed at the same time that the milk is being heated, and the tube when placed in it is prevented from cooling.

us the infant receives a food of unvarying temperature throughout the ole of the feeding.

Fig. 66.

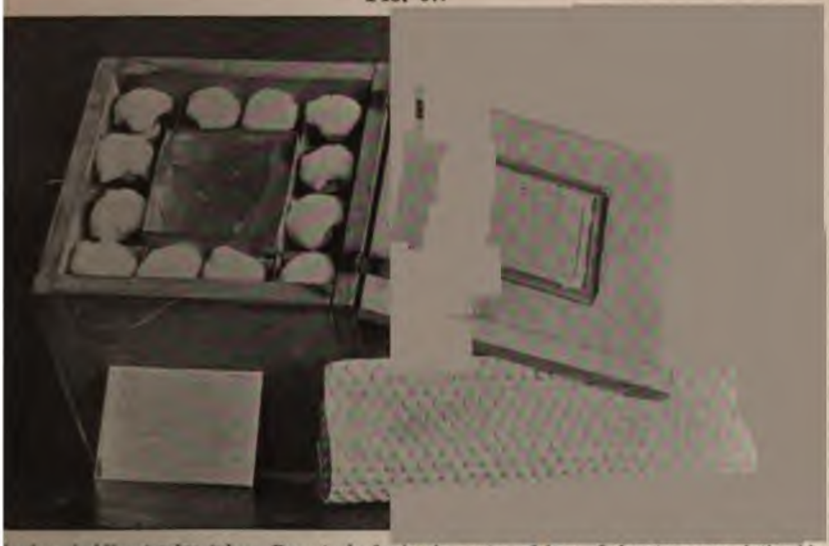


Transportation-box, containing basket and tubes.

I have here also to w to you the various ans which are used in nsporting the food en it has to be sent g distances. Here is ransportation-box (Fig. , which is used in cold ther, when ice is not essary to preserve the hness of the milk.

This box (Fig. 67) is which can be used hot weather, and has ved to be of great prac- l utility. It admi- ly serves the purposes an express box and of a home refrigerator. The ice, as you see, is ked in a metal compartment in the middle of the box, and the tubes are ced, each in its own compartment, around the sides of the ice-receptacle.

Fig. 67.



ice-box, holding twelve tubes. Receptacle for ice in centre of box. Laboratory prescription-blank ont of box, and packing-paper under end of open lid.

I shall now call your attention to two cases which were fed under my ection at the Milk-Laboratory during the first year of their lives, and ich merely illustrate the changes which naturally would be made during period in the food of a healthy infant.

The first case (Case 91) was a male, born November 18, 189 table (Table 65) shows the record of its weight and food during year:

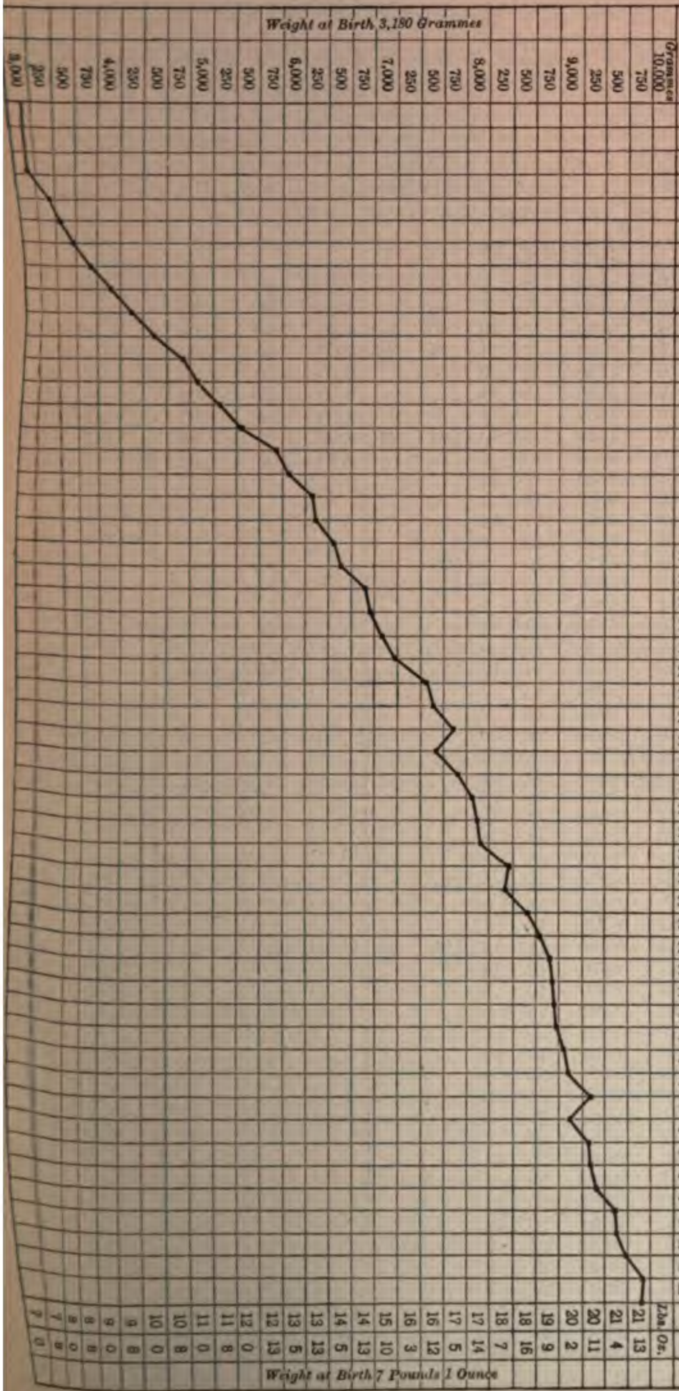
TABLE 65.

Showing Management of the Food and Increase in Weight of a Healthy Infant during the First Fifty-Two Weeks of its Life.

DATE.	Weeks of Life.	WEIGHT.			Amount at each Feeding.		PERCENTAGES OF FOO			
		Grams.	Lbs.	Oz.	C.c.	Oz.	Fat.	Sugar.	Proteids.	Lime
November 18 . .	1	3752	8	6	30	1	2.00	6.00	1.00	5.
"	2									
"	3	. . .	. .	.	. .	.	3.00	6.00	1.00	
"	4	. . .	. .	.	45	1½				
"	5	. . .	. .	.	75	2½	4.00	7.00	1.00	10.
December 23 . .	6	4284	9	9	90	3				
"	7									
"	8	. . .	. .	.	. .	. .	. .	. . .	. . .	5.
January 13 . . .	9	6944	15	8	105	3½				
"	10									
"	11									
"	12									
"	13	. . .	. .	.	120	4				
February 17 . .	14	6048	13	7	135	4½	4.00	7.00	2.00	
"	15									
"	16									
"	17									
March 17 . . .	18	6748	15	1	150	5				
"	19									
"	20									
"	21									
"	22									
April 21	23	7308	16	5	165	5½				
"	24									
"	25									
"	26									
May 18	27	7504	16	12	180	6	4.00	7.00	2.50	
"	28									
"	29									
"	30									
"	31									
June 22	32	7840	17	8						
"	33	. . .	. .	.	210	7				
"	34	. . .	. .	.	. .	. .	4.00	7.00	3.00	12.
"	35									
"	36									
"	37	. . .	. .	.	. .	. .	4.00	7.00	2.50	
"	38	. . .	. .	.	. .	. .	3.50	6.50	1.50	10
"	39	. . .	. .	.	180	6	4.00	7.00	2.00	5.
August 17 . . .	40	8820	19	11	225	7½				
"	41	. . .	. .	.	. .	. .	. .	. . .	. . .	10.
"	42	. . .	. .	.	. .	. .	. .	. . .	. . .	12.
"	43	. . .	. .	.	. .	. .	4.00	6.00	2.50	10.
"	44	. . .	. .	.	. .	. .	4.00	5.00	3.00	5.
"	45									
"	46									
"	47	. . .	. .	.	. .	. .	Whole milk.			
"	48									
"	49	. . .	. .	.	. .	. .	Whole milk and			oat je
"	50									
"	51									
November 9 . .	52	9870	22							

The grammes in the third column have been reduced to pounds and ounces on *of 28 grammes* to the ounce, and the fractions of the ounce have been disregarded

The next case (Case 92) was a female, born November 1, 1892. This hart (Chart 4) shows the line of growth in its weight from birth to the ifty-second week of its life :



Weeks	Gram's	Lbs.	Oz.	Date of Weighing	
	Date of Birth Nov. 1st.				
	Actual Weight				
1	3,180	7	1	Nov.	1
2	3.180	7	1	"	8
3	3.180	7	1	"	15
4	3,430	7	10	"	22
5	3,520	7	14	"	29
6	3,730	8	5	Dec.	6
7	3,980	8	14	"	13
8	4,160	9	4	"	20
9	4,340	9	10	"	27
10	4,590	10	4	Jan.	3
11	4,870	10	14	"	10
12	5,060	11	4	"	17
13	5,270	11	12	"	24
14	5,560	12	6	"	31
15	5,870	13	1	Feb.	7
16	6,070	13	8	"	14
17	6,300	14	1	"	21
18	6,370	14	4	"	28
19	6,510	14	8	Mar.	7
20	6,650	14	13	"	14
21	6,920	15	7	"	21
22	6.980	15	9	"	28
23	7.150	15	15	Apr.	4
24	7,240	16	2	"	11
25	7,560	16	14	"	18
26	7,600	16	15	"	25
27	7,800	17	6	May	2
28	7,780	17	4	"	9
29	7,840	17	8	"	16
30	8,070	18	0	"	23
31	8,160	18	3	"	30
32	8,190	18	4	June	6
33	8,490	18	15	"	13
34	8,470	18	14	"	20
35	8,700	19	6	"	27
36	8,762	19	8	July	4
37	8,824	19	11	"	11
38	8,950	19	14	"	18
39	8,970	20	0	"	25
40	8,980	20	1	Aug.	1
41	9,060	20	4	"	8
42	9.140	20	6	"	15
43	9,340	20	13	"	22
44	9.170	20	8	"	29
45	9.290	20	12	Sep.	5
46	9,340	20	13	"	12
47	9,470	21	2	"	19
48	9.640	21	9	"	26
49	9,630	21	8	Oct.	3
50	9,740	21	10	"	10
51	9,870	22	0	"	17
52	9,890	22	1	"	24
	13 Teeth at 12 Months				

CHART 4.

I have also arranged a table (Table 66) recording the quantity and quality of this infant's (Case 92) food during the first year:

TABLE 66.

Showing Management of the Food and Increase in Weight of a Healthy Infant (Case 92) during the First Fifty-Two Weeks of its Life.

DATE.	Weeks of Life.	WEIGHT.			Amount at each Feeding.		PERCENTAGES OF FOOD.			
							Fat.	Sugar.	Proteids.	Lime Water.
		Grams.	Lbs.	Oz.	C.c.	Oz.				
November 1 . .	1	3180	7	1						
November 8 . .	2	3180	7	1	60	2	2.00	5.00	1.00	5.00
November 15 . .	3	3180	7	1	60	2	4.00	7.00	1.00	
November 22 . .	4	3430	7	10	90	3	4.00	7.00	1.00	
November 29 . .	5	3520	7	14	75	2½	3.00	7.00	1.00	
December 6 . .	6	3730	8	5	. .	. .	3.00	6.00	1.00	
December 13 . .	7	3980	8	14	. .	. .	3.00	7.00	1.00	
December 20 . .	8	4160	9	4	90	3	4.00	7.00	1.00	10.00
December 27 . .	9	4340	9	10	105	3½	4.00	7.00	1.25	5.00
January 3 . . .	10	4590	10	4	. .	. .	4.00	7.00	1.50	
January 10 . . .	11	4870	10	14						
January 17 . . .	12	5060	11	4						
January 24 . . .	13	5270	11	12						
January 31 . . .	14	5560	12	6						
February 7 . . .	15	5870	13	1	120	4	. .	. . .	. . .	12.50
February 14 . .	16	6070	13	8	135	4½	. .	. . .	. . .	10.00
February 21 . .	17	6300	14	1	. .	. .	. .	. . .	. . .	5.00
February 28 . .	18	6370	14	4						
March 7	19	6510	14	8						
March 14 . . .	20	6650	14	13	150	5	4.00	7.00	2.00	
March 21 . . .	21	6920	15	7						
March 28 . . .	22	6980	15	9						
April 4	23	7150	15	15						
April 11	24	7240	16	2						
April 18	25	7560	16	14						
April 25	26	7600	16	15						
May 2	27	7800	17	6						
May 9	28	7730	17	4						
May 16	29	7840	17	8						
May 23	30	8070	18	0	180	6				
May 30	31	8160	18	8						
June 6	32	8190	18	4						
June 13	33	8490	18	15	195	6½	4.00	7.00	2.50	
June 20	34	8470	18	14						
June 27	35	8700	19	6						
July 4	36	8762	19	8						
July 11	37	8824	19	11						
July 18	38	8950	19	14						
July 25	39	8970	20	0						
August 1	40	8980	20	0						
August 8	41	9060	20	3						
August 15 . . .	42	9140	20	6						
August 22 . . .	43	9340	20	13						
August 29 . . .	44	9170	20	7						
September 5 . .	45	9290	20	11						
September 12 . .	46	9340	20	13						
September 19 . .	47	9470	21	2						
September 26 . .	48	9640	21	9						
October 3 . . .	49	9630	21	7						
October 10 . . .	50	9740	21	10	. .	. .	4.00	6.00	3.00	
October 17 . . .	51	9870	22	0	. .	.	Whole milk.			
October 24 . . .	52	9890	22	1	. .	. .	Whole milk and oat jelly			

The grammes in the third column have been reduced to pounds and ounces on the *of 28 grammes to the ounce*, and the fractions of the ounce have been disregarded.

I shall also mention a few cases which have a practical bearing on the ethod of substitute feeding by means of milk-laboratories.

The first case illustrates how important it is to be able to vary the perentages of the different elements of the milk, and to know that we are btaining these variations exactly as they are ordered.

An infant (Case 93) was being nursed by its mother, who was healthy and who had an bundance of breast-milk. Their summer home was by the sea-side, in a healthy situation, nd the infant was surrounded with everything that could be desired for perfect hygiene. The infant during the first two months of its life nursed well, throve, and was perfectly quiescent in its daily life. When it was three months old, the mother was very much worried by some trivial family matters and did not take much exercise. The infant now began to have colic, and, although it gained in weight, it was very restless and cried continuously. An analysis (Analysis 50) of the mother's milk at this time gave the following result:

ANALYSIS 50.

Fat	2.69
Sugar	6.15
Proteids	3.71
Ash	0.17
Total solids	12.72
Water	87.28
	100.00

The indications for treatment were, of course, to lessen the amount of mental disturbnce in the mother and to make her exercise more. The mother having followed these rections, the symptoms in the infant soon became less severe. After a few days, hower, the unfavorable symptoms returned, and it was found that the mother had not been ercising and was again mentally disturbed. As it seemed impossible to regulate the funcn of the mammary gland under these circumstances, it was decided to feed the infant m the Milk-Laboratory. The following prescription (Prescription 5) was ordered:

PRESCRIPTION 5.

℞ Fat	3.50
Sugar	6.50
Proteids	1.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).
Eight tubes, each holding	90 c.c. (3 ounces).

The infant digested this food perfectly, had no colic, and again became tranquil. As, wever, it only made a slight gain in weight during the first two or three weeks of this bstitute feeding, I changed the prescription to the following one (Prescription 6):

PRESCRIPTION 6.

℞ Fat	4.00
Sugar	7.00
Proteids	1.50

On taking this food the infant began to make regular gains in weight, and continued to thrive until it was four months old, when it was brought back to its city home, where it was subjected to many of the annoyances which you will so frequently see occurring in the families which you take care of, and which, though somewhat disastrous to the infant, te to advance our knowledge of substitute feeding. The annoyances which I refer to were, this especial case, as follows. The infant was surrounded with too much excitement, i was exposed to unnecessary changes of temperature in its home. During the process removal from the sea-side to the city it caught a slight cold, and had intestinal sympt characterized by loose discharges from the bowels and undigested food. This condition easily obviated in a few days by simply changing the prescription at the Laboratory to following one (Prescription 7):

PRESCRIPTION 7.

℞	Fat	2.50
	Sugar	5.50
	Proteids	1.00
	Lime water	10.00

Under this treatment the food was again fairly well digested, the discharges lesse in frequency and they were of a better character. The infant, however, during this s ness had lost over 224 grammes (about ½ pound) in weight.

At this juncture the grandmother of the infant so influenced the mother that insisted upon having a wet-nurse procured at once. Although I did not approve of change, the family were so urgent in their demands for a wet-nurse that I procured one them. This wet-nurse was nursing her own infant and another infant at the Infa Hospital. Both infants were thriving in every way. An analysis (Analysis 51) of wet-nurse's milk gave the following results:

ANALYSIS 51.

Fat	2.92
Sugar	6.20
Proteids	4 62
Ash	0.16
Total solids	13.90
Water	86.10
	100.00

The milk for this analysis was taken from the middle of the nursing. The percent of proteids was so high that I did not dare to allow the foster-infant to be put to the br at once. I therefore endeavored to regulate the percentages of the elements of the nurse's milk in the usual way. At the end of two days another analysis (Analysis 52 her milk was made, with the following result:

ANALYSIS 52.

Fat	3.39
Sugar	5.95
Proteids	4.78
Ash	0.21
Total solids	14.33
Water	85.67
	100.00

The extraordinarily high percentage of proteids in this analysis made me absolu refuse to allow the foster-infant to begin with its nursing from the wet-nurse. The fam however, were very impatient, and argued that, as the other two infants were gainin weight, digesting well, and looking remarkably ruddy, it must be a good milk which t were receiving from the wet-nurse.

Two days later, although the foster-infant was decidedly improving on the substitute food from the Laboratory, it happened to lose 30 grammes (about 1 ounce) in weight, and the family then insisted that this wet-nurse should be tried. Another analysis of the wet-nurse's milk was then made, and showed that the percentage of the proteids had been reduced to between 3 and 4.

I had already endeavored to find other wet-nurses whose milk would better correspond to what the infant needed, but was unsuccessful in obtaining any the analysis of whose milk showed the percentage of the proteids to be below 3.

I have here the analyses (Analyses 53 and 54) of the milk of two of these wet-nurses, which you may perhaps like to see:

ANALYSIS 53.

Fat	3.88
Sugar	6.55
Proteids	3.14
Ash	0.14
Total solids	13.71
Water	86.29
	100.00

ANALYSIS 54.

Fat	3.39
Sugar	4.50
Proteids	4.70
Ash	0.18
Total solids	12.77
Water	87.23
	100.00

The first wet-nurse was then brought to the foster-infant's home, and the infant was put to the breast. It absolutely refused to take the breast for twelve hours, although it was crying with hunger. Finally it was induced to nurse, but immediately after the nursing had an attack of colic. These attacks of colic were more or less severe and occurred after each nursing. The infant soon appeared to like the milk and took it eagerly at the regular nursing intervals. In twenty-four hours from the time when the infant began to nurse its bowels were again affected. The number of discharges became frequent, and the milk evidently was not being digested well. These conditions lasted for several days, when it was found that the infant had lost over 480 grammes (about 1 pound) in weight. As the severity of the colic was increasing, and as the infant had lost its color, the mother agreed to have the feeding by the wet-nurse discontinued. I then wrote the following prescription (Prescription 8) to be put up at the Laboratory:

PRESCRIPTION 8.

℞ Fat	2.00
Sugar	5.00
Proteids	1.00
Lime water	10.00
To be heated to	75° C. (167° F.)

This mixture was given to the infant. In twenty-four hours the number of discharges from the bowels grew less, and in a few days became almost normal. It began to gain in weight, and, though seeming very hungry, looked better and ceased to have colic.

The prescription was then changed to the following one (Prescription 9):

PRESCRIPTION 9.

℞ Fat	8.00
Sugar	6.00
Proteids	1.00
Lime water	5.00

On taking this food the infant began to make regular gains in weight, but still seemed hungry, so that at the end of another week the prescription was changed to the following one (Prescription 10):

PRESCRIPTION 10,

℞ Fat	4.00
Sugar	7.00
Proteids	1.50

The infant now improved steadily. It made the normal average daily gains in weight and soon recovered its color and former strength. From this time it continued to thrive.

This case is interesting in many ways. It was very evident that a percentage of proteids over 3 was more than this especial infant could digest. It therefore had to be weaned from its mother. The wet-nurse's milk, which was agreeing perfectly with her own infant and with another infant which she was nursing at the hospital, had a percentage of proteids between 3 and 4. As I knew from my experience with the mother's milk that this high percentage of proteids would not agree with the infant, I was not surprised to find that, instead of agreeing with it, it made it sick. This case substantiates the statement which I have made in an earlier lecture (Lecture VII., page 180), that, while there are many varieties of good milk, there are also many infants who cannot thrive on them all, but only upon such as suit their individual digestive powers.

It is interesting also to record in this case that, as the infant grew older, it was found that the percentage of the proteids could be increased in its food without harming its digestion, and that by the time it was eight months old it was having in its food percentages of proteids between 3 and 4, the very percentages which caused such serious digestive disturbance when it was younger. When it was ten months old it was able to digest 4 per cent of proteids in its food.

This case as a whole so well illustrates the use of the Milk-Laboratory that it is hardly worth while to multiply instances of its value. I will, however, give the record of the treatment of some twins (Cases 94 and 95) that have recently come under my charge, showing the utility of feeding by means of modified milk.

These infants were born at term, but were as weak and emaciated as though they had been premature. One of them had a convulsion when it was a few hours old, and the other's circulation was very deficient and showed evidence of a cardiac souffle over the base of the sternum for some days. This class of infants is very apt to die unless their food is carefully regulated at once, and the great lack of equilibrium of the percentages of the elements of the maternal milk in the early days of life is often most disastrous in its effects on the hypersensitive condition of the gastro-enteric tract at this age.

Here is a table (Table 67) showing the condition and the treatment of these infants in the first fifty days of their lives:

CASE 94.

Showing the Details of the

REMARKS.	PERCENTAGES OF FOODS.				AMOUNT AT EACH FEEDING.		INTERVALS OF FEEDING.	WEIGH	
	Fat.	Sugar.	Proteids.	Lime Water.	C.c.	Dr'ms.		Gram's.	Lbs.
Very weak. Emaciated.	. .	. . .	. .	. .	. .	. .	. . . . .	1816	4
Brandy 3 drops every 3 hours.	Equal parts cow's milk and lime water.				4	1	2 hours.	. . .	. .
Cardiac murmur at base of sternum.	. .	. . .	. . .	. . .	. .	. . .	“	. . .	. .
	Breast-milk.			. . .	. .	. . .	“	. . .	. .
Colic. Loose movements. Vomiting.	. .	. . .	. . .	. . .	16	4	“	. . .	. .
	. .	. . .	. . .	. . .	. .	. . .	“	. . .	. .
	. .	. . .	. . .	. . .	. .	. . .	“	. . .	. .
Icterus neonatorum from 8th day to 35th day. Cord fell on 8th day.	Breast omitted.			. . .	. .	. .	“	. . .	. .
	1.50	5.00	0.75	10.00	20	5	“	. . .	. .
Brandy 6 drops every 6 hours.	. .	. . .	. . .	. . .	. .	. . .	“	2056	4
Very feeble. Respirations irregular.	. .	. . .	. . .	. . .	. .	. . .	“	. . .	. .
	. .	. . .	. . .	. . .	. .	. . .	“	. . .	. .
	. .	. . .	. . .	. . .	. .	. . .	“	. . .	. .
	. .	. . .	. . .	. . .	. .	. . .	“	2176	4
	. .	. . .	. . .	. . .	. .	. . .	“	2176	4
	. .	. . .	. . .	. . .	. .	. . .	“	2270	5
	. .	. . .	. . .	. . .	. .	. . .	“	2510	5
Vomiting.	. .	. . .	. . .	. . .	. .	. . .	“	. . .	. .
	. .	. . .	. . .	. . .	. .	. . .	“	. . .	. .
	1.25	6.00	0.75	10.00	. .	. . .	“	2630	5
	. .	. . .	. . .	. . .	. .	. . .	“	2570	5
Much flatus. Six loose green dejections.	. .	. . .	. . .	. . .	. .	. . .	“	2540	5
	. .	. . .	. . .	. . .	. .	. . .	“	2495	5
Cries a great deal. Very hungry. Brandy 3 drops every 3 hours.	. .	. . .	. . .	. . .	32	8	“	2510	5
	. .	. . .	. . .	. . .	. .	. . .	“	2510	5
	. .	. . .	. . .	. . .	. .	. . .	“	2540	5
	. .	. . .	. . .	. . .	. .	. . .	“	2525	5
	. .	. . .	. . .	. . .	. .	. . .	“	2540	5
	. .	. . .	. . .	. . .	. .	. . .	“	2555	5
	. .	. . .	. . .	. . .	. .	. . .	“	2525	5
	1.50	6.00	1.00	10.00	. .	. . .	“	2540	5
	. .	. . .	. . .	. . .	. .	. . .	“	2540	5
Less vomiting.	. .	. . .	. . .	. . .	. .	. . .	“	2495	5
	2.00	6.00	1.00	5.00	. .	. .	“	2555	5
Fæcal discharges fewer in number and look better.	. .	. . .	. . .	. . .	48	12	“	2600	5
	. .		. . .	. . .	. .	. . .	“	2680	5
	. .	. . .	. . .	. . .	. .	. . .	“	2660	5
	. .	. . .	. . .	. . .	. .	. . .	“	2660	5
	. .	. . .	. . .	. . .	56	14	“	2690	5
	. .	. . .	. . .	. . .	. .	. . .	“	2705	5
	. .	. . .	. . .	. . .	64	16	“	2720	5
	. .	. . .	. . .	. . .	. .	. . .	“	2739	6½
	. .	. . .	. . .	. . .	. .	. . .	“	2769	6
	2.50	6.50	1 00	5.00	. .	. .	“	2724	6
	. .	. . .	. . .	. . .	. .	. . .	“	2754	6
	. .	. . .	. . .	. . .	. .	. . .	“	2784	6
10 feedings in 24 hours.	. .	. . .	. . .	. . .	. .	. . .	“	2799	6
	. .	. . .	. . .	. . .	. .	. . .	“	2814	6
	. .	. . .	. . .	. . .	. .	. . .	“	2829	6
	. .	. . .	. . .	. . .	. .	. . .	“	2889	6
Fæcal discharges well digested and of good color. Thriving.	3.00	6.50	1 00	5.00	64	16	“	2904	6

CASE 95.

of Cases 94 and 95 (Twins).

EIGHT.		INTERVALS OF FEEDING.	AMOUNT AT EACH FEEDING.		PERCENTAGES OF FOODS.				REMARKS.
					Fat.	Sugar.	Proteids.	Lime Water.	
Lbs.	Oz.		C.c.	Dr'ms.					
3	14	....	...	...	...	...	...	...	Hemorrhage from cord. Convulsions. Very weak. Emaciated.
...	...	2 hours.	4	1	Equal parts cow's milk and lime water.				Brandy 3 drops every 3 hours.
...	...	"	...	...	Breast-milk.			...	No more convulsions.
...	...	"	...	...	Breast-milk.			...	Colic. Loose fæcal dejections.
...	...	"	16	4	Breast-milk.			...	Cord fell on 4th day.
...	...	"	...	...	Breast-milk.			...	
...	...	"	...	...	Breast omitted.			...	Icterus neonatorum from 8th day to 30th day Less colic.
...	...	"	20	5	1.50	5.00	0 75	10.00	
...	...	"	...	...	...	...	...	...	Brandy 6 drops every 6 hours. Very feeble. Respirations irregular. Fewer fæcal dejections.
4	8	"	...	...	...	...	...	...	
...	...	"	...	...	...	...	...	...	
...	...	"	...	...	...	...	...	...	
...	...	"	...	...	...	...	...	...	
4	8	"	...	...	...	...	...	...	
4	8	"	...	...	...	...	...	...	
4	12	"	...	...	...	...	...	...	
5	4	"	...	...	...	...	...	...	Vomiting.
...	...	"	...	...	...	...	...	...	
...	...	"	...	...	1.25	6.00	0.75	10.00	Less vomiting.
5	7	"	...	...	...	...	...	...	
5	8	"	...	...	...	...	...	...	
5	7	"	32	8	...	...	...	...	Very weak. Icterus pronounced. Vomiting occasionally. Four or five fæcal discharges daily and not well digested.
5	7½	"	...	...	...	...	...	...	
5	7	"	...	...	...	...	...	...	
5	8	"	...	...	...	...	...	...	
5	8	"	...	...	...	...	...	...	
5	7½	"	...	...	...	...	...	...	
5	8	"	...	...	...	...	...	...	
5	8½	"	...	...	1.50	6.00	1.00	10.00	Fæcal discharges fewer and better digested.
5	8	"	...	...	...	...	...	...	
5	9	"	...	...	...	...	...	...	
5	8	"	...	...	2.00	6.00	1 00	5.00	Very hungry.
5	7½	"	48	12	...	...	...	...	
5	9	"	...	...	...	...	...	...	
5	10	"	...	...	...	...	...	...	
5	11	"	...	...	...	...	...	...	
5	11½	"	56	14	...	...	...	...	
5	12½	"	...	...	...	...	...	...	
5	13	"	64	16	...	...	...	...	
5	13½	"	...	...	...	...	...	...	No icterus. Much stronger. No vomiting. Fæcal discharges fewer and look better.
6	0	"	...	...	...	...	...	...	
6½	0	"	...	...	...	...	...	...	
6	0	"	...	...	2.50	6.50	1.00	5.00	
6	1½	"	...	...	...	...	...	...	
6	2	"	...	...	...	...	...	...	10 feedings in 24 hours.
6	4	"	...	...	...	...	...	...	
6	5	"	...	...	...	...	...	...	
6	6	"	...	...	...	...	...	...	
6	5	"	...	...	...	...	...	...	
6	6½	"	...	...	...	...	...	...	
6	7½	"	72	18	3.00	6.50	1.25	5.00	Fæcal dejections well digested and of good color. Thriving.

It may be instructive for you to look over a few of these prescriptions which I have sent to the Laboratory at different times, as they will give you a very fair idea of the simplicity and precision of substitute feeding.

Prescription 11.

A girl 6 years old; duodenal jaundice (functional).

℞ Fat	0.50
Milk-sugar	6.00
Proteids	3.00
Lime water	10.00

Send 12 tubes, each 4 ounces.

Prescription 12.

A boy 6 weeks old; healthy.

℞ Fat	3.00
Milk-sugar	7.00
Proteids	1.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

12 tubes, each 2 ounces.

Prescription 13.

A boy 6 months old; healthy.

℞ Fat	4.00
Sugar	7.00
Proteids	2.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

8 tubes, each 6 ounces.

Prescription 14.

A girl 4 months old; proteid digestion weak.

℞ Fat	4.00
Sugar	7.00
Proteids	0.75
Lime water	5.00
Heated to	75° C. (167° F.).

8 tubes, each 4 ounces.

Prescription 15.

A boy 6 months old; sugar digestion weak.

℞ Fat	3.00
Sugar	4.00
Proteids	2.00
Lime water	5.00
Heated to	75° C. (167° F.).

8 tubes, each 6 ounces.

Prescription 16.

A girl 4 months old; summer diarrhœa. Food has to be sent to a distant town by express.

℞ Fat	2.00
Sugar	5.00
Proteids	1 00
At time of each feeding add lime water	8 drachms.
Heated to	100° C. (212° F.).

20 tubes, each 1 ounce and 1 drachm.

In this case the diarrhœa had not been sufficiently studied to determi whether it was putrefactive or fermentative, so that a safe general prescri tion was sent to begin with. The lime water had to be introduced at e feeding on account of the 100° C. (212° F.) heating, necessitated by hot weather and the distance to be sent. If the lime water had been int duced at the Laboratory and heated to 100° C. (212° F.) with the foo reaction would have taken place between the lime and the sugar, and mixture would have turned brown and have had a peculiar taste.

FEEDING OF AVERAGE INFANTS BORN AT TERM.—When an infa born at term, is of normal development and weight, and is healthy, I in the habit of regulating the quantity of its food according to the fig which I have arranged in this table (Table 57, page 234). These figu however, are intended only to be provisional until by experiment the pro amount for the individual has been ascertained.

The quality of the food which I begin with is usually as shown in following prescriptions (Prescriptions 17, 18, 19, 20, 21, 22, 23, 24, 25, 27, 28, 29). Where these prescriptions are used the infant is supposed be digesting well and gaining in weight progressively.

PRESCRIPTION 17.

For the first twenty-four to thirty-six hours of life.

℞ Milk sugar, five-per-cent. solution, in sterilized distilled water.

PRESCRIPTION 18.

First week.

℞ Fat	2.00
Sugar	5.00
Proteids	0.75
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION 19.

Second week.

℞ Fat	2.50
Sugar	6.00
Proteids	1.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION 20.

Third week.

℞ Fat	3.00
Sugar	6.00
Proteids	1.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION 21.

Four to six weeks.

℞ Fat	3.50
Sugar	6.50
Proteids	1.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 22.

Six to eight weeks.

℞ Fat	3.50
Sugar	6.50
Proteids	1.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 23.

Two to four months.

℞ Fat	4.00
Sugar	7.00
Proteids	1.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 24.

Four to eight months.

℞ Fat	4.00
Sugar	7.00
Proteids	2.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 25.

Eight to nine months.

Fat	4.00
Sugar	7.00
Proteids	2.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 26.

Nine to ten months.

Fat	4.00
Sugar	7.00
Proteids	3.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 27.

Ten to ten and a half months.

Fat	4.00
Sugar	5.00
Proteids	3.25
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 28.

Ten and one-half to eleven months.

℞ Fat	4.00
Sugar	4.50
Proteids	3.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION 29.

Eleven to eleven and one-half months.

℞ Unmodified cow's milk.

At about the tenth or eleventh month I usually begin to give at first one and then two meals daily of equal parts of oat jelly, prepared at the Laboratory, with plain cow's milk heated to 75° C. (167° F.), and a little salt added according to the infant's taste at the time of the feeding. (Preparation of cereals described in Lecture X., p. 281.) Freshly prepared barley or wheat can, if preferred, be given with milk at this age.

In the twelfth month I usually accustom the infant to taking a little bread one day old with its milk, and to be fed from a spoon, so that by the time it is a year old it is taking bread and milk for its breakfast and supper, and oat jelly and milk for the three middle meals.

COLOR OF FÆCAL DEJECTIONS AS INFLUENCED BY THE PERCENTAGE OF FAT IN THE FOOD.

I have considered it of some scientific interest to record the color of the fæcal discharges which corresponds apparently to the percentage of fat in human milk and in the corresponding modified milk. On these two napkins (Plate III., 3 and 4, facing p. 112) are the normal yellow dejections of two infants (Cases 96 and 97) who are being nursed by their healthy mothers and are themselves digesting well and thriving.

Here are also two napkins (Plate III., 8 and 9, facing p. 112) on which are the normal yellow dejections of two infants (Cases 98 and 99) who are being fed on a modified milk which is supposed to correspond to average human milk. The percentages of the fat, sugar, and proteids in this modified milk are respectively 4, 7, and 1. The infants are digesting well and thriving.

You will notice the striking resemblance in color and consistency between these fæcal discharges resulting from human milk and from modified milk, where the percentage of fat is 4.

I have here also to show you the fæcal discharges (Plate III., 7, facing p. 112) of a healthy infant (Case 100), fed on a modified milk having a percentage of 3 for its fat, 6 for its sugar, and 1 for its proteids. You see how much lighter the color of the yellow is.

This change of color is still more strikingly illustrated in this napkin taken from this fourth infant (Case 101, Plate III., 6, facing p. 112), where its modified milk was composed of fat 2 per cent., sugar 5 per cent., and proteids 1 per cent., and where you see the resulting fæcal discharge has a very much lighter yellow color than is the case with the others.

During the last three years I have been able to test the value of this Laboratory by the feeding of nearly three thousand infants, and my experience has been controlled in the practical use of this system by about four hundred physicians. The number of infants that have been fed from the Laboratory each day was about two hundred.

yself believe that by the establishment of these laboratories a new been entered upon in the province of infant feeding, and one which ble us to produce results which have never before been obtained.

re leaving the subject of milk-laboratories, I should like to impress u that the establishment of laboratories for the modification of milk o great a degree been accomplished by the extensive knowledge of ject, the great experience, the unwavering determination, and the stic efforts of Mr. G. E. Gordon, that physicians, as well as all vho are interested in the welfare of infants, must always acknowl-eir indebtedness to him for the great work which he has carried ccessful an issue.

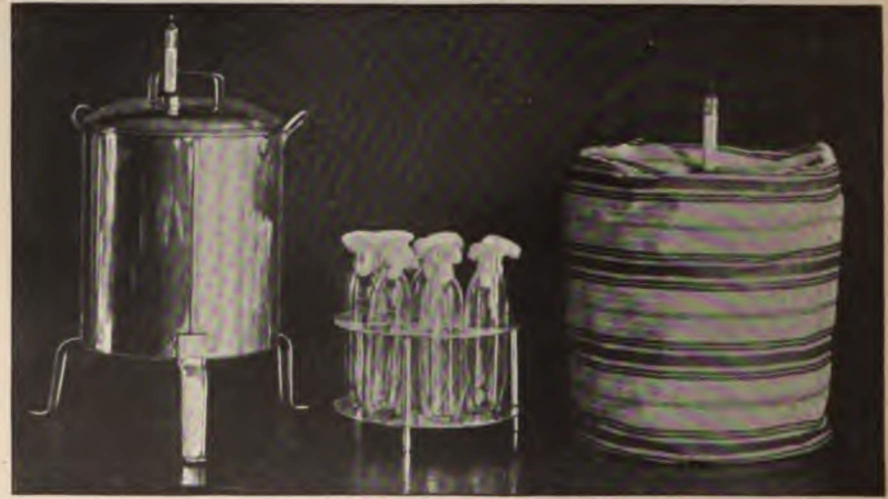
first milk-laboratory for the exact modification of milk that has tablished in the world is the one which I have just shown you, and ened to the public in 1891 here in Boston, under the name of the -Gordon Laboratory.

LECTURE X.

HOME MODIFICATION.—GENERAL REMARKS ON ARTIFICIAL FOODS FOR INFANTS.

HOME MODIFICATION.—I think that you will now agree with me that the importance of modifying milk with the most exact precision is self-evident if we expect to perfect a substitute food. Many persons are not near enough to milk-laboratories to have their infants' food prepared by this means. It is therefore necessary to provide for the preparation of the food for this class of cases in their homes. Under these circumstances I have, in conjunction with Mr. Gordon, made a recent study of the best means to accomplish this end, and I will describe them under the term of "Home Modification." I presuppose that absolute simplicity as to the materials used and such as can be obtained easily is necessary, and also that the method employed should be such as any physician can explain to a mother of ordinary intelligence.

FIG. 68.



Sterilizer and thermometer. Stand for tubes. Sterilizer covered with cozy after removal from heat.

MATERIALS.—I have here to show you the materials which will make possible the home modification of milk for substitute feeding with an accuracy closely approximate, though not equal, to that of the Laboratory. All this apparatus and the same feeding-tubes that I have already described can be procured at the Laboratory for the original outfit.

Home Sterilizer.—This is what is called the "Home Sterilizer" (Fig. 68). It is simply a tin can supported on legs so that it can be heated by a lamp, or, if preferable, the legs can be removed and the can placed on a stove.

Thermometer.—It has a lid, to which is fitted a thermometer by which the degree of heat within the can is indicated.

Tubes.—The tubes, varying in number according to the number of feedings which are required in twenty-four hours, are placed in this stand, which can be lowered into the sterilizer and be immersed in the water in the sterilizer, which is made to rise as high as the level of the milk in the tubes.

Stoppers.—You see that the tubes are stoppled with cotton-wool, according to the usage at the Laboratory.

Cozy.—I have also here another sterilizer, which has been covered with a thick cozy, through which the thermometer from the lid passes and indicates the degree of heat retained within the sterilizer after the flame has been removed.

Graduate.—The other articles to be procured at the Laboratory are this 250 c.c. (8¼ ounces) glass graduate (Fig. 55, p. 251), divided, as you see, into half-drachms.

Cotton-Wool.—Also a roll of aseptic non-absorbent cotton-wool.

Milk-Sugar.—Also some milk-sugar.

Sugar-Measure.—Also this sugar-measure, which holds 13.5 grammes (3⅜ drachms).

FIG. 69.

Sugar-measure.

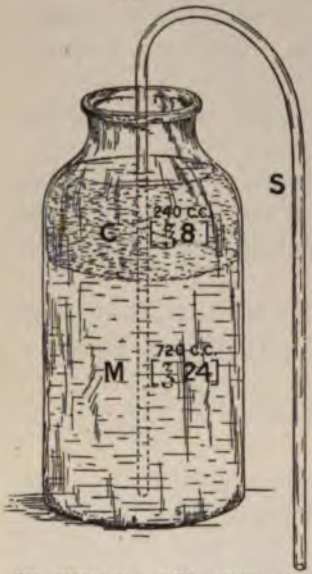
This measure obviates the expense of having the milk-sugar put up in packages by the apothecary, and is sufficiently exact to regulate the sugar percentage in the mixtures which I shall speak of presently. It is well to remember, however, that a pound of milk-sugar contains 464 grammes (7000 grains), and that if you prefer to order the sugar in packages of 13.5 grammes (3⅜ drachms) directly from the apothecary, in place of using the measure, you can simply tell him to make thirty-five packages from the pound, and you can then direct a package of milk-sugar to be used instead of a measureful.

Siphon.—Finally, they must have this glass siphon (Fig. 70), 0.6 cm. (¼ inch) calibre. The siphon can be used in any quart glass jars which the family happen to have.

The siphon should be a glass tube one-quarter to one-half inch in diameter. It can be bent in a gas-flame. The end out of which the milk is to flow should be at least six inches longer than that which is to be inserted in the jar. To operate the siphon, fill it with boiled water, close the longer end with the finger, invert the siphon, and place the shorter end in the milk. Then withdraw the finger, and the water, followed by the milk, will run out of the long arm of the siphon. Do not use the mouth to start the flow of the milk through the siphon, under any circumstances.

The mother is to be told that extreme precautions are to be taken to follow your directions to the minutest detail, or otherwise a uniformly correct result will often be lost. You must explain that the milk from a herd of cows is preferable to that of one cow, for many reasons, but especially because the elemental percentages are less likely to vary in the mixed milk of a herd than in that of the individual, and because the mixing lessens the deleterious effects on the milk arising from occasional disturbance of health in an individual member of the herd. The cows should be of a common breed, and such as give a moderately rich milk. The milk should be drawn with clean hands. The udders and teats of the cows should be cleansed, and the cows should be milked in as clean a place as possible. The milk should be thoroughly strained. You will now have a milk fairly uniform in its elemental percentages and comparatively free from bacteria and foreign matter. The composition of this milk will usually correspond to that which you see in this table (Analysis 40, page 218). The milk is then set in a vessel containing ice and water with some salt, in the proportion of 5 grammes (1 teaspoonful) to 960 c.c. (1 quart) of water, and the vessel is set in some clean place.

FIG. 70.



Jar containing milk, cream, and siphon. C, cream; M, milk; S, siphon.

(Dr. Seibert, of New York, has recommended a system of filtering through a funnel containing aseptic cotton, and asserts that the bacteria are reduced in numbers one-half by this procedure. The fats, however, according to my experience in the use of this method, are also somewhat reduced, though not to any great degree. With the precautions taken, such as I have just stated for obtaining the milk-supply, the cotton filter will probably not be necessary, but it can be used, as Seibert intends it to be, where there are known to be much dirt and many bacteria in the milk. Dr. Seibert has had carefully prepared cotton disks and funnels made for filtering milk in this way.)

You should always endeavor to prevent impurities from getting into the milk in preference to trying to eradicate them after they have begun to alter the normal composition of the milk.

A clean, freshly boiled cotton cloth is next thrown over the uncovered quart jar. The mouth of the jar is kept open for about fifteen minutes, to dispose of the animal heat. The jar is then sealed tightly, as you would do for preserving, and is left in the ice-water for six hours, care being taken that the temperature of the water does not fall below 1.66° C. (35° F.). At the expiration of six hours you are to siphon out carefully from the bottom of the jar with this siphon (Fig. 70, page 278) 720 c.c. (24 ounces) of the milk into a clean glass vessel.

You will now have your various materials ready for any combinations which you may wish to make in preparing the food for an especial infant. These materials are: the milk which you have siphoned from the jar, the cream containing ten per cent. of fat which remains in the jar, the sugar, either in packages as I have just described or in bulk, to be used when needed with the sugar-measure, some fresh lime water, and some clean drinking-water which has been boiled for five minutes.

I have arranged in these tables (Tables 68 to 80) figures by means of which you can make the various combinations which you will be likely to need, and which correspond somewhat to the prescriptions that I have already shown you at the Laboratory:

TABLE 68.

Fat	0.25
Sugar	4.00
Proteids	0.25
Lime water	5.00

To obtain this combination with our materials, and to provide a sufficient quantity of food to last for twenty-four hours, you should give the following orders.

Set enough milk to raise cream sufficient for the mixture required. For each twenty ounces, or part of twenty ounces, use the following formulæ:

Cream	½ ounce.
Milk	1 ounce.
Lime water	1 ounce.
Water	17½ ounces.
	20 ounces.
Milk-sugar	2 measures.

The milk-sugar is to be thoroughly dissolved in the water of the mixture before the other materials are added.

TABLE 69.

Fat	1.00	Cream	2 ounces.
Sugar	5.00	Milk	2 ounces.
Proteids	0.75	Lime water	1 ounce.
Lime water	5.00	Water	15 ounces.
			20 ounces.
		Milk-sugar	2 measures.

TABLE 70.

Fat	2.00	Cream	4 ounces.
Sugar	5.00	Milk	None.
Proteids	0.75	Lime water	1 ounce.
Lime water	5.00	Water	15 ounces.
			20 ounces.
		Milk-sugar	2 measures.

TABLE 71.

Fat	2.00	Cream	4 ounces.
Sugar	5.50	Milk	1½ ounces.
Proteids	1.00	Lime water	1 ounce.
Lime water	5.00	Water	13½ ounces.
			20 ounces.
		Milk-sugar	2¼ measures.

TABLE 72.

Fat	2.50	Cream	5 ounces.
Sugar	6.00	Milk	None.
Proteids	1.00	Lime water	1 ounce.
Lime water	5.00	Water	14 ounces.
			20 ounces.
		Milk-sugar	2¼ measures

TABLE 73.

Fat	3.50	Cream	7 ounces.
Sugar	6.50	Milk	1 ounce.
Proteids	1.50	Lime water	1 ounce.
Lime water	5.00	Water	11 ounces.
			20 ounces.
		Milk-sugar	2¼ measures

TABLE 74.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	None.
Proteids	1.50	Lime water	1 ounce.
Lime water	5.00	Water	11 ounces.
			20 ounces.
		Milk-sugar	2¾ measures

TABLE 75.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	2½ ounces.
Proteids	2.00	Lime water	1 ounce.
Lime water	5.00	Water	8½ ounces.
			20 ounces.
		Milk-sugar	2½ measures

TABLE 76.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	5 ounces.
Proteids	2.50	Lime water	1 ounce.
Lime water	5.00	Water	6 ounces.
			20 ounces.
		Milk-sugar	2¼ measures

TABLE 77.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	7½ ounces.
Proteids	3.00	Lime water	1 ounce.
Lime water	5.00	Water	3½ ounces.
			20 ounces.
		Milk-sugar	2 measures

TABLE 78.

For weaning.

Fat	4.00	Cream	8 ounces.
Sugar	5.00	Milk	7½ ounces.
Proteids	3.00	Lime water	1 ounce.
Lime water	5.00	Water	3½ ounces.
			20 ounces.
		Milk-sugar	1 measure.

TABLE 79.

For weaning.

Fat	4.00	Cream	8 ounces.
Sugar	5.00	Milk	8 ounces.
Proteids	3.25	Lime water	1 ounce.
Lime water	5.00	Water	3 ounces.
			20 ounces.
		Milk-sugar	⅞ measure.

TABLE 80.

For weaning.

Fat	4.00	Cream	8 ounces.
Sugar	4.50	Milk	12 ounces.
Proteids	3.50		20 ounces.

After the various materials have been mixed, in the proportions which I have shown you in these tables, the mixture is prepared for the "home sterilizer." The requisite amount of food for one feeding is poured into each of the tubes. They are stoppled with cotton-wool, care being taken to have a reasonably tight stopple in and a dry neck to the tubes. The tubes are then placed in the rack and lowered into the sterilizer, and the water in the sterilizer is adjusted to the level of the milk in the tubes. Heat, by means of a lamp or stove, is then applied to the sterilizer, which is watched, with the cover off, until the thermometer shows that the water-bath has reached a point of 77.2° C. (171° F.). The lamp is removed as soon as this temperature is reached, the cover put in place, and the cozy over it. The thermometer should mark a temperature of between 75° C. (167° F.) and 77.6° C. (170° F.) for thirty minutes, at the expiration of which time the tubes are to be removed from the sterilizer, and are to be kept in a cool place, preferably the ice-chest, until needed.

OATS.—For the preparation of oat jelly the following method should be employed:

120 grammes (4 ounces) of coarse oatmeal are allowed to soak in a quart of cold water for twelve hours. The mixture is then boiled down so as to make a pint, and is strained through a fine cloth while it is hot.

When it cools, a jelly is formed, which is to be kept on ice until needed. Different proportions of this jelly can be used, but usually it is best to begin with equal parts of jelly and cow's milk. When needed, this mixture is warmed and a little salt is added.

BARLEY.—Barley water is made by boiling 150 grammes (5 ounces) of granulated barley in a quart of water until the volume is reduced to a pint, and then straining.

If a barley jelly is to be made, 120 grammes (4 ounces) of barley flour are employed, and the same process is gone through with as for the preparation of oat jelly. The resulting jelly is treated in the same way with milk as I have directed for oat jelly.

WHEAT.—Wheat can be prepared by the same method as that described for oats and barley.

PEPTONIZED MILK.—For peptonizing milk, the following rules are the most practical and simple:

In a clean glass jar containing 4 ounces of cold distilled or boiled water dissolve 1 gramme (15 grains) of bicarbonate of soda and 0.25 gramme (5 grains) of pancreatine (extractum pancreatis), to which add 12 ounces of whole milk. Set the jar in a vessel of water at a temperature of 41.6° C. (107° F.) for from seven to ten minutes. Cool immediately, and keep on ice until used.

To peptonize modified milk an amount of the powders should be used corresponding with the percentage of the proteids in the mixture, taking the standard of whole milk to be represented by four per cent. of the proteids.

SWEET WHEY.—Sweet whey is best made by the following method:

For each pint of whey needed take one quart of whole fresh milk, to which add 8 c.c. (2 drachms) of the essence of pepsin, or one square inch of rennet. When the proteids have been precipitated, break the curd finely with a fork, and pour off the fluid, straining it through two thicknesses of boiled cheese-cloth.

This removes such of the proteids as are coagulable by acids.

Place this strained liquid in a clean porcelain pot, and raise the temperature to the boiling-point by a stove or a lamp, but do not allow it to boil. Strain this hot liquid through a cloth as before.

This removes the proteids coagulable by heat.

Cool the resulting fluid slowly to a temperature of 10° C. (50° F.), and keep on ice until needed.

ARTIFICIAL FOODS FOR INFANTS.—It would seem hardly necessary to suggest that the proper authority for establishing rules for substitute feeding should emanate from the medical profession, and not from non-medical capitalists. Yet, when we study the history of artificial feeding as it is represented all over the world, the position which the family physician occupies, in comparison with that of the venders of the numberless patent and proprietary artificial foods administered by the nurses, is a humiliating one, and should no longer be tolerated.

If we are abreast of the times, if we but recognize and do justice to the work which has lately been done by our own profession, we surely will not hesitate to relegate to oblivion the statements of the food proprietors, which on box and can, on bottle and printed circular, attempt to stem the slow but inevitably progressing wave of scientific investigation.

It may be well to bear in mind that the attempts which in the past have been made to manufacture cheap foods have been markedly failures. We must first, regardless of expense, learn to produce by modification a perfected substitute food, and not endanger the success of our undertaking by allowing the mercantile side of the question to cripple us in the use of costly methods, which, however, we know to be the best. We should, in fact, remember that the human milk, which we are endeavoring to copy, far from being a cheap product, is a very expensive one.

My own opinion in regard to patent foods, as a whole, is that they must necessarily be unreliable. They are thrown on a market where the competition is extreme, and when once they have been advertised into public notice I cannot but feel that irregularities and changes—slight, perhaps, in the eyes of the makers—may unintentionally creep in and carry their composition still further from that of the standard, human milk.

Analyses show that there is a lack of uniformity in these foods from year to year, and that original claims are apparently forgotten or allowed to give way to cheaper production. In fact, as my experience in the feeding of infants increases, and as I examine year by year the effects of the different foods on infants, I am strongly impressed with the belief that with our present physiological, chemical, and clinical knowledge all the patent foods are entirely unnecessary. The claims made for them are not supported by intelligent and unprejudiced investigation. Those who manufacture them are not in a position to judge correctly concerning them. The merit at times of their apparent success does not belong to them, but to accompanying circumstances. They do great harm by impressing upon the public the false idea that a cheap, easily prepared food is for the good of the infant and is better than anything which can be procured elsewhere. They vary too greatly in their analyses to keep even within the acknowledged varying limits of human milk. It is therefore high time for physicians to appreciate exactly how inefficient in themselves and how misleading in their claims are these artificial foods, and also in what a false position, as the protector of and adviser to the public, our profession is placed whenever it lends itself to even a toleration of their use. I speak of them here simply because there is no doubt that they are kept in the market by the physician rather than by the manufacturer. The latter is only doing what any capitalist interested in a business venture would do. The former, it seems to me, is, perhaps unintentionally, aiding the business interests of others at the expense of his own future reputation as a scientist. It makes little difference to physicians as to what is claimed for these foods when they are placed in the market. It makes a great difference what the mixture contains when given by the mother to the infant according to the directions on the label. For instance, a food may show by its published and certified analysis a fair percentage of fat or sugar, and yet this same food when diluted for the infant's feeding may have these constituents reduced far below the reasonable limits of nutrition.

LECTURE XI.

THE SECOND AND THIRD NUTRITIVE PERIODS.

SECOND NUTRITIVE PERIOD.—During the eleventh and t months of life the amylolytic function of the infant has become almo developed. In accordance with the rule regarding the use of the di functions, which I have already spoken of,—namely, that a function not be taxed before it is developed, but that when its development is completed it should be brought into use,—we should in the latter part first year begin to use that function of the digestive tract by means of the amylaceous elements of the food are converted into sugar.

In speaking of weaning I have already explained to you the w using preparations of oats or barley mixed with milk. I have also you how to reduce gradually the percentage of sugar in the modifie which is being given at the tenth and eleventh months and at th time to increase the percentage of the proteids. The reason for ch the relative percentages of these elements is that the power to digest p has much increased during the latter part of the first year. The c for digesting a high percentage of sugar is just as great at this perio an earlier one, but the amount of sugar, given directly as such, which quired in the later is not so great proportionately as in the earlier A large portion of the sugar which is needed for nutrition in thi period is intended to be introduced into the economy by means of element in the food,—starch. A certain amount of sugar is, as directly introduced into the gastro-enteric tract from the milk-sugar milk, and the starch when converted into sugar supplies the remainin tion of sugar needed for nutrition. In a normal infant with normal di functions a considerable percentage of starch can be digested and ab with benefit in the eleventh and twelfth months.

I am therefore in the habit of giving preparations of oats or when I have decided that starch should be introduced into an infant There is a larger percentage of starch in oats than in barley. It more nutritious in every respect, as it contains a considerable percen fat. The starch in oats takes a somewhat longer time to be convert sugar than does that of barley, so that in the case of an infant amylolytic function is not fully developed or is somewhat weak, p tions of barley will be better to begin with, because they do not duce so high a percentage of starch into the food, and also beca starch will be more readily converted into sugar. Preparations seem to be the best form of food to be added to the modified milk w infant has reached a period at which it needs a change in the character of its food.

When the infant has reached the fifth or sixth month of its life it normally should be able to digest four per cent. of fat in its food. This percentage of fat corresponds, as I have already shown you, to that which exists in average cow's milk. It is natural to suppose that at the eleventh and twelfth months a still further increase in the amount of fat which is provided in the infant's food is required, as well as the new element, starch. This fat is supplied, as I have already told you, in considerable quantity from the oats.

We have therefore, in preparations of oats, both for purposes of weaning and for establishing a new regimen of diet for the infant, a food which in combination with cow's milk satisfies completely the demands which the digestive functions at this period are making for a perfect nutriment.

The second nutritive period may be reckoned to last from the twelfth to the twenty-eighth or thirtieth month of life. That is about the second half of the period which we are in the habit of calling infancy. It also includes the time when the last four teeth of the first set appear. In this second nutritive period the element of variety in the food becomes important. It is undoubtedly important that the actual nutritive values of the food which it is best to give to infants in this period be considered, but it is much more important that special attention be paid to its variety. Foods should be given which while containing a fair percentage of nutritive elements yet differ in the combination of these elements to such a degree that they fulfil the requirements of this period of life. It is best to increase gradually the variety of articles of diet from the twelfth to the twentieth month, always adapting the food to the especial infant. Thus, some infants may be able to digest and assimilate proportionately large quantities of starch; others may both need and digest larger proportions of the proteids or of sugar than the infants first spoken of.

Between the twelfth and thirteenth months I am in the habit of giving the infant five meals during the day. At this time it is well to accustom it to take its food from a spoon, and as soon as possible to omit feeding from the bottle. The five meals should be arranged in the following manner:

For breakfast, bread and cow's milk, slightly warmed.

For lunch, equal parts of oat jelly and cow's milk, warmed, with a little salt added according to the infant's taste.

This meal of oat jelly should be repeated in the middle of the afternoon.

In the middle of the day, broth of some kind, either chicken or mutton, carefully prepared so as to be free from fat on its surface, can be given with some bread.

The fifth meal should be given in the latter part of the afternoon, and should consist of bread and milk.

In some cases it is impossible to make infants swallow bread for a long period after the usual time of twelve to thirteen months. At times it is not until they are two and one-half to three years old that they can be induced to take bread. In these cases we must feed them according to our judgment of the individual case.

When the infant is fourteen to fifteen months old, some thoroughly boiled rice can be added to the broth in the middle of the day, and if it digests this well it can also have bread given with this meal.

When the infant is sixteen months old, it can have a small amount of butter on its bread. When it is seventeen to eighteen months old, it can have a thoroughly baked white potato, mixed with butter and salt, added to its mid-day meal of broth. When it is nineteen to twenty months old, eggs can become part of its diet.

There are not many fruits which should be given to the infant in its second year. A baked apple can be given at the evening meal when the infant is fourteen to fifteen months old; or, for variety, the apple can be made into a simple sauce, never, however, having the sauce made with much sugar. When peaches are in season, a ripe peach can often be given with benefit, especially if the infant is inclined to be constipated. Other fruits should be avoided, as they are not necessary for the infant's nutrition and at times produce serious trouble.

This is the diet which is sufficient for the infant during the second nutritive period. It is important for the subsequent integrity of the infant's digestion and general nutrition that the parents should insist that no other articles of food be employed, except such as are similar to those which I have spoken of,—namely, the cereals in a variety of forms, according to the taste, judgment, and knowledge of cooking which exists in the specia household. For instance, preparations of wheat and barley cooked i various forms may be given in place of oatmeal. Bread also in differen forms may be given. The crust of French bread is easily digested, and i supposed to have less starch in proportion to its gluten than the usual home made bread. It is well to begin with some form of bread of this kin when we are getting the infant accustomed to take starch in the form of bread. If it is constipated, Graham bread and preparations of rye will als be found useful. Fresh bread should never be given, and bread one da old is the preferable form which should be provided.

The infant should never be given cake or candy even to taste. I thin that it is necessary to state this very decidedly, because it is an erroneou view which is held by most mothers that it can do no harm to give occasion ally to an infant in its second year of life, or to a young child, a little cand or a little cake. This may be true so far as the immediate effect these article may have on the digestion is concerned, but it is of far more importance tha the infant should not have its taste perverted from those articles of die which are best for its nutrition. These new articles appeal more strongl to its sense of taste, and allow it to know that there is something whic tastes more agreeable than the food which it is accustomed to have. Whe an infant has acquired a taste for cake or candy, it will cease to enjoy the food by which its development will be best perfected. It is, in fact, kinder to the infant never to allow it to taste cake or candy. When these articles are withheld, it will continue to have a healthy appetite and taste for necessary and proper articles of food.

I am so often asked by mothers what is the best method of preparing simple broths for their infants that perhaps it may be well for you to know how these broths should be made.

CHICKEN BROTH.—A fowl weighing about five pounds should be boiled for about twelve hours. The fluid should be strained while hot through a fine sieve. It should then be allowed to cool in an earthen jar for about twelve hours in the ice-chest. The resulting jelly can be used in full strength or diluted with water. When the jelly has been thoroughly cooled, the fat can be either partially or entirely removed from the top.

MUTTON BROTH.—A shoulder of lamb, when it can be obtained,—otherwise of mutton,—weighing from five to seven pounds, is treated in the same way as is the fowl for the preparation of chicken broth.

THE THIRD NUTRITIVE PERIOD.—The third nutritive period I have arbitrarily made to begin at about the thirtieth month of life.

At this time it will be well to begin to accustom the child's digestive functions to a still greater variety of food. In summer the more easily digestible vegetables, such as squash, young peas, and young beans, can be given. The variety of fruits can also be increased at this period, but they should be cooked. The principal change which is to be made in the diet to which the infant has been accustomed is a very decided increase in the proportion of the proteid element of its food. This is accomplished by means of giving the child meat. The quantity of meat which should be given towards the end of the third year should be small at first, and should be given at intervals of a day or two. Meat as a regular article of diet for each day is not, as a rule, required until the child is between three and four years old. The kinds of meat which should be given in this early period of childhood are chicken, mutton-chop, roast beef, and beefsteak. These meats should be cut into small pieces, and a little salt added according to the child's taste. It is well, during the latter part of the third year and the first half of the fourth year, to give the child an egg on one day and meat on the next.

When the child has reached the age of five or six years, we should allow it to have a somewhat more varied diet, but during the whole period of childhood up to the age of puberty the closest attention should be given to the regulation of the kind and the amount of food to be given to the child, and any deviations from the rules which I have just laid down are to be deprecated.

DIVISION V.

PREMATURE INFANTS.

LECTURE XII.

I SHALL next speak of that class of infants which is designated
mature, because they are born prior to the usual two hundred and
days which represent the normal duration of intra-uterine life. I d
this class of cases directly after what I have just told you about infa
ing not only because it is essentially the proper management of t
which preserves the lives of these infants, but because I consider t
best way to feed premature infants is by means of food carefully prep
milk-laboratories. This method of feeding premature infants is far s
to even breast-feeding, and, in my opinion, the use of milk-laborat
these cases will result in a decided reduction in their mortality.

Very few cases are reported, and none of them appear to be ab
authentic, where an infant has survived which was born much bef
twenty-seventh or twenty-eighth week of intra-uterine life. The pr
infant in its intra-uterine development is unprepared to meet the co
of extra-uterine life, and often dies within a few days, and usually
a few hours.

A sufficient number of careful investigations regarding the chara
appearances and the development of the fœtus during the last four
of intra-uterine life has not yet been made and recorded to enabl
state definitely what age the infant represents when it is born. T
facts which we possess concerning this subject must, however, be m
of, and, though not absolutely correct, are sufficiently so to be of gre
to us in our management of these cases. One reason for the difficult
arises in every case in determining the age of the fœtus is that th
tions which influence its growth during intra-uterine life are very
The health of the mother and her hygienic surroundings, together w
influence of heredity on the size of her offspring, present good rea
decided variations in the growth of the fœtus in different cases at t
period of intra-uterine life.

If the infant is living when it is born, we should at once carry rom its small weight and general characteristics have the appear- ne whose development is incompatible. Whatever advances we in the future in preserving the lives of premature infants born ier date than is supposed to be compatible with life,—namely, from y-fourth to the twenty-eighth week,—it would hardly be practical ie to discuss the treatment of infants born before the twenty-fourth

NTY-FOUR WEEKS.—A fœtus born at about the twenty- ek of intra-uterine life usually breathes feebly, and dies in the a few hours, apparently from an inability to accommodate itself ons for which it is not prepared. At this stage of development ill have fine hair (lanugo) over the whole of its body, but it is case that this hair, commonly found from the sixteenth to the week, has disappeared. At this age it still has very little deposi- in the subcutaneous cellular tissue, and it has a decidedly emaci- rance. In other respects, except its size, it does not differ very its appearance from the fœtus of some weeks' later development. s have separated, though it is so feeble that, as a rule, it cannot shut them.

stimation of the length of the fœtus is difficult to make, and, on , unsatisfactory and inexact. These measurements, in all proba- fer very much when made by different investigators, owing, as pointed out, to the many changes in the curvature of the longi- xis of the human embryo, which make it impracticable to employ ystem of measurement in obtaining comparable results for all ages. figures, however, are probably as reliable as any we know of. to this author, at about the twenty-fourth week the fœtus meas- o 34 cm. (11¼ to 13½ inches). Its weight, according to Lusk, is grammes (23 ounces).

NTY-EIGHT WEEKS.—By the time the fœtus has reached the of intra-uterine development is less likely to insure continuance of life after premature birth than a later stage, provided the same precautions are taken in each case.

Hecker's and Lusk's figures, in a general way, state that when the fœtus is born at about the twenty-eighth to the twenty-ninth week it measures from 35 to 38 cm. (about 13¾ to 15 inches) and weighs about 1170 grammes (39 ounces). The skin is still wrinkled, is of a dull red color, is covered with vernix caseosa, and there is very little deposition of subcutaneous fat. The infant can move its limbs slightly, cries feebly, and often dies in a few hours or days. Yet it is this class of prematurely born infants whose lives I expect to see preserved in the future, when all the precautions which I am about to describe against external and dangerous influences have been taken and improved apparatus has been employed.

THIRTY-TWO WEEKS.—Again, using Hecker's and Lusk's figures for the thirty-second, thirty-sixth, and thirty-eighth weeks, at about the thirty-second week of intra-uterine life the fœtus measures from 39 to 41 cm. (about 15½ to 16½ inches) and weighs about 1560 grammes (52 ounces). The hair of the head by this time has increased in thickness, and the lanugo, which in many cases is pronounced from the twenty-eighth to the thirty-second week, has either begun to disappear or has entirely disappeared from the face. The nails, which between the twenty-eighth and thirty-second weeks are often not well developed, now present a normal appearance, though they frequently do not quite reach the tips of the fingers. At this age, also, in boys, it is often possible to feel the testicle in the scrotum. There is usually, also, at this age, in a healthy fœtus, considerable deposition of subcutaneous fat, and the senile aspect of the earlier periods of intra-uterine life is much lessened.

THIRTY-SIX WEEKS.—At about the thirty-sixth week the length of the fœtus is from 42 to 44 cm. (about 16¾ to 17½ inches) and its weight is about 1920 grammes (64 ounces). The lanugo has usually at this period disappeared, and the infant, although less energetic than at full term, is decidedly stronger than in the previous periods which I have mentioned. It sleeps a great deal, and is still in a condition to die easily unless carefully looked after.

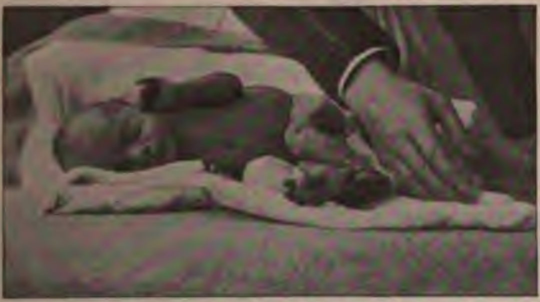
THIRTY-EIGHT WEEKS.—At about the thirty-eighth week of intra-uterine life the infant measures about 45 to 47 cm. (about 17¾ to 18¾ inches) and weighs about 2310 grammes (77 ounces).

WEIGHT.—It is important to remember that the weight of premature infants of the same age varies at birth, just as we have seen that it does in the case of infants born at term.

In treating these cases, observance of their weight is of the greatest importance, and until we have obtained a regular progressive daily increase in their weight we are never sure that they are thriving sufficiently to live. The daily gain which the premature infant should make has not yet been determined, but it is much less than is expected when an infant is born at ull term, and may be stated to be about 10 to 20 grammes (⅓ to ⅔ ounce). Any decided loss in weight, such as 30 to 40 grammes (1 to 1⅓ ounces), beyond what would occur from natural causes, should make us look upon the infant as being in a critical condition and impress upon us the importance of taking active measures to prevent further loss. This loss in weight must, as it is relatively so small, be carefully adjusted to the loss which naturally occurs from the fæcal discharges. Thus, the total amount of loss in weight from the fæcal discharges may amount in these premature infants to from 30 to 60 grammes (1 to 2 ounces) for each fæcal discharge, and this may entail a considerable loss of the infant's weight in the twenty-four hours beyond that occasioned by defective nutrition.

I have here to show you an infant (Case 102) prematurely born at about the twenty-eighth week of intra-uterine life.

Case 102.



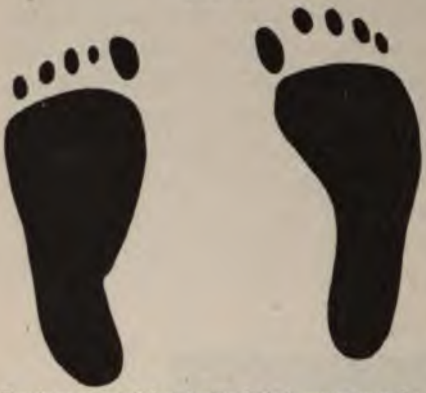
Infant premature at seventh month. Birth-weight, 1740 grammes; present weight, 1540 grammes; present age, 10 days.

Its weight at birth was 1740 grammes (about 3⅚ pounds). It is now ten days old and has lost about 240 grammes (about ½ pound). You see that it is in a very somnolent condition, that it has very little hair on its head, and very little subcutaneous fat. You will also notice the senile expression of its face, that there is no appearance of lanugo, and that the nails are well formed. The small size of the infant will be still more appreciated if you compare it with the hand of the nurse, which, for comparison, is placed beside it.

There have been so few observations recorded of the development of the various parts of the fœtus in the later months of intra-uterine life that I am not prepared to describe systematically the development of the premature infant as I have already done that of the infant at term (Lecture III., page 54). There are, however, some facts which I have observed and others which have been recorded.

HEAD, THORAX, AND ABDOMEN.—Looking at this infant (Case 102) critically, we notice that all those anatomical conditions which I have emphasized in my description of the infant at term as being especially prominent are still more marked in the premature infant. Thus, you will notice how large the head is in comparison with the thorax, and how very large, in proportion, is the abdomen. The abdomen is in almost every case much distended in premature infants, owing to the large proportionate size of the liver. This distention of the abdomen lasts for many weeks, and even months, and its gradual return to the normal size and appearance is one of the signs that the infant is doing well and is gradually acquiring the normal anatomical development of the infant born at term.

FIG. 71.

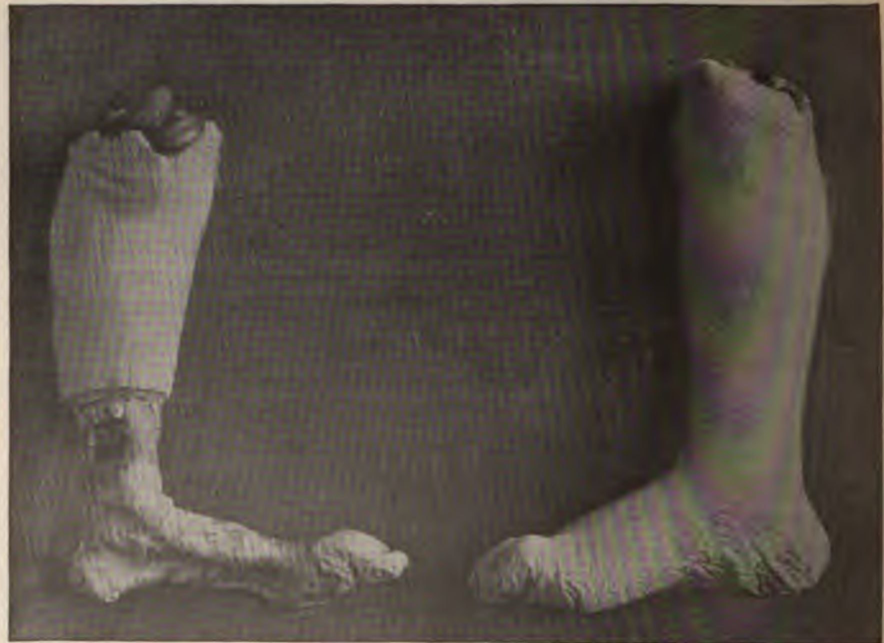


Tracings of fœtal feet (natural size), seven months old.

SKIN.—The various changes in the color of the skin, which I have already described as represented by erythema neonatorum and icterus neonatorum, I have noticed to occur in the premature infant as they do in the infant at term.

SWEAT-GLANDS.—I have told you that the function of the sweat-glands is, as a rule, not developed at birth, and that we do not expect the infant in the early weeks of life to perspire. I have also told you that there is a great variation as to the time of the development of the function of the sweat-glands. In an infant premature at seven and one-half months I have noticed free perspiration take place after it had been born one week.

FIG. 72.



Legs and feet (natural size) of infant premature at seven months. Left foot dissected and showing internal arch. Partially dissected metatarso-phalangeal joint of great toe in shadow. Inferior edge of scaphoid shows as a shaded line.

FEET.—I have already told you how few observations have been made on the development of the various parts of the fœtus in the later months of intra-uterine life, and I think all facts determined at this period of existence should be recorded. I therefore consider Dane's observations on the instep of a seven months' fœtus (Case 103) born alive to be of value in connection with what I have already said about the feet of infants born at term (Lecture II., p. 50). These tracings (Fig. 71) represent this premature infant's feet, and you see how admirably the presence of the instep is shown.

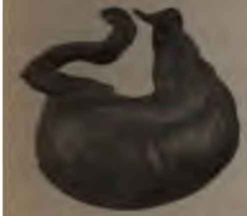
The infant died a few hours after birth, and I have here to show you (Fig. 72) its feet and lower legs. The skin of the right leg is badly wrinkled by the alcohol in which it was preserved. It looks strikingly like the foot as it appears in adults, and its arch stands out plainly, unmasked by any pads of fat. The left foot has been dissected down to the ligaments. The arch made by the os calcis, cuboid, and fifth metatarsal bones represents the lower edge of the cut. On the inner border of the foot the metatarso-phalangeal joint of the great toe is seen partially dissected. Behind this the enlargement at the tarso-metatarsal joint forms a considerable bulging. Behind and above the latter the inferior edge of the scaphoid shows as a shaded line.

From these specimens and from the tracings we see that the foot at seven months closely approaches in external appearances the well-developed foot of the adult, and that when the infant was supported with its feet on smoked paper it left an excellent impression. The dissection also shows a remarkably well constructed bony framework.

GASTRIC CAPACITY.—As the question of the proper amount of food to be given to a premature infant is of the utmost importance, it is well to know about what the average gastric capacity of the fœtus is during the later months of intra-uterine life. No series of complete and reliable observations on this point have been made, that I know of, and the rules by which we are guided must for the present be very general ones. The less the weight of the infant, the less, in many cases, is the gastric capacity. I have here to show you some fœtal stomachs.

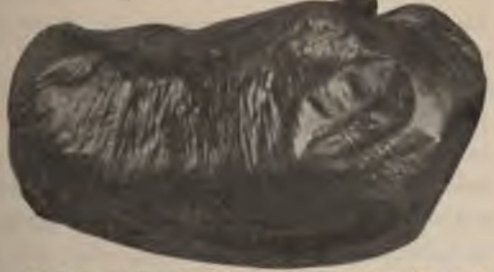
The first stomach (Fig. 73) is that of a fœtus about four and one-half months old, and is interesting merely as showing the relatively advanced development of the lesser and greater curvatures at this age, as well as the rapid growth which takes place between the fourth and the seventh month.

FIG. 73.



Fœtal stomach (natural size), four and one-half months old.

FIG. 74.

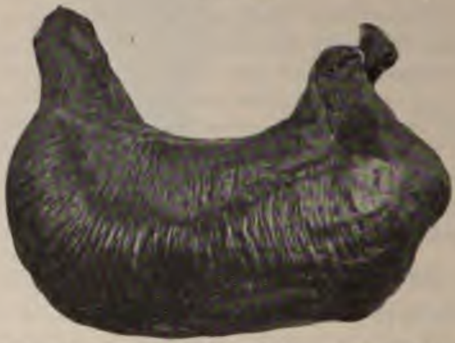


Fœtal stomach (natural size), seven and one-half months old. Weight of fœtus, 1920 grammes. Gastric capacity, 18 c.c.

The next stomach (Fig. 74, p. 293) was taken from an infant born prematurely at about the twenty-ninth to the thirtieth week. It is of a rather peculiar shape, corresponding to that which I have described to you in a previous lecture (Lecture IV., Fig. 29, p. 89). The weight of this fœtus was 1920 grammes (about 4 pounds). Its gastric capacity was 18 c.c. (about 4½ drachms).

This next stomach (Fig. 75) was taken from a fœtus at about the thirty-second week, which died in forty-five minutes from the time of its birth. The gastric capacity was 22 c.c. (5½ drachms). The weight of this infant was 1230 grammes (2 pounds 9 ounces).

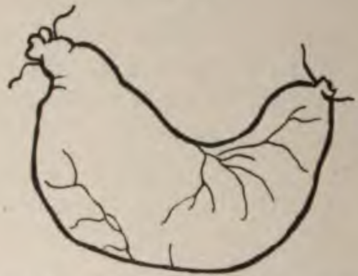
FIG. 75.



Fœtal stomach (natural size), eight months old. Weight of fœtus, 1230 grammes. Gastric capacity, 22 c.c.

The next stomach (Fig. 76) was taken from a fœtus born at about the thirty-second week of intra-uterine life, and weighing 1440 grammes (about 3 pounds). Its gastric capacity was 8 c.c. (about 2 drachms).

FIG. 76.



Fœtal stomach (natural size), eight months old. Weight of fœtus, 1440 grammes. Gastric capacity, 8 c.c.

INTESTINAL CONTENTS.—The meconium in premature infants presents the same appearance as is seen in infants at term. When the food is properly regulated, the fæcal discharges assume the consistency and color which are seen in those of infants who have been born at term. This color in its usual varieties is well represented in this plate (Plate III., 6, 7, 8, 9, facing p. 112).

AMYLOLYTIC FUNCTION.—The amylolytic function of the infant at term is so slightly developed that we may safely assume that it should not be depended upon for the digestion of starch in the premature infant under any circumstances.

SUGAR.—Although we must assume that the function of absorbing
r is not developed to the same extent in the premature infant as in the
nt at term, yet, in all probability, it is more highly developed than the
r functions of digestion. Sugar is needed to keep up the animal heat
he premature infant, which is so very much more readily lessened than
he infant at term. Sugar, therefore, is an important element in the
ature infant's food, but should be given at first in a much lower
ntage than later, when the equilibrium of the gastro-enteric tract has
acquired.

AT AND PROTEID DIGESTION.—The function of digesting fat
proteids is in a much more undeveloped condition in the premature
t than in the infant born at term, and should, therefore, not be de-
ed upon to the same degree as can safely be done in arranging the food
he older infant. Much smaller percentages of these elements should
iven to the premature infant than to the infant at term, both for pur-
of digestion and of absorption, for, in all probability, the power of
rption of the gastro-enteric tract in premature infants is in a very un-
loped condition.

IDNEY.—We should expect, from the lack of development of the
ey in premature infants, to find a considerable deposit of uric acid, such
have described as appearing in the early days of life in infants at term
ture IV., page 111, Plate III., 1). This is, in fact, the case, and the
arance of uric acid on the napkins of premature infants is, therefore,
ecessarily to be looked upon as denoting an abnormal condition. It
ld, however, be carefully watched, for where it becomes excessive it is
ndication that the infant's food has not been properly adjusted to its
tive powers and that the infant may soon begin to fail.

IRCULATION.—The heart in premature infants has not yet arrived
e complete stage of development needed to render it a reliable central
which can fulfil the demands that will be made on it in the external
d to sustain the equilibrium of the circulation. Therefore as little
as is possible should be thrown upon the heart, and the infant
ld be kept quiet, and not be carried about, as is customary with
ts born at term.

n a number of cases which I have carefully examined I have failed
etect a cardiac murmur, which leads me to think that the foramen
e closes soon after birth in the same manner as it does in the infant
rm.

NIMAL HEAT.—The animal heat of the premature infant is much
e easily reduced, and is even more important to its vitality, than it is in
infant at term. Following the rule that the smaller the size of the
an being the greater proportionately is the entire surface, and, therefore,
greater the opportunity for lowering its temperature, an atmosphere
ch is suitable for the infant at term is too cold for the premature
nt.

Premature infants should be thoroughly protected from changes of te perature of the atmosphere in which they live, and this temperature sho be raised to a point which will correspond in some degree to that of int uterine life.

AIR.—Just as a necessity exists for the premature infant to live some weeks in an atmosphere where the air approaches in its temperat the warmth which exists in intra-uterine life, so is it almost to the degree important that the air which it breathes should be free from dust micro-organisms. The lung is in a very undeveloped condition, and altho it may be sufficiently developed to carry on the function required of it extra-uterine life, yet all its tissues are exceedingly sensitive, as are those the nose and naso-pharynx through which the air must be introduced to lungs. The air of the ordinary room where infants live when they are b necessarily contains many impurities, both irritating and morbid. T irritation of the respiratory passages may of itself be sufficient to reduce vitality of the infant beyond the limits of life.

TOUCH.—Premature infants have to be carefully handled, as they easily from influences which would have little or no effect upon the in born at term. In intra-uterine life they are floating in a fluid which p tically prevents what in the external world corresponds to *handling*. W. they are living in the amniotic fluid they are almost completely prote from the influence of touch, which necessarily affects them as soon as t are born. *Touch*, then, is an important element, to be as much as poss avoided when the premature infant is born, as it has a decided tendenc lower the vitality.

An instance of the care which is needed to preserve the lives of t infants came to my notice in the case of an infant (Case 104) prematur eight months which was in my service at the City Hospital.

During the first week or ten days of its life this infant was in charge of an unusu careful and experienced nurse, who appreciated the risk of handling it. It was gainin weight and was doing well; but unfortunately another nurse was substituted who did understand this class of infants so well. She allowed the patients in the ward to ha the infant, to talk to it, and to surround it with various similar deleterious influences. a few days it lost in weight, and then it suddenly died. There is no doubt that it was able to withstand the amount of handling which would have done no harm to an infant.

LIGHT.—The premature infant should live in comparative dark during the early weeks of its life. Light is not requisite for the devel ment of the infant in the earlier stages of its existence, and too much li will impair its vitality. It is important to adapt the light to the st of its development, and gradually to accustom it to more light as it gr older.

SOUND.—In the normal intra-uterine conditions the infant is v slightly exposed to sound, and all its functions are adapted to silence rat than to the many noises which unavoidably surround it in the exter world. We should therefore arrange that from the minute it is born it is protected from noise.

PULSE, TEMPERATURE, AND RESPIRATION.—I have not any very exact records of the average pulse, temperature, and respiration found in premature infants. These infants seem to present rather irregular types of temperature and pulse, as well as of respiration. They have to be so carefully handled that observations as to these physical signs must be made with great caution. The main point in regard to these three conditions of the premature infant is that they are all represented by irregularity. The *temperature* of the premature infant, when it has once begun to gain in weight and to thrive, is usually a little above the normal temperature of the infant at term. Before it has begun to gain in weight and when its vitality is much depressed, the temperature, as would naturally be expected, is rather below the normal standard; and we should watch this sign with the greatest solicitude, as a decided and continuous depression is often indicative of death.

The *pulse* is difficult to take in the premature infant, and, as a rule, is somewhat quicker than in the infant at term.

The *respirations*, irregular in the infant at term, are still more irregular in the premature infant, at times being rapid for a few seconds, and then becoming almost imperceptible for some minutes.

This infant which I have had brought here to show you (Case 105) was prematurely born at the thirty-second week, and illustrates the fact that a premature infant, if its weight is not extremely small and if its development is somewhat above the average expected for its age, can live and thrive without all the precautions being taken for its preservation which I have already spoken of. These cases, however, merely emphasize the fact that if we are guided by them in our treatment of premature infants in general, we shall make many fatal mistakes and far fewer lives will be saved.

This infant weighed at birth 2954 grammes (about 6½ pounds). This would indicate that its chances for living were good, the other conditions of its development being normal, as you will understand by referring to this table (Table 2, p. 49) of the relation of weight to vitality. You see that the weight of this infant is between 2500 and 3000 grammes, showing that the vitality has risen above what is designated as low; in fact, it is within 49 grammes (about 1¾ ounces) of the 3000 grammes which represent a fair vitality. The infant was kept in a room where the temperature was 23.8° C. (75° F.). The air which was around its bed, which was in a basket, was heated to about 29.4° C. (85° F.). The infant was wrapped in fresh absorbent cotton. During the first twenty-four hours one teaspoonful of food was given every hour. After that time it was fed every hour during the day, and every two hours during the night. On the third day the mother had a sufficient supply of breast-milk, which flowed easily. The infant was therefore fed with the breast-milk from a spoon for a week, was then put directly to the breast, and continued to nurse until the end of the third week, when, as its mother's milk failed, it again had to be placed upon a carefully regulated substitute food.

There is nothing else especially interesting to record either in its history or in its physical condition, except that it had a small umbilical hernia, which did not cause any discomfort, and which closed at the end of the third month.

With this attention to its warmth and food it throve as any infant at term would have done, and has since been well and strong.

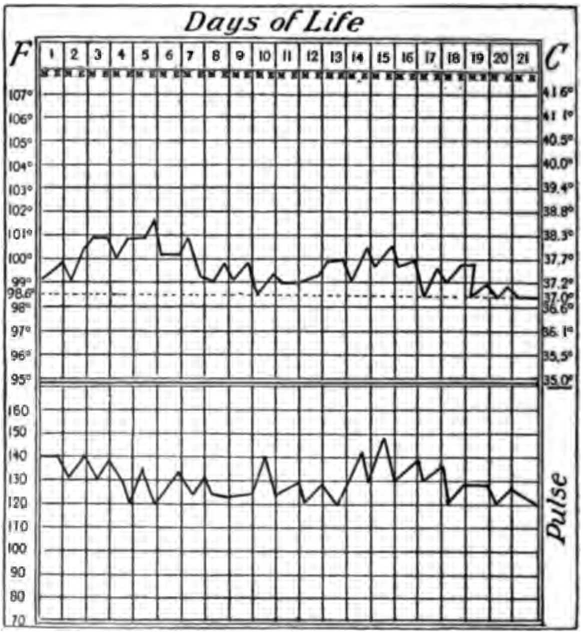
Here is a table (Table 81) which represents its weight for sixty-one days, and here is the record (Chart 5) of its temperature and pulse during the first three weeks of its life.

TABLE 81.

Weight for Sixty-One Days of Infant Premature at Thirty-Two Weeks.

Day of Life.	Grammes.	(Pounds.	Oz.)	Remarks.
Birth-weight . . .	2964	(6	8)	Cow's milk, with spoon.
Third	2724	(6	0)	Mother's milk, with spoon.
Sixth	2814	(6	3)	" " " "
Ninth	2964	(6	8)	Mother's milk direct from breast.
Thirteenth	3178	(7	0)	" " " " "
Sixteenth	3388	(7	7)	" " " " "
Twentieth	3598	(7	14)	Modified milk.
Twenty-third . . .	3812	(8	6)	" "
Twenty-seventh .	4116	(9	1)	" "
Thirtieth	4236	(9	5)	" "
Thirty-third . . .	4476	(9	13)	" "
Thirty-seventh . .	4600	(10	2)	" "
Forty-first	4840	(10	10)	" "
Forty-fourth . . .	4900	(10	12)	" "
Forty-eighth . . .	4994	(11	0)	" "
Fifty-first	5084	(11	3)	" "
Fifty-fifth	5234	(11	8)	" "
Fifty-eighth . . .	5324	(11	11)	" "
Sixty-first	5384	(11	13)	" "

CHART V.



All the possible causes which may reduce the premature infant's vita must be thoroughly understood and obviated. You must also apprec that a failure to recognize and obviate one of these causes may defeat benefit which may arise from attending to all the others.

The premature infant should, so far as is possible, be restored to

ion that it has been forced out of,—namely, a condition of darkness,
, and warmth.

u see, therefore, that there are a great many points to be considered
you undertake to treat intelligently an infant prematurely born, and
is treatment which I shall endeavor to explain to you.

MOUNT OF FOOD AT EACH FEEDING.—I have already stated
e amount of food to be given at each feeding is very important. By
ng to the weights and gastric capacities of the premature infants
y described (Figs. 74, 75, 76, page 294), you will see how misleading
weight of the infant if we take it as an exact index of the gastric
ty. We must, however, take the weight into account, as, from even
ry imperfect data at our command, the weight of premature infants
s to bear a decided relation to their gastric capacity. We should
st be more inclined to increase rapidly the initial amount of food
in the case of an infant of large weight than in that of a small one.
etter to begin with too small rather than too large a quantity. By
ing carefully for signs of hunger, a desire which the infant expresses
ble but continuous cries, which stop when the food is given to it,
n gradually increase the amount until it seems to want it at regular
als, is satisfied, and sleeps quietly during the intervals of feeding.

referring to these fœtal stomachs (Figs. 73, 74, 75, 76), you will
tand that it is safer to begin with 4 or 5 c.c. (about 1 drachm) and
lly to increase the amount up to a point where our very imperfect
edge on this subject, derived partly from the weight of the infant,
us believe that the stomach is full, than to begin at once with the
amount. It is absolutely necessary that we should avoid undue dis-
of the stomach, as this may prove fatal.

TERVALS OF FEEDING.—The premature infant's stomach is
and is, in all probability, emptied quickly, and, as food is necessary
eping up the animal heat required for the maintenance of its life, the
als of feeding should be much shorter than those required for the
at term. In the early days, and in fact weeks, of life, I have found
is better to feed the premature infant regularly every hour. Four or
eks after birth, if it is gaining in weight and is digesting well, these
als can be so lengthened that by the time it arrives at term we can
make the feeding intervals one and a quarter to one and a half
and a few weeks later two hours.

OMPOSITION OF FOOD.—The careful adjustment of the pre-
e infant's food to its digestive organs is of even greater importance
in the case of the infant at term. There is no doubt that if we con-
the hypersensitive condition and the undeveloped state of the digestive
s prior to birth, the most exact adjustment of the food to these
ive organs is absolutely necessary. This adjustment is best accom-
d by means of carefully prepared prescriptions at the Milk-Laboratory.
gh this instrument of precision three important advantages are gained:

(1) we insure a clean food free from micro-organisms; (2) we can obtain low and properly balanced percentages of the constituents of the milk; (3) we have, at any time, the power of exactly varying, to within a fraction of one-half of one per cent., the percentages of the three most important elements of the milk,—namely, the fat, the sugar, and the proteids. In addition to these latter two advantages possessed by the substitute over the maternal method of feeding are others of almost equal importance. One advantage is the absence of variation in the substitute food arising from emotional causes, and another is that the infant need not be taken from the incubator to be fed.

This prescription (Prescription 30) is the one which I should begin with in feeding an infant premature from the twenty-eighth to the thirty-sixth week:

PRESCRIPTION 30.

℞ Fat	1.00
Sugar	3.00
Proteids	0.50

24 meals, each 4 c.c. (1 drachm).
Heat to 75° C. (167° F.).
Reaction faintly alkaline.

If the infant is over twenty-nine weeks, or if it is unusually large fo its age, and especially if it is unsatisfied, it is well in a few days to chang the prescription to this one (Prescription 31):

PRESCRIPTION 31.

℞ Fat	1.50
Sugar	4.00
Proteids	0.50

24 meals, each 8 c.c. (2 drachms).

If the infant is over thirty-two weeks, vary the prescription in a fe days, under the same conditions as in Prescription 31, to—

PRESCRIPTION 32.

℞ Fat	1.50
Sugar	5.00
Proteids	0.75

24 meals, each 12 c.c. (3 drachms).

If the infant is over thirty-six weeks, the milk should, after forty-ei hours, be increased and strengthened to—

PRESCRIPTION 33.

℞ Fat	2.00
Sugar	5.50
Proteids	1.00

24 meals, each 16 c.c. (4 drachms).

e infant, however, under all circumstances, must be watched critically,
ny or all of the percentages of the elements or amounts of the food
ed or decreased according to the individual indications.

hen the infant is born at the thirty-eighth or thirty-ninth week its
pment is usually so near that of the infant at term that the incubator
ot be needed, and the food can be given in about the proportions
would be adapted to the early days of the infant at term (Prescription
e 181).

EIGHING.—A knowledge of the weight of the infant is exceedingly
tant in the management of its feeding, and changes in the degree of
lity take place so rapidly that the daily increase or decrease in its
t becomes the principal index by which we are guided in changing the

ie handling, however, which is necessary to obtain the daily weight is
a serious obstacle to the maintenance of its vitality. We should,
re, endeavor to obtain the weight without reducing the vitality. The
for doing this I shall describe later.

EANSING AND CLOTHING.—A premature infant should not
hed beyond what is necessary for simple cleanliness.

should not be dressed, but should be wrapped in absorbent cotton.
otton soon cleanses it thoroughly, and, if changed twice daily, or
r if necessary, supplies the place of both clothes and bath. As a rule,
or ointment should be applied to its skin.

CUBATORS.—I have already told you, when speaking of milk-
tories, that it is better not to spare expense in obtaining the very best
for preserving life which comes within our power. If you appreciate
reat principle, which lies at the root of all successful methods of pre-
g the lives of premature infants, you will understand that even the
st details which I have spoken of, and which I shall again mention
at length, are not to be considered trivial or beneath your earnest and
l attention. The premature infant's life is so difficult to preserve
re should make use of every device which our ingenuity can suggest.
the very moment it enters the world its viability is likely to be
ht to an end, and every minute is of importance in our endeavors
nbat this tendency. We should, therefore, be ready to protect it at
from the adverse influences which surround it. We should have
d views of how to treat this early period of life, and also have
eans which we think should be best employed ready to be supplied
e.

the treatment of premature infants only one of the principal methods
intaining their viability usually receives much attention. It is com-
y supposed that if the atmosphere which surrounds the infant is kept
sufficiently high temperature all that is requisite has been done for its
r. This until very recently has been accomplished by placing the
t in a room where the temperature is as high as the nurse in charge

of it is able to endure. This procedure is necessarily a very uncomfortable one for the nurse, and at times renders it almost impossible for her to use her mind intelligently. It also requires a much more frequent change of nurses than would be the case if the atmosphere of the nursery were cooler. In addition to this means of preventing undue loss of heat, the infant is wrapped in cotton-wool and placed in a basket lined with hot-water bottles, or it is placed at once in an apparatus which is called an incubator. These incubators have been used for many years in different parts of the world, notably in Paris. They are of different forms, which I need not describe here, as there is nothing especially important to recommend about them when we compare them with the latest form of incubator, which I shall presently describe to you (Fig. 80, page 306). The purpose of them all is the same,—namely, to keep the infant warm. Some of them are made of tin, with double walls, so that hot water can be continually kept in them, and thus sufficient warmth be applied to the infant. Others are made of wood, and kept warm by means of hot-water bottles introduced into them from below. None of them combines in the best way the many requisites necessary to preserve the premature infant's life.

The name incubator has been applied to these various devices for keeping up the animal heat of the infant. It is a misnomer, for incubation means hatching, and, in the precise sense of the word, the premature infant is already hatched and has been incubated. What we accomplish by this apparatus is analogous to what is done to keep up the animal heat and preserve the lives of young chickens after they are hatched, and the name *brooder* would be more applicable to machines devised for preserving the lives of premature infants than the term *incubator*. The word incubator is, however, so generally used to represent an apparatus intended to preserve the premature infant's life until it has attained the age of two hundred and eighty days, that it will, in all probability, for the present be retained. The true meaning, however, of what I am endeavoring to explain to you is so much better expressed by the word *brooder*, meaning warming, and not hatching, that I shall use it in speaking of the latest apparatus which has been invented for the purpose of human brooding.

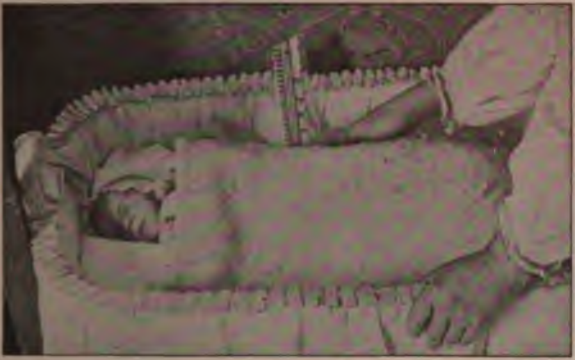
Before speaking of the treatment of premature infants where every detail can be carried out in the most approved manner, I shall mention a few cases which illustrate the different points to which I have just referred. For instance, where it is impossible to obtain an incubator at once for preserving the premature infant's animal heat, it must be treated in the way which I have already referred to, by placing it in a room where the temperature has been raised to 32.2° C. (90° F.).

I have here a picture (Case 106) representing an infant premature at the seventh month, and now fourteen weeks old.

It is in this basket, enveloped in cotton-wool, and covered with blankets. You that the thermometer is kept in the basket beside it, and the nurse has continually watch it.

It was under the care of Dr. Hunt, of West Newton, with whom I saw it in consultion. It was placed in the incubator when it was four weeks old. It was taken out of e incubator when it was twelve weeks old. At this time it had gained very little in

FIG. 77.

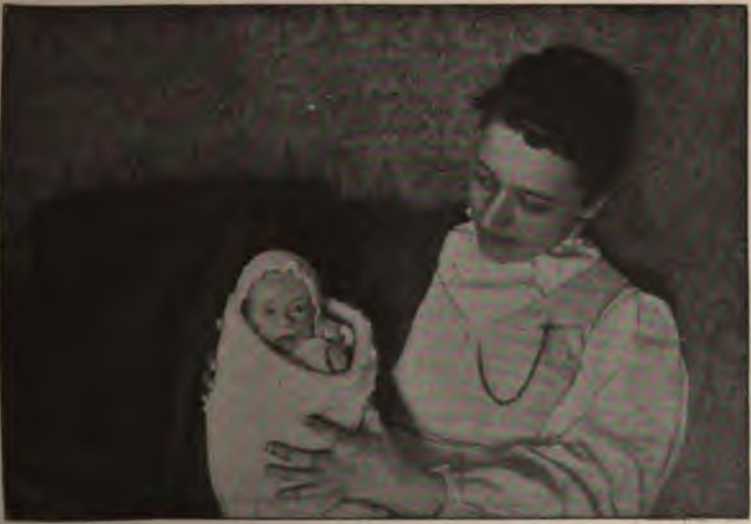


Infant premature at twenty-eighth week. Birth-weight, 1200 grammes. Present age, fourteen weeks. ated in basket heated by hot-water bottles. Temperature of air in basket shown by thermometer infuced between side of the basket and the blanket. The infant was removed from the incubator when as twelve weeks old.

ght, was emaciated, puny, and feeble. Its abdomen was much distended, and its skin kled, dry, and yellowish in color.

Here is a picture (Fig. 78) of this infant taken when it was fourteen weeks old, ch shows the senile expression of the face so characteristic of premature infants at and later when they are not thriving.

FIG. 78.

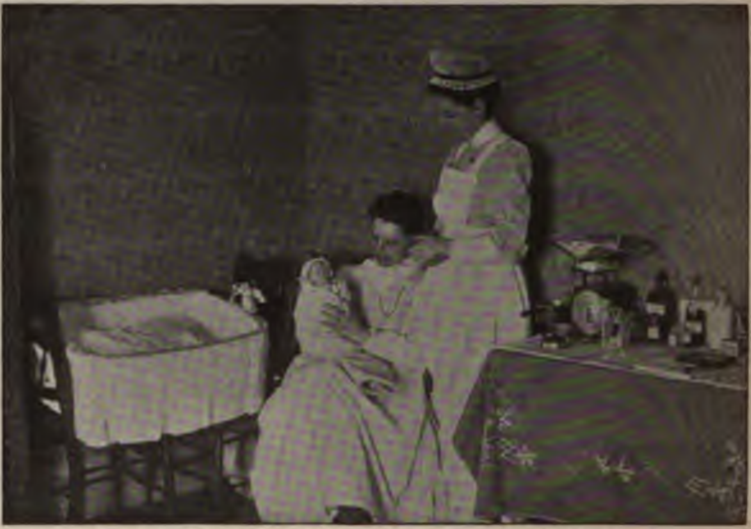


Infant premature at twenty-eight weeks. Present age, fourteen weeks.

Here is another picture (Fig. 79) of this infant, with its day nurse and its night nurse, basket, and the scales on which it was weighed daily.

This picture is instructive in making you appreciate how small this infant was, as is well shown by comparing the size of its head with that of the nurses' heads.

FIG. 79.



Infant premature at twenty-eight weeks. Present age, fourteen weeks.

The next infant (Case 107) which I shall speak of was one which was prematurely born at about the thirty-third week. It was treated in a basket warmed with heaters, and in a room where the temperature was kept from 29.44° C. (85° F.) to 32.22° C. (90° F.). It was carefully nursed by a night nurse and a day nurse.

It weighed 2490 grammes (about 5 pounds 3 ounces). It was under the care of Dr. Edward Reynolds, with whom I saw it in consultation. Its food was carefully regulated at the Milk-Laboratory, and the first prescription which was written for it, and which proved to be adapted to its digestion during the first week or ten days, was this one (Prescription 34):

PRESCRIPTION 34.

℞ Fat	1.00
Sugar	3.00
Proteids	1.00
Lime water	5.00

The mixture to be heated for twenty minutes at 68.38° C. (155° F.).

From my later experience with these cases, I should begin with the percentage of proteids 0.50, as I have already described in this prescription (Prescription 30, p. 300). In the early days of this infant's life oxygen had to be administered to it for two or three minutes every hour. It was fed every hour, and received six drops of brandy with each feeding. At my first examination, which was made when it was six hours old, a distinct cardiac murmur was heard at the base of the sternum, and there were a few fine moist râles throughout both lungs. The murmur and the râles disappeared in the course of a week, and the infant, after losing 135 grammes (about 4½ ounces) in the first three days, began to make small gains in weight, and when it was seven weeks old it weighed 2730 grammes (about 5 pounds 11 ounces), was plump, had a healthy color, and seemed very well. It began to perspire when it was seven weeks old.

This case received the very closest attention, and was treated with all the details for safety which were possible to be attained without the use of an incubator, but we must consider that its weight, 2490 grammes (about 5 pounds 3 ounces), and its age, thirty-were such as to make the preservation of its life a much more simple matter the infant (Case 106) whose picture I have just shown you, and whose light ed towards so undeveloped and premature a condition that any omission in re- losest detail of treatment would have been likely to prove fatal.

nt had progressed so far in its general condition and development that at the weeks it was taken out of the cotton in which up to that time it had been was dressed. At this time it was taking 56 c.c. (1¾ ounces) at each meal, and in two hours.

t case (Case 108) is that of an infant which was four weeks premature, and r a premature infant, tolerably vigorous at birth. It was under the care of Breck, with whom I saw it in consultation. It was not placed in an incubator. y, its nurse had no idea of the importance of protecting it from external influ- s fed on a carefully prepared food from the Milk-Laboratory, and began to t, and in every way showed no evidence of its vitality being interfered with; was possessed with the idea that it needed plenty of cold fresh air. The e infant's room was left open one night when the weather was quite cool. g day it did not take its food well, was somewhat cyanotic, and was found to ost 240 grammes (½ pound). It was then placed, as it should have been in g, in a warm room, treated with the utmost care, and not handled much. None ures, however, were sufficient to prevent a still further lessening of its vitality. ed from the first blow which was struck at its vitality, and lost its life practi- the ignorance of the nurse who was in charge of it.

nortem examination showed nothing abnormal, except that the mesenteric omewhat enlarged.

case (Case 109) was that of an infant born at about the twenty-fifth week of life. Its weight was 1080 grammes (about 2¼ pounds). There are a number points to be recorded in this case.

ot strong enough to suck, and had to be fed with a spoon. Its mother's milk, Analysis 55) of which I have here to show you, at once caused such disturb- dified milk from the Laboratory had to be substituted.

ANALYSIS 55.

	1.29
	4.10
ls	6.83
	0.26
olids	12.28
	87.72
	100.00

e prescription for the modified milk which it digested well:

PRESCRIPTION 35.

Modified Milk.

	1.00
	3.00
ds	0.75

nt's temperature in the rectum was 36.7° C. (98° F.). It seemed to be doing ut did not gain in weight, and on the fifth day of its life was unable to swal- then fed by gavage.

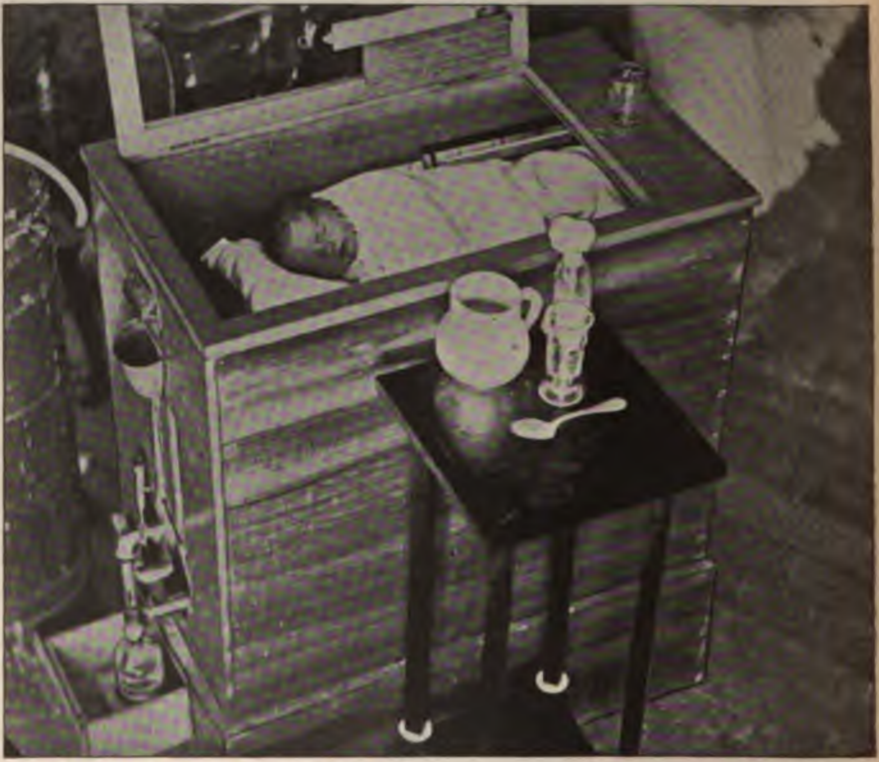
reated with great care so far as keeping it warm was concerned, but an incu- ot be obtained for it, and it died when it was seven days old.

resting in this case to notice that the meconium came as is usual in the infant began to change its color on the third day, and that by the fifth day the fæcal were yellow and well digested.

These particulars were given to me by Dr. Woods, who was in charge of the case. Its death was evidently due to the lowering of its vitality consequent upon its age and lack of sufficient development to withstand the influences surrounding it in extra-uterine life.

I now wish you to examine this incubator (Fig. 80), which was devised by Dr. Worcester, of Waltham, Massachusetts. It is far superior in its mechanism and in its general utility to the other incubators which I have already referred to, except that of Tarnier, which it closely resembles. It is practically a wooden box, 76 cm. (2½ feet) long, 45.5 cm. (1½ feet) wide, and 76 cm. (2½ feet) high. This box, as you see, has a glass lid, which can be raised when necessary, but which is intended to be kept closed and to be used as a window through which to observe the infant. Two or three holes at the end and at the bottom of the box allow the entrance of air. A hole at the top and end of the box, fitted with an anemometer, serves as an exit for the air. The continuous motion of the anemometer shows that the ventilation is being carried on properly. At the bottom of the box is a metallic boiler. A pipe from this boiler is brought through the end of the box, turns upward for a few inches, and then turns back and enters the box, where it connects again with the boiler. Outside of the end

FIG. 80.



To left of incubator is the oxygen tank. To left of incubator on the floor is the lamp. At upper right end of incubator is an anemometer.

Showing Details of Sixty-Four Days of Li

DAYS OF LIFE.	INTERVALS BETWEEN MEALS.	AMOUNT AT EACH MEAL.		PERCENTAGES OF FOOD.				FÆCAL DISCHARGES.			W	
				Fat.	Sugar.	Proteids.	Lime Water.	No.	Character.	W'ght.		
		C.c.	Dr'ms.							Oz.	Gr'ms.	Lbs.
1	. . . . . .	. . .	. . . .	. . .	3 per ct. sol. in aq. dis.	. . . . .	. . . .	. .	meconium.	. . . .	2040	4
2	1 hour.	4	1	1.00	3.00	1.00	5.00	2	brown, small.	. . . .	2040	4
3	1 hour.	4	1	1.00	3.00	1.00	5.00	2	"	. . . .	2040	4
4	1 hour.	4	1	1.00	3.00	1.00	5.00	2	"	. . . .	2040	4
5	1 hour.	4	1	1.00	3.00	1.00	5.00	2	only fairly digested.	. . . .	2010	4
6	1 hour.	4	1	1.00	3.00	1.00	5.00	2	"	. . . .	2040	4
7	1 hour.	4	1	1.00	3.00	1.00	5.00	2	"	. . . .	2040	4
8	1 hour.	4	1	1.00	3.00	1.00	5.00	2	"	. . . .	2055	4
9	1 hour.	4	1	1.00	4.00	1.00	5.00	2	yellow.	. . . .	2010	4
10	1 hour.	4	1	1.00	4.00	1.00	5.00	2	well digested.	. . . .	2025	4
11	1 hour.	8	2	1.00	4.00	1.00	5.00	2	"	. . . .	2025	4
12	1 hour.	8	2	1.00	4.00	1.00	5.00	2	"	. . . .	2055	4
13	1 hour.	8	2	1.00	4.00	1.00	5.00	2	"	. . . .	2070	4
14	1 hour.	8-10	2-2½	1.00	4.00	1.00	5.00	2	"	. . . .	2070	4
15	1 hour.	8-10	2-2½	1.00	4.00	1.00	5.00	2	"	. . . .	2070	4
16	1 hour.	12	3	1.50	5.00	1.00	5.00	2	"	. . . .	2160	4
17	1 hour.	12	3	1.50	5.00	1.00	5.00	2	"	. . . .	2160	4
18	1 hour.	12	3	1.50	5.00	1.00	5 00	2	"	. . . .	2100	4
19	1 hour.	12	3	1.50	5.00	1.00	5.00	2	"	. . . .	2130	4
20	1 hour.	12-14	3-3½	1.50	5.00	1.00	5.00	2	"	. . . .	2160	4
21	1 hour.	16	4	1.50	5.00	1.00	5.00	2	"	. . . .	2175	4
22	1 hour.	16	4	1.50	5.00	1.00	5.00	2	"	. . . .	2220	4
23	1 hour.	16-18	4-4½	1.50	5.00	1.00	5.00	2	"	. . . .	2235	4
24	1 hour.	16-18	4-4½	1.50	5.00	1.00	5.00	2	"	. . . .	2250	4
25	1 hour.	18-20	4½-5	2.00	5.00	1.00	5.00	1	"	. . . .	2280	4
26	1 hour.	18-20	4½-5	2.00	5.00	1.00	5.00	2	"	. . . .	2280	4
27	1 hour.	18-20	4½-5	2.00	5.00	1.00	5.00	1	"	. . . .	2295	4
28	1 hour.	18-20	4½-5	2.00	5.00	1.00	5.00	2	"	. . . .	2310	4
29	1 hour.	20	5	2.00	5.00	1.00	5.00	2	"	. . . .	2310	4
30	1 hour.	22	5½	2.00	5.00	1.00	5.00	3	slightly green.	. . . .	2290	4
31	1 hour.	22	5½	2.00	5.00	1.00	5.00	4	"	. . . .	2287	4
32	1 hour.	22	5½	2.00	5.00	1.00	5.00	4	"	. . . .	2295	4
33	1 hour.	24-26	6-6½	2.00	5.00	1.00	5.00	5	"	. . . .	2295	4
34	1 hour.	28	7	2.00	6 00	1.00	10.00	8	"	. . . .	2295	4
35	1¼ hrs.	32	8	2.00	6.00	1.00	10.00	5	yellow and well digested.	. . . .	2340	4
36	1¼ hrs.	32	8	2.00	6.00	1.00	5.00	5	"	. . . .	2400	5
37	1¼ hrs.	32	8	2.00	6.00	1.00	5.00	3	"	. . . .	2460	5
38	1¼ hrs.	48	12	2.00	6.00	1.00	5.00	3	"	. . . .	2490	5
39	1¼ hrs.	48	12	2.00	6.00	1.00	5.00	3	"	. . . .	2520	5
40	1¼ hrs.	48	12	2.00	6.00	1.00	5.00	5	"	3	2550	5
41	1½ hrs.	48	12	2.00	6.00	1.00	5.00	2	"	2½	2550	5
42	1½ hrs.	48	12	2.00	6.00	1.00	5.00	2	"	2½	2550	5
43	1½ hrs.	48	12	2.00	6.00	1.00	5.00	1	"	. . . .	2640	5
44	1½ hrs.	48	12	2.00	6.00	1.00	5.00	2	"	2½	2700	5
45	1½ hrs.	48	12	2.00	6.00	1.00	5.00	5	"	8	2700	5
46	1½ hrs.	48	12	2.00	6.00	1.00	5.00	6	"	9	2640	5
47	1½ hrs.	48	12	2.00	6.00	1.00	5.00	6	"	9	2640	5
48	1½ hrs.	48	12	2.00	6.00	1.00	5.00	4	"	. . . .	2730	5
49	1½ hrs.	48	12	2.00	6.00	1.00	5.00	4	"	. . . .	2790	5
50	1½ hrs.	56	14	2.00	6.00	1.00	5.00	6	"	6	2850	5
51	1½ hrs.	56	14	2.00	6.00	1.00	5.00	5	"	7	2850	5
52	1¾ hrs.	64	16	2.00	6.00	1.00	5.00	6	"	5½	2880	6
53	1¾ hrs.	64	16	2.00	6.00	1.00	5.00	5	"	. . . .	2880	6
54	1¾ hrs.	64	16	2.00	6.00	1.00	5.00	2	"	3	2970	6
55	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	1	"	3	2970	6
56	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	1	"	2	3030	6
57	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	3	"	5	3030	6
58	1¾ hrs.	64	16	3.00	7 00	1.00	5.00	1	"	. . . .	3090	6
59	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	3	"	. . . .	3120	6
60	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	1	"	. . . .	3210	6
61	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	3	"	. . . .	3150	6
62	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	5	"	. . . .	3240	6
63	1¾-2 hrs.	64	16	3.00	7.00	1.00	5.00	2	"	. . . .	3240	6
64	1¾-2 hrs.	64	16	3.00	7.00	1.00	5.00	2	"	. . . .	3270	6
65	1¾-2 hrs.	64	16	3.00	7.00	1.00	5.00	2	"	. . . .	3270	6
68	1¾-2 hrs.	64	16	4.00	7.00	1.00	5.00	2	"	. . . .	3300	6

At six months weighed 7080 grammes (14 pounds 12 ounces) and was taking 150 c *se 110, Premature at Thirty Weeks.*

ULSE.	RESP.	TEMPERATURE OF INCUBATOR.		REMARKS.
		°C.	°F.	
135	60	32.2	90	Nails formed. No lanugo. Heart normal. Lungs normal. Emaciated. Cry feeble.
. . .	. . . .	32.2	90	Uric acid on napkin. Food heated to 75° C. (167° F.).
. . .	. . . .	32.2	90	Somnolent.
. . .	. . . .	32.2	90	No uric acid. Fed with dropper.
. . .	50-60	32.2	90	Temperature went up in evening. Perspired freely. Temperature of incubator lowered to 28.3° C. (85° F.). Hiccough relieved by brandy.
. . .	. . . .	28.3	83	Respirations irregular; 10 quick and then imperceptible for 10 seconds. Cord fell. Somnolent.
. . .	. . . .	26.6	80	Cry a little stronger. As still perspiring a little, temperature of incubator reduced to 26.6° C. (80° F.).
. . .	. . . .	26.6	80	Slight ophthalmia neonatorum. Icterus neonatorum. Black cloth over lid of incubator.
. . .	. . . .	26.6	80	Somnolent.
. . .	. . . .	26.6	80	Less icterus.
120	60	26.6	80	Hiccough.
120	60	26.6	80	Hands and feet cold.
. . .	. . . .	26.6	80	Hands and feet warmer. Oxygen in fresh-air-box for 10 minutes three times daily. Seems hungry.
. . .	. . . .	28.8	84	Every other feeding takes 2⅖ drs. Oxygen as on 13th.
. . .	. . . .	28.8	84	Oxygen as on 13th.
120	60	29.4	85	Oxygen 5 minutes twice daily. Feet cold when incubator below 29.4° C. (85° F.).
. . .	. . . .	29.4	85	
. . .	. . . .	29.4	85	Less icterus.
. . .	. . . .	29.4	85	Oxygen.
. . .	. . . .	29.4	85	
. . .	. . . .	29.4	85	Very hungry. Oxygen.
. . .	. . . .	29.4	85	
. . .	. . . .	29.4	85	Oxygen.
. . .	. . . .	29.4	85	Occasional cyanosis.
. . .	. . . .	29.4	85	
. . .	. . . .	29.4	85	
. . .	. . . .	29.4	85	
. . .	. . . .	29.4	85	Oxygen.
. . .	. . . .	29.4	85	
. . .	. . .	29.4	85	
. . .	. . . .	29.4	85	
. . .	. . . .	29.4	85	Brandy 5 drops every 2 hours.
. . .	. . . .	28.3	83	Feet and hands not cold except when temperature of incubator as low as 26.6° C. (80° F.). Seems hungry.
. . .	. . . .	28.3	83	Oxygen. Brandy 5 drops three times daily.
. . .	. . . .	28.3	83	Brandy 5 drops three times daily. Oxygen. Began to feed with nipple.
. . .	. . .	26.6	80	Respirations deeper and more regular. Slight cyanosis. Oxygen.
140	40	29.4	85	
. . .	. . . .	29.4	85	
. . .	. . . .	26.6	80	Oxygen. Brandy 5 drops.
. . .	. . . .	26.6	80	
. . .	. . . .	26.6	80	Oxygen. Brandy 5 drops every other feeding.
. . .	. . . .	26.6	80	
. . .	. . . .	26.6	80	
138	48	25.5	78	Allowed to have a little light in incubator. Omit oxygen.
. . .	23	25.5	78	
. . .	. . . .	23.8	75	Oxygen. Brandy. Somnolent.
. . .	. . . .	23.8	75	No light.
. . .	. . . .	23.8	75	Is brighter. Oxygen.
. . .	. . . .	23.8	75	
. . .	. . . .	23.8	75	Oxygen. Brandy.
. . .	. . . .	23.8	75	Seems stronger. More light.
. . .	. . . .	23.8	75	Oxygen.
. . .	. . . .	23.8	75	Brandy.
. . .	. . . .	23.8	75	Seems hungry.
. . .	. . . .	23.8	75	Oxygen.
. . .	. . . .	22.2	72	Omit oxygen.
. . .	. . . .	22.2	72	Sleeps and takes food well. Seems stronger and brighter, and is tranquil. Does not cry.
. . .	. . . .	22.2	72	
. . .	. . . .	22.2	72	
. . .	. . . .	22.2	72	Very bright and tranquil.
. . .	. . . .	21.1	70	
. . .	. . . .	21.1	70	Taken out of incubator. Cried 9 hours. Vomited. Put back into incubator.
. . .	. . . .	21.1	70	
. . .	. . . .	21.1	70	Tranquil. Does not cry.
. . .	. . . .	. . . .	. . . .	Taken out of incubator and washed in water at 35° C. (95° F.).
. . .	. . . .	. . . .	. . . .	Sleeps well. Does not cry. Is growing stronger.
. . .	. . . .	. . . .	. . . .	Thriving. Brandy omitted.

Looked bright, had a good color, and was well developed and vigorous.

that when the source of heat is outside of the incubator there is a
t the free flame may set fire to the nurse's dress.

the boiler is a shelf, on which the infant's bed is placed, sufficient
g left between the ends of the bed and the box for a free circula-
e contained air.

rmometer is attached to the water apparatus of the boiler, and
he heat of the water.

mometer is attached to the lid of the box, and is intended to show
rature of the air in the box.

e here a picture (Fig. 80) of an infant (Case 110, page 306) in
ator, prematurely born at about the thirtieth week of intra-
e.

of the incubator is open, representing a time when the infant is to be fed. On
e incubator part of the oxygen tank is shown. On a table beside the incubator
suring glasses, a glass tube with a cotton stopper containing the infant's food,
repared at the Milk-Laboratory, a pitcher of warm water to keep the food warm,
poon with which the infant was fed. In the bed beside the infant you will see
another thermometer, which it was found necessary to use, as the thermometer
the lid was subject to such variations in temperature through the glass, accord-
ariations of the temperature in the room, that it did not indicate exactly the
of the air by which the infant was surrounded. In the treatment of this
e incubator much difficulty arose in keeping the ventilation perfect, and at
ir for hours had to be forced through the air-box by fanning the air through the
rance.

fant was taken care of in an unusually exact way, and with such intelligence
of the nurses and parents that the details of its life in the incubator become
value in our study of the treatment of this class of cases. I shall therefore
details of its existence in the incubator from the time when it was born until
iently developed to be safely taken care of in the ordinary way.

fant and its mother were under the care of Dr. George Haven and Dr. W. L.
, with whom I saw it in consultation in the early hours of its life and by whom
ed in my charge. At birth its nails were fairly developed. Its face was not
wrinkled, but its body and limbs did not show much evidence of subcutaneous

any one who has charge of a premature infant in an incubator, as it illustrates exactly what emergencies are likely to arise and how they can be me

The infant, as is seen by referring to the column of remarks, cam very near dying a number of times, and unquestionably would have die had it not been carefully managed, as, for example, by the administratio of oxygen, by prompt changes in its food, by the regulation of the temperature of the incubator, and by the constant attention of a day nurse and a night nurse.

I have now in a general way told you the main facts which are know about premature infants, and the results of my experience with this class o cases. The last case (Case 110) which I have described as being treated i Dr. Worcester's incubator was the one from which I learned how very inadequate are our usual methods of treating premature infants. In th direction of this case I received so much information as to the mechanica management of the many difficulties which were continually presenting themselves in the daily care of the apparatus from Mr. J. P. Putnam, that i was at once impressed upon me that a domicile in which an infant migh have to live for several months should be devised and regulated as to it ventilation and general practical usefulness even more carefully than th houses in which adults live. This meant that such apparatus needed th skilled attention of an expert in building and in ventilation. I therefor placed in Mr. Putnam's hands the construction of what I prefer to call *brooder*. I am also indebted for many valuable suggestions as to the construction and use of the brooder to Mr. G. E. Gordon, who has had considerable experience in preserving the lives of premature calves.

Before inspecting the brooder more closely I should like you to examin this table (Table 83), in which I have condensed what I have already tol you concerning the requirements needed to preserve the lives of prematu infants.

TABLE 83.

Indications for conserving the Viability of Premature Infants.

I. There should be a receptacle which shall guard the infant from the deleterio influences of extra-uterine life.

II. There should be an apparatus that can be obtained quickly and transported rapidl and which therefore should be kept at some central and convenient station.

III. The place where the brooder is kept should be free from the influence of an disease.

IV. The brooder should be so constructed as to make it possible for it to be absolutel cleansed and disinfected each time after it has been used, hence it should be made of meta

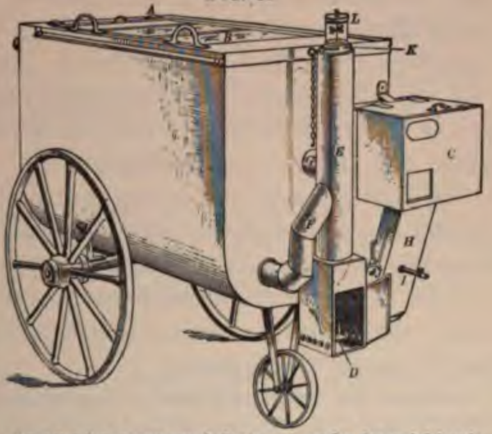
V. The brooder should, as soon as the infant is placed in it, be under the observatio of trained nurses night and day.

VI. The food for the infant should be regulated with the greatest precision, with th closest attention to minute details, and, if possible, at a milk-laboratory.

These are the principal rules which should be attended to where th physicians of any community wish to provide the best means for preservin the lives of the premature infants in that community. The expense of suc means, while too great for any one individual, is comparatively insignificant for a number. The brooder at present must necessarily be an expensive machine, but if provision should be made for it in combination with such scientific facilities for infant feeding as I have already recommended, I believe that any community would find it of infinite benefit. I am also sure that there would result saving of life for the people, and saving of time and expense for the physicians, combined with the greatest satisfaction to both people and physicians. Such a combination, in cities of a milk-laboratory or in the country of a Babcock milk-tester with a brooder kept in one central station, I hope to see established everywhere. One such station for districts which might be included in a radius of ten or even of twenty miles would be amply sufficient to accomplish very favorable results.

BROODER.—You will now, I hope, appreciate that it is often quite necessary to provide not merely a receptacle but an actual habitation for premature infants during a period of months. Such a habitation, which I prefer to call a *brooder* in order to represent it by the name which explains it rightly, I have here to show you (Fig. 81).

FIG. 81



Brooder for premature infants. *A*, scales for weighing infant; *B*, glass lid of incubator; *C*, fresh-air box, containing clock-work and fan; *D*, lamp for heating water-jacket; *E*, chimney; *F*, return flue from heating-flues; *G*, return fresh-air flue; *H*, entrance for fresh air; *I*, connection for oxygen tank; *J*, mixing-valve; *K*, ventilating exit; *L*, anemometer.

This apparatus has been made to fulfil the conditions of a house for the premature infant, and it practically meets the indications called for in this table (Table 83, p. 308). After being used, it can be completely disinfected and cleansed. It is kept at the Milk-Laboratory, whence it can be obtained at a moment's notice. For purposes of disinfection, and that it may not absorb micro-organisms or dirt of any kind, which in wooden receptacles invariably cause a decided odor, it is made entirely of metal.

The brooder is supported, as you see, on three wheels, preferably made of light steel, two behind and one guiding wheel in front. A handle is used to push it to different parts of the room, or, if necessary, to an adjoining room, so that the mother can see her infant if she is too sick to leave her bed. The top of the brooder is about 91 cm. (3 feet) from the floor, so that the nurse does not have to stoop unnecessarily, but at the same time can, when sitting down, see into it from above. It is 76 cm. (2½ feet) wide and 91 cm. (3 feet) long. The body is made of copper; the walls are double, and insulated on the outside, to prevent radiation. The water used for heating circulates on all sides, and the infant is thus warmed by direct radiation. The top of the brooder is covered in the middle by a thick plate-glass lid, which can be raised sufficiently to allow the hands and arms of the nurse to be freely used in the brooder, and is by a simple contrivance kept from falling down while the infant is being fed or touched. A chain prevents the lid from falling backward. On the under side of the glass lid is a fine wire sliding screen, which comes directly over the infant's head and between it and the glass. This is simply a precaution against the possible breakage of the glass lid and consequent injury to the infant.

This plated box (*C*), which you see attached to the upper front end of the brooder contains some strong clock-work with a fan attachment. This oval opening in the clock-box admits the air to the brooder. Below the opening for the fresh air is a window, through which the fan and clock-work can be watched.

Just below the air-opening and above the clock-work is a fine open wire shelf, on which is spread a thin layer of cotton-wool. The air, which by means of the fan is drawn into the box, is sifted through the cotton and carried down the air-shaft (*H*) directly into the brooder. In this air-shaft (*H*) you see there is a small stop-cock (*I*). This is the point of attachment for the tube from the oxygen tank, to be used when oxygen is needed to be mixed with the entering air-supply.

In this air-shaft, also, is attached a valve, which is so regulated by a register handle that the air can be utilized either above or below the boiler, according as it is needed and as I shall explain later.

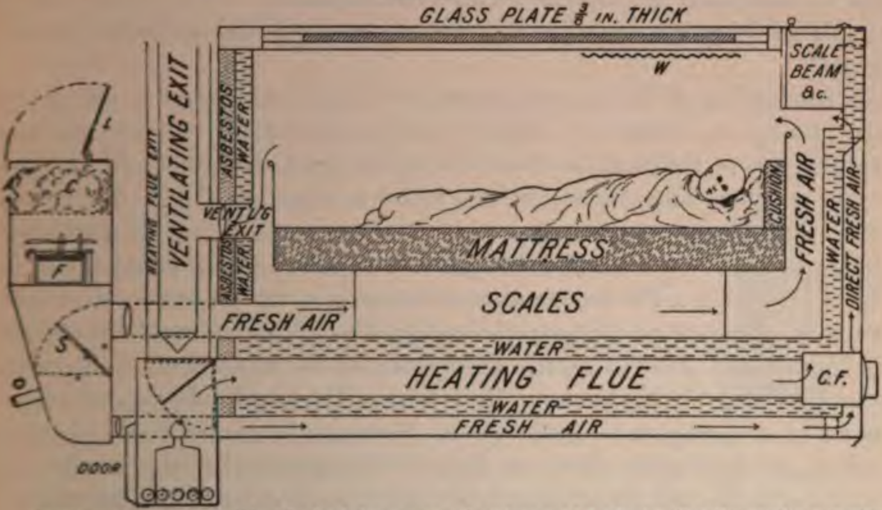
The bottom of the brooder constitutes an air-chamber, and in this is a boiler which, with its heating or combustion direct and return flues, warms the interior of the apparatus.

Above the boiler is placed the platform of a scale. The balance power of this scale is on the top of the back end of the brooder. The platform of the scales acts as the support for a metal pan 61 cm. (2 feet) long and 30.5 cm. (1 foot) wide, on which the infant is placed. This pan should be made of sheet iron, enamelled on both sides with white porcelain enamel, and should have handles at either end to facilitate its removal from the brooder. From the ends of this pan is hung by wires, which can be easily attached or detached, a light frame made of four steel rods crossed. On this frame is tied with tapes a piece of strong cotton cloth. This cloth is the infant's bed, on which it is placed wrapped in clean absorbent cotton. This cotton cloth is about 2.5 cm. (1 inch) above the bottom of the pan. The infant's head is turned to the back end of the brooder.

At the front end, opposite the foot of the infant's bed, is the exit (*G*) for the vitiated air. This exit passes through the end of the brooder and enters a ventilating pipe which has at its top an anemometer (*L*). The bottom of the shaft is outside the brooder, and has a closed cone-shaped end, which is enclosed in a metal box in such a way that a lamp (*D*) can be placed under it. The heat from this lamp answers two purposes. One is by keeping the ventilating shaft hot, to aid the ventilation, and the other is to heat the water in the boiler. A register-valve (*J*) attached to the pipe can shut off the heat if necessary from the boiler, and allow it to go directly up the double pipe (*E*, *K*), whereby its entire power will be used in promoting ventilation, or the valve may be set so as to direct the flame partially into the boiler, thus placing its temperature completely under control. In this way the heat from the lamp (which is enclosed in the box) is without danger entirely utilized for heating and ventilation.

I have now shown you the brooder and its general features. I shall still further explain to you its mechanism by means of this sectional diagram (Diagram 6).

DIAGRAM 6.



Section of brooder. *L*, lid of fresh-air box, open; *A*, entrance of fresh air; *C*, cotton, resting on wire shelf above clock-work; *F*, clock-work and fan; *S*, valve regulating hot and cold fresh air; *O*, pipe for oxygen attachment; *C. F.*, cleaning-flue; *Door*, door to lamp-box; *W*, wire frame to protect against breakage of lid.

The smoke-flue of the lamp, marked "Heating Flue," passes through the centre of the boiler, marked "Water" in the diagram, as far as to the cleaning-flue, marked *C. F.* Thence it returns and enters the upright pipe marked "Heating Flue Exit." The horizontal return-flue is not shown in the diagram, because it is behind the horizontal arm shown. The little gate-valve shown directly above the lamp regulates absolutely the amount of heat which is allowed to pass through the boiler, and the temperature of the warm water therein may be tested by a chemist's thermometer, inserted at any opening which may be provided for it as directed when the brooder is built.

The fresh-air flues are constructed, as shown, one above and one below the boiler. One flue comes in contact with the upper or hottest part of the boiler, and presents a very large surface of contact therewith by being flattened so as to cover completely the upper side of the boiler. The other flue touches the bottom of the boiler only in one line, or not at all, so that the air passing through it is practically unaffected by the boiler heat. By this arrangement the temperature of the fresh air can be regulated at will by the attendant by simply raising or lowering the valve *S*.

In virtue of the large amount of heating surface of the heating flue in this apparatus, it is found that a very small flame suffices to keep up the desired temperature, and it results from this that no injurious products of combustion contaminate the air of the room. A very small alcohol lamp can be used, while with a less scientific arrangement this fuel might be found too expensive.

It is probable that an electric current will be found most suitable to supply the heat in place of the lamp, as well as to drive the fan, and this can be very easily accomplished with a small battery.

By packing the water-jacket with asbestos, external radiation is prevented.

The heating of the brooder varies as to time and degree according to the atmosphere of the room where it has been standing. If, however, the temperature of the room is 21.1° C. (70° F.), and the temperature of the water which is introduced into the boiler is about 40.5° C. (105° F.), it will be found that after the cool air in the brooder has been displaced the temperature of the air in the brooder will in about fifteen minutes rise to 35° C. (95° F.). The temperature will remain at this point for about half an hour. As soon as the temperature begins to fall the alcohol lamp should be lighted, and as soon as the temperature of the water in the boiler rises above 35° C. (95° F.) the lamp should be extinguished. By careful regulation of the lamp and regulating the fresh air by means of the register-valves, an intelligent nurse can keep the temperature of the brooder at whatever degree the physician orders. The thermometer should, in order to show accurately the temperature of the air which the infant is breathing, be beside it on its bed, as when attached to the lid it is influenced by changes of temperature in the room.

If any difficulty arises from the temperature not responding quickly enough to the register-valves and lamp, it is well to draw off a little hot water and replace it by some cold water if it is desired to lower the temperature, while to raise the temperature the withdrawn water is to be replaced by hot water.

NURSES.—The brooder is not intended to obviate the necessity of skilled nursing. On the contrary, a nurse should be in constant attendance night and day. She should have all the details of the infant's care and the mechanism of the brooder explained to her minutely, for an emergency may arise at any time, and always requires to be dealt with immediately.

The brooder supplies the means for exact treatment, but intelligent minds and trained gentle hands are indispensable. The nurse should frequently observe the infant through the glass lid, and should be certain that the anemometer is in constant motion.

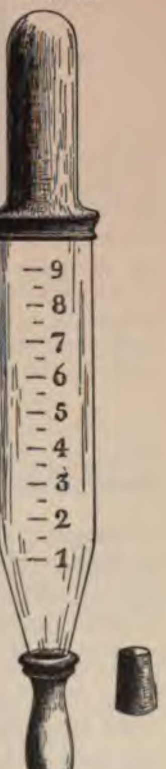
APPARATUS CONNECTED WITH THE BROODER.—A stethoscope like this one (Fig. 85, p. 323) is the best adapted for examining the infant in the brooder. It can be bent in any direction, and the small calibre of its cup is best adapted to the infant's size.

A piece of dark cloth should be kept over the glass lid, to preclude the light, while the sun should be allowed to shine freely into the room.

The method of feeding the infant in the brooder is important. It frequently happens that the premature infant is too weak not only to suck the breast, but also to be fed from the bottle. In such cases it is customary to use a spoon or a medicine-dropper. These, however, are very unsatisfactory instruments. The food is liable to be spilled, the spoon or dropper has to be frequently filled, and much time is taken to complete the feeding. The lid of the brooder, also, should not be kept open for a longer time than is unavoidable.

I have lately made use of a device suggested by Dr. Breck, who first brought it to my notice when I was seeing a premature infant with him in consultation where there was much difficulty in getting the infant to swallow, and where it would not suck.

FIG. 82.



Feeder for premature infants (reduced one-half).

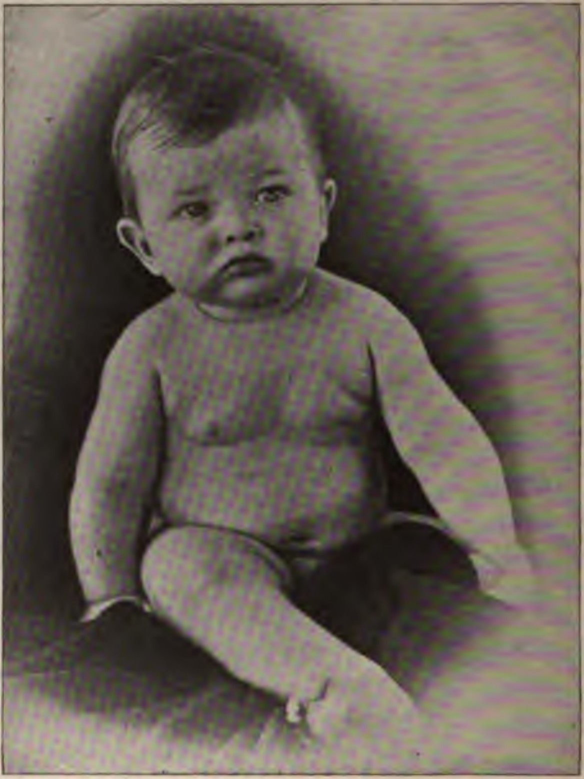
It is simply this glass cylinder (Fig. 82), 12 cm. ($4\frac{3}{4}$ inches) long and 2.4 cm. (1 inch) in diameter. The cylinder is graduated to 2 c.c. ($\frac{1}{2}$ drachm), and holds 36 c.c. (9 drachms). It is shaped at one end so as to have a small rubber nipple fitted to it. The large end is covered by a rubber cot. The rubber cot, which has no holes, acts as an air-reservoir, and by simply introducing the small perforated nipple into the mouth and gently pressing the rubber cot the food is slowly forced down the infant's throat, without choking it and without the infant having to suck or apparently to use any effort. To fill the tube the rubber nipple and cot are removed, a rubber stopper like this (Fig. 82) plugs the small end of the cylinder, and the required amount of food is poured in at the large end.

This method of feeding is especially desirable for a weak premature infant in a brooder, because it entails no loss of strength on the part of the infant, and can be easily managed by the right hand of the nurse while her left hand supports the infant's head. This method is far preferable to that of gavage, which is not so easily managed by the nurse and causes more exhaustion to the infant.

The question is often asked whether premature infants, even if their lives are saved, can be as well developed physically and mentally as are those born at term. In my experience, there seems to be no question that when once we have succeeded in making the infant gain steadily in weight and assume the appearance of an infant at term its subsequent condition differs in no respect from that of infants born at term.

I have here to show you a picture of the infant (Case 110, p. 307) which was treated in Dr. Worcester's incubator.

FIG. 83.



Infant premature at thirty weeks. Birth-weight, 2040 grammes. Treated in incubator eight we Present age, nine months. Present weight, 8400 grammes.

This picture was taken when the infant was nine months old, and it weighed at t time 8400 grammes (17½ pounds). As its birth-weight was 2040 grammes (about pounds), you see that it has quadrupled its weight. It was fed entirely on modified m from the Laboratory during the first year, and is now a fine large boy, walking and talki at two years of age. It is perfectly healthy and well developed both physically and m tally.

His sister, who was premature at the twenty-eighth week, is now eight years old. S is well developed and strong, and is unusually bright and intelligent for her age. She is fact, decidedly in advance mentally of the other children of her age at her school.

I have here the record of an infant prematurely born at about t thirtieth week, and weighing 2850 grammes (about 5 pounds 15 ounc which was the first premature infant that happened to be treated in t brooder (Fig. 83).

his infant (Case 111) was born at ten minutes past three on February 16. It was
in the brooder at 9 P.M. of the same day, the temperature of the brooder being
C. (94° F.).

n the following day, February 17, the infant was given by the nurse 4 c.c. (1
m) of diluted cow's milk every hour for three feedings, which he vomited almost im-
tely after taking. The intervals of feeding were then increased to two hours, but
ilk was not retained. The nurse then gave him 2 c.c. (½ drachm) every three hours
g the night, which he retained for a number of feedings, but then vomited bile
ucus, together with the undigested food which had been given him.

ebruary 18 the infant was found to have lost 420 grammes (14 ounces) in weight, to
y weak, and to be unable to retain the milk diluted with water. The meconium came
on this day, and there was a uric acid stain on the napkins. The infant was very
s. Its respirations were irregular, and its feet and hands were cold. The tempera-
f the brooder, which up to this time had been kept at 34.4° C. (94° F.), was lowered to
C. (93° F.), as the infant had begun to perspire. A substitute food was ordered from
ilk-Laboratory on this day, the prescription for which was as follows (Prescription 36):

PRESCRIPTION 36.

Fat	1.00
Sugar	3.00
Proteids	0.50
To be heated for thirty minutes at 75° C. (167° F.).	
Lime water	5.00
24 tubes, each containing 4 c.c. (1 drachm).	

This food was given to the infant every hour.

On the following day, the 19th, the record was that the food had been retained, that
nfant had seemed so hungry that the amount had to be increased to 10 c.c. (2½
ms), and that it was found advisable to feed it every two hours rather than every
There was no vomiting. There were two movements of the bowels, which still
d evidences of undigested milk and some meconium. The infant's weight on this
as found to be the same as on the previous day, 2300 grammes (5 pounds 1 ounce).

n the following day, February 20, the infant was found to have gained 30 grammes
ce). It was taking its food regularly every two hours, alternating with the mother's
which had come in considerable quantity. There were still evidences of uric acid in
ne. The temperature of the brooder was kept at 31.6° C. (89° F.).

n the following day, February 21, the weight was as on the previous day, 2230
nes (5 pounds 2 ounces). The color of the fæcal discharges was yellowish brown.
was only one discharge in the twenty-four hours, obtained by the use of a supposi-
The temperature of the brooder was kept at 30° C. (86° F.).

n the following day, February 22, it was found that the infant had lost 60 grammes
nces). The substitute food was then given every two hours, alternating with the
-milk. On that day there were three yellow well-digested movements. The tem-
re of the brooder was kept at 29.4° C. (85° F.). The infant seemed stronger, was
uiet, and slept except when it awoke to receive its food.

n the following day, February 23, there is no record of the infant's weight, but it
vidently in a very precarious condition and seemed exhausted. It did not take its
hment readily. It had five small fæcal discharges in the twenty-four hours, which,
er, were yellow and fairly digested.

n the following day, February 24, the breast-milk was omitted, and 4 c.c. (1 drachm)
dified milk were given every two hours, the percentage of the sugar being raised from
5. There were four small fæcal movements during the day; the first one was green,
t three were yellow and decidedly better digested. The temperature of the brooder
ept at 29.4° C. (85° F.). During the day the infant gained 60 grammes (2 ounces)
ight. It was so weak on these two days that it would have been dangerous to
t out of the brooder to weigh it, so that the continual record of the weight which

could be obtained by the scale-bed of the brooder was of the utmost value in regulating the changes in the food necessary to save the infant's life.

On the following day, February 25, the infant's weight was found to be 2260 grammes (5 pounds 3 ounces), an increase of 30 grammes (1 ounce). The percentages in the modified milk were then changed to the following (Prescription 37):

PRESCRIPTION 37.

℞ Fat	1.50
Sugar	4.00
Proteids	0.75

One drop of brandy was given with each feeding. There was one fæcal discharge, which was yellow and well digested. On this day 4 c.c. (1 drachm) of food were given to the infant every two hours until its feeding at 10.30 P.M. After this it seemed so hungry that at midnight 36 c.c. (9 drachms) were given, at 3 A.M. 40 c.c. (10 drachms) were given, and at 5.30 A.M. 30 grammes (1 ounce) were given. The weight was now found to be 2420 grammes (5 pounds 5 ounces), an increase of 60 grammes (2 ounces) in the twenty-four hours. The amount of food which the infant had taken in the previous twenty-four hours was found to have been 375 grammes (12½ ounces). The fæcal discharges were yellow and well digested. Brandy was continued to be given. The temperature of the brooder was kept at 29.4° C. (85° F.). At times a little breast-milk was given to the infan in order to satisfy the mother, but it evidently did not agree with it.

On February 27 the weight was found to be 2450 grammes (5 pounds 6 ounces). Th prescription for the modified milk was then changed as follows (Prescription 38):

PRESCRIPTION 38.

℞ Fat	2.00
Sugar	5.00
Proteids	0.75

30 grammes (1 ounce) of this were given to the infant every two hours during th day, and every two and one-half hours during the night. One yellow well-digested fæca discharge was obtained by means of a suppository. The temperature of the brooder wa then reduced to 27.7° C. (82° F.).

The following day, February 28, the weight was found to be 2480 grammes (5 pound 7 ounces). The brandy was still continued, and there was one yellow well-digested fæca discharge. The breast-milk had been entirely omitted, and 450 grammes (15 ounces) o modified milk had been taken in the twenty-four hours.

On the following day, March 1, it weighed 2510 grammes (5 pounds 8 ounces). Th amount of modified milk given was 495 grammes (16½ ounces) in the twenty-four hour and one drop of brandy was given with each feeding. There was great improvement i the infant's appearance, and it was much stronger.

On the following day, March 2, there had been no increase or loss in weight. Th temperature of the brooder was kept at 27.2° C. (81° F.). 510 grammes (17 ounces) o the modified milk were taken in the twenty-four hours. There was one fæcal movemen well digested and yellow.

On the following day, March 3, the weight was found to have increased to 26 grammes (5 pounds 11 ounces). The percentages of the modified milk were then change to the following (Prescription 39):

PRESCRIPTION 39.

℞ Fat	2.50
Sugar	5.00
Proteids	1.00

There were two well-digested fæcal discharges on this day. The temperature of t incubator was reduced to 25° C. (77° F.). 615 grammes (20½ ounces) of the modified mil were given in the twenty-four hours.

The following day, March 4, the infant was found to have lost 60 grammes (2 ounces), and the temperature of the brooder was therefore raised to 26.6° C. (80° F.). 630 grammes (21 ounces) of modified milk were taken in the twenty-four hours, and there was no especial change in the infant's condition.

On the following day, March 5, 30 grammes (1 ounce) in weight were found to have been gained, and the infant was looking better and decidedly gaining in strength. It was evident that the proper temperature for this especial infant at this age and at this period of its development was 26.6° C. (80° F.).

After this time the infant continued to develop normally, and on being taken out of the brooder in April was thriving in every way.

It is now five months old, and weighs 7110 grammes (14 pounds and 13 ounces).

DIVISION VI.

GENERAL PRINCIPLES OF EXAMINATION AND TREATMENT.

LECTURE XIII.

METHOD OF EXAMINING A SICK CHILD.—DRUGS.

BEFORE beginning in detail the actual study of the various classes o disease which I shall later present to you for examination, I should like t have you understand a few of the general principles a knowledge of whic I consider of importance in dealing with sick children.

When a physician is called to see a sick child, he must, if possibl ascertain before entering the nursery what is the temperament of the chil with whom he will have to deal, and by the aid of this information regu late the manner in which he approaches it.

An infant in the early months of life too young to fear a stranger, child of quiet phlegmatic temperament, or one that is too sick to object being handled, can be examined as soon as it is seen, with the regulari and precision which one would employ with the adult.

It is an entirely different task, however, when one is called upon to e amine children who are nervous, excitable, or timid, or who are spoil and vicious. In dealing with the first and more difficult class of these cas much deliberation in the way in which you approach the child is neede and much diplomacy in speaking to it is indicated. In the second, t spoiled and vicious class, you will not gain time by delaying the exam nation, and the sooner you have made it with firmness and persisten the less trying it will be for the child and for the mother. As a rule, t more the child cries and resists needlessly, the less likely is it to have a disease of serious import.

You will find that it is wise at first to make the child think th you are not taking any notice of it, and that you are not even aware o its presence. It is well to notice its toys, and to appear to take gre interest in them and also in the pictures in the nursery. The child ve soon will become accustomed to your presence, and will begin to take th same interest in you that you seem to take in its toys. A nervous, timid child will often from this point of the examination allow you to examine it without further trouble.

The physician, however, must always be gentle both in his voice and in his touch, and on the slightest appearance of timidity, or manifestation of a desire to avoid him, he must at once stop the special part of the examination which he is making, and devote himself again to the child's toys.

All these preliminaries and minute details, which seemingly delay the examination, in fact expedite it, since when once the timid child is thoroughly frightened, the rest of the examination becomes very unsatisfactory, for it is almost cruel in cases of this kind to attempt to force an examination, which in the case of the vicious child can be done usually without this feeling of cruelty and without hurting the feelings of the mother.

You should acquire the faculty of examining the child when it is crying and excited with the same precision as when it is quiescent, though perhaps by a somewhat different method. The trained hand and ear can detect an abdominal or pleuritic effusion or a solidified lung almost as well when the child is screaming as when it is perfectly docile.

This is an accomplishment which should be mastered at once by every practitioner who expects to have children under his care. In fact, if this were more universally understood, we should hear less of the impossibility of determining what is the matter with a child on account of its being fractious.

As the physical examination of a child is somewhat more difficult than that of the adult, and requires to be made more quickly, you should make use of every means at your command which will tend to throw light on the final result.

HISTORY.—A complete history of the case is very valuable, and should be obtained from the mother and the nurse, preferably before seeing the child, for in this way the physician can obviate asking many questions in its presence, a procedure which frequently fatigues it and renders it more difficult to examine. It is well to allow the mother and the nurse to tell you in their own language what they know about the child and its sickness. After they have finished, you can easily systematize the history of the case by any questions which you may wish to ask. Although the history given by the mother and the nurse is usually imperfect and disconnected, yet it is very likely to supply certain important points which you in your questions might easily overlook. The mother and the nurse are so constantly with the child that they notice all the slight shades of difference in its condition from hour to hour, a knowledge of which is of great importance in obtaining a correct appreciation of the general condition of the child, whatever the disease may be.

Having now systematized in his own mind the history of the case, the physician on entering the nursery should proceed with his inspection of the child. I am supposing that the child is in one of the two classes which I have mentioned as being especially necessary to manage with diplomacy.

TEMPERATURE.—Of course it is so important to ascertain what the temperature of the child is that, if possible, the temperature should be taken before the child has become frightened or fractious. The place for taking the temperature in these cases is usually in the axilla. You will find that the most successful method of obtaining the temperature under these circumstances is to explain to the mother and nurse exactly what you wish to have done. You should direct them to take the thermometer and show it to the child as though it were a toy, to put it under the child's arm, and to play with the child until you tell them to remove the thermometer.

INSPECTION.—One of the most valuable means of making a diagnosis of disease in children is the careful inspection of the child before attempting to percuss or to auscult it. In fact, where children are irritable and restless the inspection becomes of the utmost importance, and an eye which has been trained to understand the different aspects of disease in children readily makes the diagnosis in many cases without further assistance. A rule to be remembered, and one which you will find of great practical value, is, if possible, to have the child entirely undressed, so that you can see the whole surface of its skin in front and behind. Not only will you thus be able to recognize the symptoms attributable to a simple irritation of the skin, where otherwise you might be led to consider them as representing a more general and constitutional disturbance, but you will also find the skin to be a valuable index by which you can judge of diseases of the other organs. The cyanosis which so frequently represents some disturbance in the heart or lung, the quick respirations of either a thoracic or an abdominal type, a sunken or a distended abdomen, and the position of the child, all point towards symptoms belonging to special diseases. By means of all these symptoms, which we can see at a glance, the diagnosis of the special disease can usually be made without much aid from other sources.

RESPIRATION.—Either when the thermometer is under the child's arm or when you are beginning your regular inspection you can usually determine the rate and rhythm of the respiration. Having determined the temperature and respiration, if you have seen all that is necessary about the child when it is quiescent, you can proceed with the remaining part of your examination.

PALPATION.—Palpation is a very valuable means of diagnosticating disease in children, whether it be of the abdomen or of the thorax. It is well to begin with an attempt to take the pulse. Sometimes this can be readily accomplished. At other times it is impossible; and, as a rule, I rely less on the rapidity of the pulse in the child than on the information which is received from the temperature and respiration. It takes so little to increase the rate of the pulse in a young child that if we were to judge in every case by it we should often be misled in our diagnosis. What we wish especially to learn is whether there is a slow pulse or whether it intermits. This we can usually ascertain by keeping our finger for even only wo or three seconds on the child's radial artery. When we have once btained a fair idea of the rate and rhythm of the pulse we can proceed rith the remainder of our examination by palpation.

A young child's thoracic walls are so thin, and vibration is so proounced in them, that often we can detect what process is going on in the ung by merely putting our hand on the chest, and we can feel in a chronic ronchitis what will prove on auscultation to be coarse sonorous râles. We an also sometimes feel a pleuritic or a pericardial friction-rub, and freuently a roughening of one of the valves of the heart. It is not altogether npossible in certain cases to distinguish the difference presented to the hand etween a pleuritic effusion and a solidified lung. The examination of the bdomen, even when the child is crying, can be accomplished with considerble precision. Waiting until the child stops crying for a second and elaxes its abdominal walls, you can, by firm but gentle pressure, so depress he abdominal walls as to obtain a fair knowledge of whether you have an bdominal tumor to deal with. You can also readily detect by palpation uid in the abdominal cavity.

A rectal examination is often important in infants and young children. t can readily be done without hurting the child, and the finger is able to each much further proportionately into the child's pelvis than into that of he adult, and very much more can be learned by this method than in adult ases. An invagination or an appendicitis can be diagnosticated by the ombined examination through the rectum and by external pressure where xternal palpation alone has failed to give evidence of disease.

In the infant the head should be carefully examined in regard to the ontanelles. Measurements should be taken of the head and of the thorax.

At this stage of the examination you will have determined almost always rhat disease is affecting the child, but you should, of course, make use of very known method for verifying your diagnosis. You should, therefore, ndeavor to percuss and auscult the child, but in a somewhat different way rom that which you would naturally employ with the adult. The louder he child cries, the easier is it to obtain evidence through vocal fremitus rhat the disturbance is in the chest.

PERCUSSION.—Even when the child is crying and resisting, percusion may be of the greatest importance. Light percussion, as a rule, is referable to the deeper and heavier percussion which is often so valuable in he adult. The chest-walls, as I have said, are so resonant that deep perussion rather masks the process which is directly under the finger by bringng out sounds from all parts of the chest. Direct percussion with the nger I have always found preferable to using any instrument, as in this ray both palpation and percussion may be combined. Palpatory percusion in my hands has always proved exceedingly valuable for diagnostic urposes. A few light taps over the normal boundaries of the heart and ung, which I have described to you in a previous lecture (Lecture IV., ages 121, 122, 124), will give you much information, even though you are unable to effect a more extended percussion of the chest. If the child is crying, you should watch until it takes its breath. Just as it draws in its breath it necessarily stops crying, and at that minute you can get a perfectly clear result from your percussion.

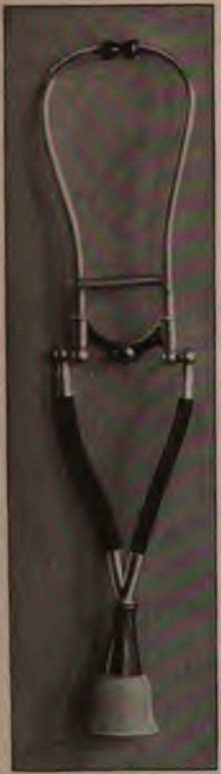
You should be careful not to make your physical examination too protracted. Rapidity of motion, both in palpation and in percussion, is very important, and you should learn to examine a young child with much greater rapidity than is usual or necessary in the case of an adult. You will in this way obtain much more information than if you wearied the child by continual efforts to make sure that you had not made a mistake in the evidence which you have acquired up to this point of the examination.

The sounds which can be elicited from a young child's chest are so varied that it is more difficult to differentiate them than in the adult. If, therefore, you allow yourself to hesitate and to doubt, you will not arrive at as correct a result in your examination as when you have trained your mind to grasp at once the salient points in the special physical examination, and to depend somewhat more on the first idea which you form than would be wise in the case of an adult.

AUSCULTATION.—I am accustomed next to auscult the child. A word may, perhaps, not be deemed unnecessary in regard to the form of stethoscope which I am in the habit of using in examining infants and young children. It is, I think, unwise to accustom yourselves to the use of one form of stethoscope, as you will often have to examine children at times when you have not your stethoscope with you, and yet when it may be of the greatest importance that a definite diagnosis of the case should be made. I have noticed that children are much more sensitive to the feeling of the stethoscope than are adults. In many cases they shrink from it as though it hurt them, even when they have not been frightened by the previous part of the examination with palpation and percussion. It is, therefore, exceedingly important to make the examination as pleasant to the child as possible. I have found that a rubber cup applied to the end of the stethoscope serves this purpose well. The feeling of the soft rubber is pleasant to the child, and it conveys the sound with almost as much clearness as does the hard rubber end of the stethoscope. This rubber cup can be applied to any stethoscope, such as this one (Fig. 84), which, however, does not convey the sound quite so clearly as does this other stethoscope (Fig. 85), which is of such small calibre that it can easily be introduced between the ribs of even a young child, and which differentiates the sounds much more clearly than is done by any other stethoscope which I have seen.

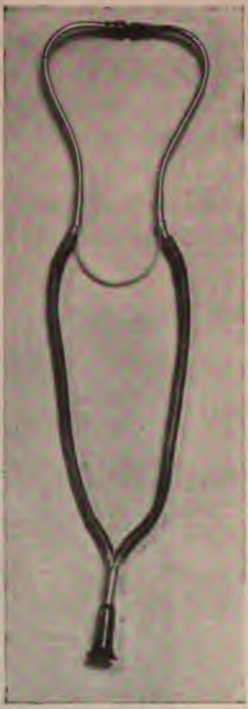
In my opinion, it is often of great aid in the proper appreciation of the sounds which are heard with the stethoscope in infants and in young children, especially when they are crying, to use a stethoscope which does not convey the sound so clearly and intensely as do others. We can often in this way differentiate a soft cardiac murmur which if a more delicate in-ment were used would be entirely obscured by the loud sounds coming n the trachea and bronchi of a crying or screaming child. We can, , often distinguish the fine râles of a broncho-pneumonia in contra- inction to the loud coarse râles which tend to obscure the other sounds he chest. For a routine examination, however, and for rapidity in its pletion in cases where we see that a prolonged auscultation will prove e impossible, the smaller stethoscope (Fig. 85) is best adapted for our pose.

FIG. 84.



Stethoscope.

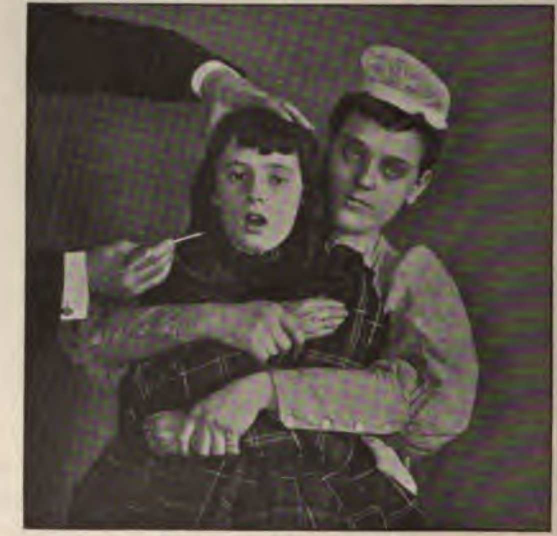
FIG 85



Stethoscope.

EXAMINATION OF THE THROAT.—We have now examined the ld in every way except one, which is an exceedingly important one, the ission of which might be productive of errors in diagnosis. This is the amination of the throat. I have left the examination of the throat to a me when we have practically finished with the general examination of the hild, because, as a rule, it is the procedure of all others which irritates it, nd after we have once attempted to examine the throat we shall seldom be orgiven by the child at that special visit. Some children will allow you to ook into their throats without being at all disturbed. As a rule, however, t frightens them, and we should use the most gentle and rapid methods for ccomplishing our purpose. We must not expect to be able to sit down in front of the child and examine its throat for some minutes, as is possible with adults. We must adopt some definite method by which we can control the child and catch a glimpse of the mouth, tongue, and pharynx. The more quickly we do this, the less it frightens the child, and it is important that we should not make extensive preparations, which it will notice and which will indicate what we are going to do. The mothers are often much disturbed by seeing the child first frightened with the idea that it is going to have a spoon put in its mouth, and then, while screaming and crying, forced to the window and compelled to open its mouth. It is far better under all circumstances to tell the mother and the nurse what to do, and not to go near the child until they are entirely prepared to control its limbs and are holding it in a position in which it is practically helpless. It frightens the child much less to have it sit in the nurse's lap with its face to the window than to examine it on its back. I can illustrate best the proper method of examining a child's throat where we expect to meet with resistance, by picking out a really vicious child, and one which has been made vicious by being spoiled, for in these cases we meet with the greatest difficulty, and they are cases where diplomacy, persuasion, and delay are of no avail. I happen to have here to-day a child of this kind (Case 112).

Case 112.



Clinical examination of throat.

She is eight years old and well developed, and she will be determined to resist our efforts to examine her throat. My directions for examining the throat of such a child are as follows:

I do not let her see what I am going to examine the throat with, nor do I go near her until she is ready to be examined. The nurse is instructed to lead the child to a window, place a chair in front of the window, and sit down in it, with her face to the window. She then lifts the child into her lap, holding its back upright against her chest, and holds it by clasping her arms around its arms. By clasping the child's ankles between her feet or knees, the nurse can absolutely control its movements. She cannot move her arms or her legs, nor can she slip down in the nurse's lap, but she is forced to sit upright. All she can do is to move her head. When she is once in this position I place my left hand on the top of her head, and thus control the movement of the head. She will, as you see, open her mouth, and then, watching me, quickly shut it up again just as I am about to put the handle of the spoon in her mouth. I next carefully place the handle of the spoon between the child's lips. If necessary, in cases which are very intractable, closing the nostrils will make the child open its mouth to get breath. This is usually not necessary, and all that we have to do is patiently, firmly, gently, and persistently to watch our opportunity, and take advantage of it when it comes, to introduce the handle of the spoon between the teeth, and gradually put it on the tongue. When the end of the handle of the spoon touches the soft palate the child will gag, and by steady pressure at this moment on the base of the tongue a perfectly clear view of the throat will be obtained, and in this one glance you should take in all that is to be seen.

You will thus successfully accomplish an examination in a few seconds which the mother had feared would be prolonged and harrowing.

I prefer to use a spoon for examining the throat, because in every household you have one at your command, and it obviates the use of the same instrument in a number of mouths, which is something to be considered in children, where infection by the mouth is so common. Of course, for those who prefer to use the usual tongue-depressor the danger is reduced to a minimum if a careful disinfection of the instrument is made after it is used; but in the case of infants, who should also be examined in an upright position, the spoon is decidedly preferable. This is so because the neck of the infant is so short that its chin is in close proximity to its chest, and the handle of the tongue-depressor interferes with the proper downward pressure of the instrument. The spoon-handle, on the other hand, is exactly the shape which is best adapted to the infant's mouth and tongue, and the spoon, being comparatively straight, does not encroach upon the thorax when the downward pressure is made.

In regard to the examination of the throat, this part of the child may be affected often, and may be the only source of the symptoms which you will be called upon to explain, and yet these symptoms may not be what you would expect to find where the trouble is in the throat. Young children are so apt not to complain of trouble in the throat, and to show merely signs of general constitutional disturbance, that the physician is very likely to be misled and to overlook the real seat of the disease unless he makes it a rule always to examine the throat at his first visit.

INSPECTION OF THE MOUTH.—It is well when the physician is examining the throat of an infant in the first two years of its life, and even later if there are any symptoms which point towards the mouth, to examine carefully the gums. I need scarcely caution you to wash your hands carefully before introducing your fingers into the mouth. This is in accordance with the common rules of cleanliness, and also is required in order that you should avoid the introduction of pathogenic organisms into the infant's mouth. In examining the gums you can judge whether they are swollen or reddened, dry, moist, or hotter than normal, and also at times, as I shall explain to you when speaking of diseases of the mouth in children (Lecture XL., page 797), you will in this way be able to decide whether there is a condition of the gums which indicates the use of the lancet.

EXAMINATION OF THE EARS.—One of the most important means of rightly interpreting the symptoms of restlessness, of evident pain, of heightened temperature, of undue somnolence, as well as a great many other symptoms, is the examination of the ears of infants and of young children. A slight irritation in the throat may at times cause a congestion in the vessels of the membrana tympani which may produce all these symptoms.

It is, therefore, very important, unless you are sure that the symptoms do not arise from some condition in the ear, that you should examine the ears at some time during your visit, choosing that time which seems most favorable in the especial case. I consider a thorough knowledge of the possible symptoms which may arise from the ear of the very greatest importance for the general practitioner to possess.

DRUGS.—An important fact to remember in the treatment of infants and young children is that drugs play a very insignificant part in the actual cure of their diseases. According to my observation, numbers of children are being treated by drugs, and yet often, so far as I can see, this time-honored means of satisfying parental prejudices is but prolonging the symptoms of a disease which, self-limited, has run its course. I do not for a moment question the direct benefit obtained from quinine in malaria and mercury in syphilis: it is the promiscuous use of drugs in every case of sickness to which I am especially opposed, for in many cases the child will recover with equal or even greater rapidity without them.

Instances probably arise in the practice of every physician where he feels that the drugs which have been given have either directly harmed the child or, by disturbing its digestion and thus interfering with its nutrition, have indirectly produced more serious symptoms than those presented by the original disease. The greatest caution should be employed where drugs are used with young children, and there should be a thorough understanding of their action during the various periods of development. The well-known susceptibility of children to the action of opium and its alkaloids should make us careful to begin with minimum doses when it is necessary to use this drug. In like manner, although it is traditional that children have a great tolerance for belladonna and arsenic, we must allow that an overdose of the former, although not usually fatal, may certainly produce most alarming symptoms, while the administration of the latter as I have seen it given in the treatment of chorea has in a number of cases produced a multiple neuritis.

The treatment of diseases by special drugs because these drugs have been given in the past, because their administration has apparently done no harm, or because no new or better remedy has been found, rests upon a lack of comprehension of what treatment really means.

The custom of combining many drugs in one prescription is fallacious, nd should be discountenanced, especially where infants and young children re being treated. A single drug given in the smallest dose which will ccomplish its purpose, and in the most agreeable form which is compatible ith the function of digestion, will produce the best results in any given isease.

The delicate skin of infants and young children is peculiarly sensitive to eflex disturbances caused by drugs in the gastro-enteric tract, and thereore we must be careful not to mistake the appearances produced by such eflex irritation for the various lesions of the skin which may occur in a pecific disease. Thus, the similarity of the efflorescence produced by bellaonna to that accompanying scarlet fever is striking. Almost any drug, as ell as certain articles of diet, may in some individuals produce forms of apular erythema, resembling very closely some of the dermal lesions of yphilis. It is therefore wise to avoid these possible disturbances of nutriion by giving drugs only where they are actually known to be necessary, nd by omitting them as soon as possible.

It has always seemed to me irrational to prescribe syrups as a mentruum for the administration of drugs to children. Their well-known endency to fermentation is sufficient to stamp them as unfit for the treatent of a period of life when the undeveloped condition of the digestive unction indicates the vital importance of protecting this function in every ay.

Each case must be treated according to its special pathological lesion or pecific micro-organism. As year by year we are discovering the organisms hich cause special diseases, so the treatment of the future will be the ctual destruction and speedy elimination of these organisms while supportng the strength until such elimination has been accomplished. Where no nown organisms exist, the treatment should be if possible to remove the ause, and to support the vitality until natural processes have healed the pecial lesion, produced either by exposure or by trauma.

In connection with what I have said regarding the unnecessary use of rugs in early life, the following case is of considerable significance:

An infant five months old was reported to me to have tubercular meningitis. The istory of the case was as follows:

A healthy breast-fed infant (Case 113), with a healthy mother, had been for two weeks howing signs of fretfulness, which, as afterwards proved, were closely connected with irriation of the two lower middle incisors, which were in the process of coming through the um. The infant had had a slight cold for two days, and on the second day had been more estless than usual in the afternoon, and had screamed a great deal. The attending physiian prescribed a mixture of fifteen drops of tincture of opium in thirty teaspoonfuls of ater, to be given in teaspoonful doses at intervals during the night, if it was found necesary to quiet the infant. During the night the infant's hands and feet were reported to be old, and by morning it was found to be almost unconscious. The physician at this time ade the diagnosis of tubercular meningitis, and on the following day, when it was seen by e with him, it was found to have contracted pupils, cool skin, a rectal temperature of 37° . (98° F.), a fontanelle somewhat depressed, a regular pulse, 120, and respirations quiet and not especially slow. It did not notice anything, except when it was roused, at which time it would cry vigorously, as though it were annoyed at being disturbed.

On inquiry, it was found that the nurse during the night had given eight teaspoonfuls of the mixture which I have just mentioned. This amount must have contained at least four drops of tincture of opium.

A dose of sulphate of atropia of 0.0003 ($\frac{1}{200}$ of a grain) was given at once by the mouth. Four hours later the pupils became less contracted, but were reacting sluggishly. An hour later another dose of sulphate of atropia of the same strength was given, and the pupils then dilated, the infant grew brighter, and recovered within twenty-four hours.

After the second dose of atropia had been given, an efflorescence, which probably was the result of the physiological action of the atropia on the skin, appeared on the chest and face for a few hours, and the skin then became normal. This efflorescence, it is well to record, was at first mistaken for that of scarlet fever, so that in the same case an erroneous diagnosis of two entirely different diseases was made, and in each case the symptoms supposed to represent these diseases were really caused by the drugs which had been given to the patient.

DIVISION VII.

THE BLOOD IN INFANCY AND CHILDHOOD.

LECTURE XIV.

ERATURE.—NOMENCLATURE.—BLOOD-KEY.—METHODS.—CHEMISTRY.—ORIGIN.—FŒTAL BLOOD.—THE NORMAL CONDITIONS OF THE BLOOD IN EARLY LIFE.

As our knowledge advances regarding the etiology of disease, it is be- ing very evident that we should not only direct attention to the pathology the tissues outside of the blood, but should also investigate the varied ditions which exist in the blood itself. The blood does not merely absorb waste matter from the tissues and carry fresh oxygenated material to lace it. It plays a far greater part in the economy than this, and is inti- tely connected with many diseases.

It is not only in the corpuscular elements of the blood that we find ious changes corresponding to certain conditions existing in the indi- ual. We must in the future go still further and read what the blood um is waiting to disclose to us.

Although an immense amount of labor has been expended on examina- ns of the blood, both chemical and microscopic, especially in that of ults, the present state of our knowledge concerning its diseases, and its ditions as representative of other diseases, is very unsatisfactory.

Our knowledge of the blood in early life is still more meagre than at a later iod. Although in the last few years the literature of the blood in general become very extensive, yet that pertaining to infancy is small. We must, ed, confess that what we definitely know of the diseases of the blood in first few years of life is wanting in exactness and veiled in obscurity.

It is exceedingly important, therefore, that the results of individual stigation in this class of cases should be published as soon as possible, he purpose of rendering mutual aid in unravelling the mysteries of this esting subject. For many years I have met with cases which have been ult to classify beyond their evident connection with the blood.

During the last two years I have endeavored to formulate more sys- tically my clinical observations on these cases, and I have been enabled to collect some valuable data for diagnosis and prognosis. In the accomplishment of this work I have received much assistance from Dr. William F. Whitney, who has with great patience and labor differentiated the specimens as they were brought to him from the several cases on slides for microscopic examination. I wish especially to direct attention and award great merit to Dr. A. H. Wentworth's work. He has labored in this field for me unceasingly during the past two years, going to my cases, preparing the slides, and estimating the red and white corpuscles and hæmoglobin. Up to the present time very little work on infants, corresponding to Dr. Wentworth's, has been done in this country, and it is therefore especially valuable.

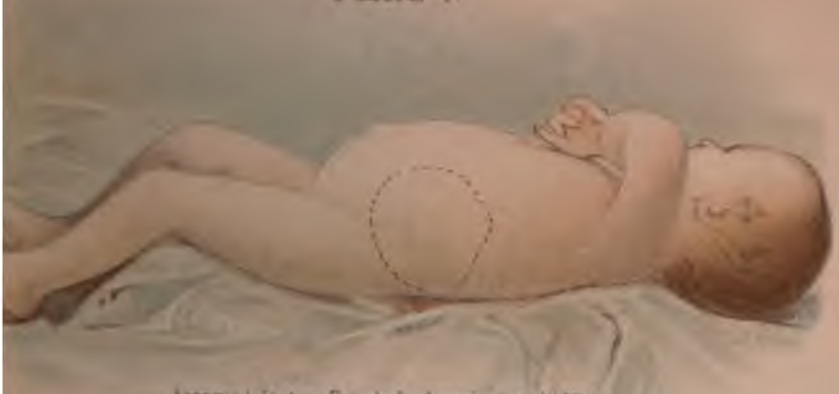
NOMENCLATURE.—The various terms used to designate the elements of the blood will soon become as familiar to the general practitioner as those now used in clinical medicine. I think, however, that you may not deem it unnecessary for me to explain to you the meaning of some of the words which I am about to use. I have endeavored to do this in the following table (Table 84), and by means of this colored plate, showing the various elements of the blood (Plate V.).

TABLE 84.

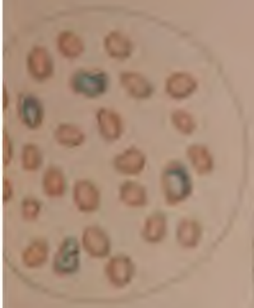
Meaning of the Terms used in Describing the Blood.

ERYTHROCYTES	Normal red corpuscles	(Plate V., 5.
1. Hæmatoblasts of Neumann.	Nucleated red corpuscles	(Plate V., 6.
(1) Normoblasts	Size of erythrocytes, having a small deeply staining nucleus	(Plate V., 6.
(2) Megaloblasts	Large nucleated red corpuscles, having a large, often fragmented, nucleus, staining faintly	(Plate V., 6
(3) Microblasts	Small nucleated red corpuscles.	
2. Hæmatoblasts of Hayem and Bizzozero.	Blood plates, supposed by these authors to be young red blood-corpuscles.	
3. Microcytes	Abnormally small erythrocytes	(Plate V., 7
4. Macrocytes or Megalocytes.	Abnormally large erythrocytes.	
5. Poikilocytes	Abnormally-shaped erythrocytes	(Plate V., 7
Oligocythæmia	Reduction in number of erythrocytes.	
Hæmolysis	Destruction of erythrocytes.	
Hæmoglobin	Coloring matter of the blood.	
Oligochromæmia	Reduction of hæmoglobin.	
Hæmoglobinæmia	Presence of hæmoglobin in the serum.	
Hæmoglobinuria	Presence of hæmoglobin in the urine.	
LEUCOCYTES	White corpuscles.	
1. Lymphocytes or small mononuclear. (Young (unripe) elements of Uskow.)	Round mononuclear cells about the size of erythrocytes, with faintly staining protoplasm. The nucleus stains deeply, and fills nearly the whole cell	(Plate V.,
2. Large mononuclear (Mature (ripe) elements of Uskow.)	Fully double the diameter of erythrocytes, with oval or round faintly-staining nucleus, filling a relatively small part of the cell	(Plate V.,

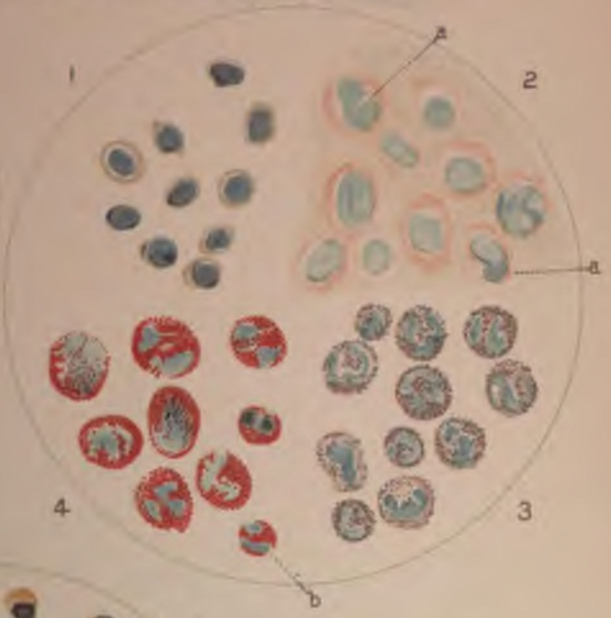
PLATE V.

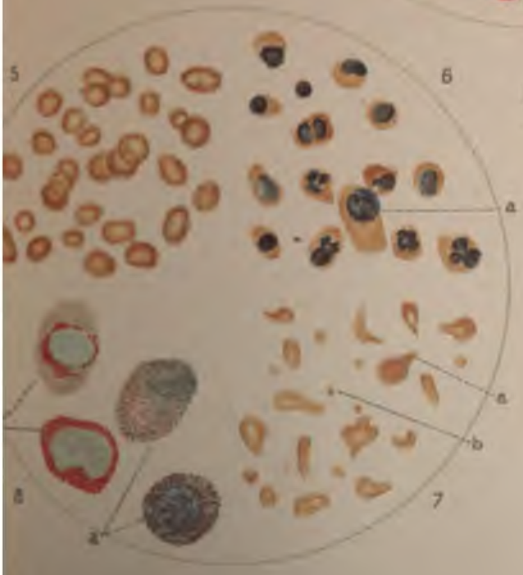


Anaemia Infantum Pseudo-Leukaemica von JAKSCH.



Plasmodium Malariae
Oil Immers. REICHERT 1/12
Ocular N°3.





LEITZ Oil Immers. 1/12 Ocular N°3.

1. Small Mononuclear.
   (Lymphocytes.)
2. Large Mononuclear.
   a. Transitional.
3. Polynuclear Neutrophiles.
4. Polynuclear Eosinophiles.
   b. Dwarf Eosinophilic Cell.
5. Normal Red Corpuscles.
6. Nucleated Red Corpuscles.
   (Normoblast type.)
   a. Megaloblast type.

7a. Poikilocytes.
   b. Microcytes.

8a. Large Mononuclear Neutrophiles, or Myelocytes.
   b. Large Mononuclear Eosinophiles.

TABLE 84.—*Continued.*

nal forms lated or indented of Uskow.)	Cells like the above, but having an indented nucleus	(Plate V., 2*a*.)
hiles lynuclear cells correctly poly- nuclear. Old ipe) elements tow).	Considered by most observers the oldest variety of the leucocytes. The nucleus stains with basic stains; the plasma stains faintly with neutral aniline stains, and the granules stain with a combination of both basic and acid stains, and hence are called *neutrophiles.* The nucleus is really polymorphous, though sometimes (apparently) broken	(Plate V., 3.)
tes e mononuclear philes. "Mark- " of the Ger-	Large, round, or ovoid cells, with one (seldom two) large faintly-staining nuclei. The plasma is filled with small granules that take a neutral stain	(Plate V., 8*a*.)
iles	Characterized by the presence of large, round, highly refractile granules, which stain with all acid coloring matters	(Plate V., 4.)
lymorphnuclear		(Plate V., 4.)
nonuclear		(Plate V., 8*b*.)
tosis	An increase in the number of leucocytes, the increase being in the polymorphnuclear neutrophiles.	
osis	An increase in the number of microcytes.	
omatophilic	Taking only one stain.	
matophilic	Taking more than one stain.	
ic	Stained by basic stains.	
ilic inophilic.	Stained by acid stains.	
hilic	Stained by neutral stains.	
hilic	Stained by both basic and acid stains.	
lasts	A term used by some authors to describe certain very early stages in the development of erythrocytes found only in the blood-forming organs.	
asts	A similar term applied to the early stages in the development of leucocytes.	
ryokinesis. ect cell division.)	A division of nucleus and cell in which the division is preceded by certain definite changes in the arrangement of the morphological constituents of the nucleus and cell.	
s t cell division.)	A simple division of nucleus and cell, not accompanied by previous alteration in the constituents of either.	

D-KEY.—I should like you to examine these colored pictures facing page 330), which represent all the principal normal nal conditions of the blood in early life. They are, in fact, a Dr. Wentworth has so arranged that, by first becoming familiar with the pictures in the plate, and then calculating the percentages from your microscopic blood-slides, you will be able to read and understand the special case which you are studying. Thus, knowing the special combination of the blood-elements which constitute a certain disease, and seeing that combination under your microscope, you can easily make the diagnosis of the disease.

This method is, of course, more satisfactory than showing the blood-field of any single case; for what we see is of no particular value until a histological computation of the relative percentages of the different elements composing the blood has determined the especial combination which represents the particular disease.

I shall now briefly describe to you the general methods by which blood-counts are made. For the special technique I must refer you to the admirable laboratory courses that are given in another department of the school.

The instrument used for estimating the number of red and white corpuscles is called the Thoma-Zeiss. It consists of two parts, a glass counting-slide and a mixing-pipette. By pricking the lobule of a carefully washed ear a single drop of blood is made to exude. The first drop of blood that comes having been wiped away and a second having taken its place, the end of the mixing-pipette is brought in contact with it and a portion is sucked up in the capillary tube, the amount taken being shown on a scale. The end of the pipette is then immersed in a diluting fluid, and the blood, with either one hundred or two hundred times its volume of the diluent, is sucked into the bulb to which the capillary tube enlarges. After this the pipette is shaken for at least two minutes to insure the even distribution of the corpuscles. For counting the erythrocytes the best diluting solution is that known as Toison's, the formula for which is,—

PRESCRIPTION 40.

℞ Sulphate of sodium	8.000
Chloride of sodium	1.000
Glycerin	30.000
Methyl violet	0.025
Distilled water	160.000

Having blown out the first few drops from the pipette, in order to be sure of getting a drop that represents a fair average, the next is put in the small depression that is made for it in the counting-slide. Around this central well is an overflow moat, bounded by a slightly raised glass plate. The whole is so constructed that, when the central well is evenly full of fluid and sealed over by laying on the cover-glass, its depth is exactly one-tenth of a millimetre. On the floor of this well a square millimetre has been ruled off into four hundred small squares. The drop of blood, just large enough to fill this well, but not to overflow the moat, shut in by its cover-glass, is laid aside for two or three minutes, so that the globules nay all fall to the bottom. Then, under the microscope, the number of ed and white corpuscles lying in a definite number of the small squares an readily be counted, and, as each square is just one four-thousandth of a ubic millimetre, a very simple multiplication will give us the number of orpuscles in a cubic millimetre of the undiluted blood. All blood examiations are reported in terms of cubic millimetres. In my cases, one undred and sixty small squares were counted in each of four successive rops of blood, and the average made up from these.

Although, owing to the blue color given them by the Toison's soluion, the leucocytes can be counted at the same time as the erythrocytes, yet it is often more convenient to use a solution that, by rendering the red cells invisible and emphasizing the nuclei of the whites, will render the task of counting the relatively small proportion of white cells easier. For my counts a three-tenths of one per cent. solution of acetic acid was used, the blood diluted one hundred times, and twenty thousand squares counted.

The hæmoglobin percentage is generally determined by means of the Fleischl hæmoglobinometer. A fine piece of glass tubing set at right angles in a handle is used as the measure for the blood, which is then dissolved out in distilled water held in a glass-bottomed cell. The colored water is compared by artificial light with a graduated red glass prism, and when the exact equivalent is found an index on the instrument shows the percentage of hæmoglobin.

The third step in the process is the making of the dried cover-glass preparations. This is the one that can be most readily done by the general practitioner, and the one that will give him the most information. The cover-glasses must first be carefully cleansed with acid and alcohol. One of the clean cover-glasses is taken in the forceps and gently touched for a moment to a fresh drop of blood as it rests upon the ear; the second glass is then dropped upon the first. If both were perfectly clean, the blood will be seen immediately to spread itself out between the two as a delicate film. The glasses are at once separated by a sliding motion, and allowed to dry in the air. When dry, if protected from dust and moisture, they can be kept indefinitely. The next step in the process is to harden them. This may be done by soaking them in benzine for ten minutes, or in equal parts of ether and absolute alcohol for half an hour; or they may be heated, preferably by leaving them for two hours on a metal plate kept just hot enough to vaporize a drop of water, but for rough clinical work holding them in the fingers for a couple of minutes over the flame of an alcohol lamp is sufficient.

The last step in the process is the staining. To accomplish this a great variety of stains have been used, each bringing out some one peculiarity of ell-structure with greater distinctness than the others. The cells shown in he colored plate (Plate V., facing page 330) were stained with the Ehrlich riple stain, which is made as follows:

PRESCRIPTION 41.

℞ Saturated watery solution of orange G	125 c.c.
Saturated watery solution of acid fuchsine (containing 20 per cent. alcohol)	125 c.c.
To this are added slowly, while constantly shaking the mixture, a	
Saturated watery solution of methyl green	125 c.c.
Absolute alcohol	75 c.c.

This stains the nuclei of the leucocytes a blue or bluish-green color, the neutrophilic granules a purple, and the eosinophilic granules a reddish tinge. The erythrocytes are stained a faint yellow. According to the amount of heating the cover-glasses have had, they are left in the staining fluid from one to four minutes, and then washed in plain water and dried. Finally, mounted in cedar oil or Canada balsam, they are ready for examination with the oil-immersion objective.

Of these various procedures the estimation of the hæmoglobin percentage must be done at once. The mixing-pipette, having been filled and shaken, can safely be left from twelve to fourteen hours or even longer without any change taking place in the corpuscles that will interfere with their being counted. The cover-glass films can, as I have already told you, be kept indefinitely.

CHEMISTRY.—The chemistry of the blood in early life has not yet been fully investigated. It may, however, be of interest to you to have a general idea of this fluid medium which we are about to study in health and in disease as it exists in the adult. In this way you will be better prepared to understand the more intricate pathological questions which must be dealt with later. This general idea can best be acquired by directing our attention to the chemistry of the blood.

The chief chemical facts which are known concerning the blood have been so lucidly stated by Foster that I can best assist you by quoting from what he says on the subject. Foster states that the average specific gravity of human blood is 1055, varying from 1045 to 1075 within the limits of health. The reaction of the blood as it flows from the blood-vessels is found to be distinctly, though feebly, alkaline. If a drop be placed on a piece of faintly red highly glazed litmus paper and then wiped off, a blue stain will be left.

The whole blood contains a certain quantity of gases, such as oxygen, carbonic acid, and nitrogen, which are held in the blood in a peculiar way, and which vary in venous and in arterial blood, and so serve especially to distinguish them from each other. These may be given off from the blood when exposed to an atmosphere, according to the composition of that atmosphere.

The normal blood consists of corpuscles and plasma. If the corpuscles be supposed to retain the amount of water proper to them, blood may, in general terms, be considered as consisting by weight of from one-third to somewhat less than one-half of corpuscles, the rest being plasma. The plasma is resolved by the clotting of the blood into serum and fibrin.

The serum contains, in 100 parts,—

Proteid substances	about 8 or 9 parts.
Fats, various extractives, and saline matters	about 1 or 2 parts.
Water	about 90 parts.

The proteids are paraglobulin and serum-albumin in varying proportions, there being probably more than one kind of serum-albumin. We may perhaps say that they occur in about equal quantities.

The fats, which are scanty, except after a meal or in certain pathological conditions, consist of the neutral fats, stearin, palmitin, and olein, with a certain quantity of their respective alkaline soaps. The complex fat lecithin occurs only in very small quantities. The amount present of the peculiar alcohol cholesterin, which has so fatty an appearance, is also small. Among the extractives present in serum may be put down nearly all the nitrogenous and other substances which form the extractives of the body and of food, such as urea, kreatin, sugar, and lactic acid. A very large number of these have been discovered in the blood under various circumstances, the consideration of which must be left for the present. The odor of blood or of serum is probably due to the presence of volatile bodies of the fatty acid series. The faint yellow color of serum is due to a special yellow pigment. The most characteristic and important chemical feature of the saline constitution of the serum is the predominance, at least in man and in most animals, of sodium salts over those of potassium. In this respect the serum offers a marked contrast to the corpuscles. Less marked, but still striking, are the abundance of chlorides and the poverty of phosphates in the serum as compared with the corpuscles. The salts may, in fact, briefly be described as consisting chiefly of sodium chloride, with some amount of sodium carbonate—or, more correctly, sodium bicarbonate—and potassium chloride, with small quantities of sodium sulphate, sodium phosphate, calcium phosphate, and magnesium phosphate. Of even the small quantities of phosphates found in the ash, part of the phosphorus exists in the serum itself, not as a phosphate, but as phosphorus in some organic body.

The red corpuscles contain less water than the serum, the amount of solid matter being variously estimated at from 30 to 40 per cent. or more. The solids are almost entirely organic matter, the inorganic salts amounting to less than 1 per cent.

The red coloring matter which in normal conditions is associated with this stroma may by appropriate means be isolated, and in the case of the blood of many animals obtained in a crystalline form. It is called *hæmoglobin*, and may by proper methods be split up into a proteid belonging to the globulin group, and into a colored pigment, containing iron, called *hæmatin*. Hæmoglobin is therefore a very complex body. It is found to have remarkable relations to oxygen, and indeed the red corpuscles by virtue of their hæmoglobin have a special work in respiration, for they carry oxygen from the lungs to the several tissues.

Of the organic matter, again, by far the larger part consists of hæmoglobin. In 100 parts of the dried organic matter of the corpuscles of human blood about 90 parts are hæmoglobin, about 8 parts are proteid substances, and about 2 parts are other substances. Of these other substances one of the most important, forming about a quarter of them and apparently being always present, is lecithin. Cholesterin appears also to be normally present. The proteid substances which form the stroma of the red corpuscles appear to belong chiefly to the globulin family. As regards the inorganic constituents, the corpuscles are distinguished by the relative abundance of the salts of potassium and of phosphates. This at least is the case in man. The relative quantities of sodium and potassium in the corpuscles and serum respectively appear, however, to vary in different animals; in some the sodium salts are in excess, even in the corpuscles.

The proteid matrix of the white corpuscles is composed of myosin, or an allied body, paraglobulin, and possibly other proteids. The nuclei contain nuclein. The white corpuscles are found to contain, in addition to proteid material, lecithin and other fats, glycogen, extractives, and inorganic salts, there being in the ash, as in that of the red corpuscles, a preponderance of potassium salts and of phosphates.

The main facts of interest, then, in the chemical composition of the blood are as follows. The red corpuscles consist chiefly of hæmoglobin. The organic solids of the serum consist partly of serum-albumin and partly of paraglobulin. The serum or plasma contrasts in man, at least, with the corpuscles, inasmuch as the former contains chiefly chlorides and sodium salts, while the latter are richer in phosphates and potassium salts. The extractives of the blood are remarkable rather for their number and variability than for their abundance, the most constant and important being perhaps urea, kreatin, sugar, and lactic acid.

ORIGIN.—According to Ziegler, the regeneration of the colorless blood-corpuscles takes place principally in the lymphadenoid tissues of the lymph-glands, the spleen, and the intestinal tract. The lymph-bulbs contain regions, sharply differentiated from the surrounding tissue, in which are always to be found stellate figures which for the most part belong to free cells. These regions are called by Flemming "germ-centres." In addition to this, a division of leucocytes takes place in the lymph-channels of the lymph-glands and other tissues, and there can be little doubt that leucocytes also divide while circulating in the blood or wandering among the tissue-clefts.

This division may be either by mitosis, which gives cells with peculiarly lobate or crown-like nuclei, or by amitosis, in which case the nucleus appears broken into fragments.

The mitotic division is that which leads to the formation of vigorous cells. To what extent the amitotic division—that is, the breaking down of the nucleus—is also followed by cell division is difficult to determine, but it is probably true that this represents the result of a process of destruction, d that the change from mononuclear to polynuclear cells should therefore looked upon as a degeneration. Frequently under pathological conditions ere occurs an increase in leucocyte-formation.

Since in leucæmia the spleen, the lymph-glands, or the bone-marrow ow a hypertrophic condition with increased cell-production, it may be pposed that they furnish the increase of leucocytes to the blood. The generation of the red blood-corpuscles occurs probably by mitotic division the red nucleated young forms. In adults this division takes place only the bone-marrow, which is true also of mammals, birds, reptiles, and the illess amphibiæ; in the tailed amphibiæ and in fishes it can occur in e spleen as well. In the embryo this process can go on in the entire ascular system. Later it becomes concentrated in the liver, spleen, and one-marrow, and finally becomes restricted, according to our present nowledge, to the marrow alone. Where these nucleated young forms riginate is still a matter of dispute, some investigators considering that ey are the direct descendants of the young forms of embryonic life and at they have always held hæmoglobin, others maintaining that they are eveloped from pre-existing nucleated forms without hæmoglobin, which in eir turn are said by some to multiply in the vessels of the marrow, and y others to originate also in the spleen.

Neumann believes that there either occurs a development of the nucle- ted blood-corpuscles out of the leucocytes of the blood which after birth re conveyed through the arteries to the bone-marrow, or that they spring om the tissue elements of the bone-marrow.

FATE OF THE RED CORPUSCLES.—About the length of life and the ltimate fate of the red corpuscles little is known. Osler points out at the bile coloring matters and certain of the urinary pigments have eir origin in altered hæmoglobin, which would require the daily destruc- ion of many red blood-corpuscles. So far as we can see, these corpuscles re removed without undergoing much alteration. Certain evidence, however, seems to point to the spleen and liver as organs in which they are broken up, and in which they are perhaps used again in making the new corpuscles.

FŒTAL BLOOD.—In accordance with the fact that the younger the individual the more unformed, or rather unripe and undeveloped, are the elements of the blood, certain stages of the corpuscular development being transmitted directly from the fœtal conditions, it will be wise to speak first of the character of the blood in intra-uterine life. Taking these conditions as a starting-point and using the adult blood for comparison, we can obtain a fairly comprehensive understanding of the various conditions which are known to be present in the blood of infants and children.

According to Scherenziss, the specific gravity of the fœtal blood at the moment of birth is somewhat lower than that of the adult's. That of the serum is markedly lower. The red corpuscles are poor in hæmoglobin and rich in stroma. Compared with the adult, the hæmoglobin is as 76.8 to 100.

The amount of fibrinogen is relatively small, and as compared with the mother's blood is as 2 to 7.

Fœtal blood is not well qualified for the method of quantitative analysis by means of washing with salt solution, because many of the elements which are loosely held in the red corpuscles, especially the hæmoglobin, are easily washed out.

The fœtal blood is somewhat richer in sodium and considerably poorer in potassium than is adult blood. The amount of chlorine not combined with sodium and potassium is much less than in adult blood. The sex and weight of the child at the moment of birth do not appear to have any influence upon the quantitative composition of the blood.

Some authors have thought that they found a lessened tendency to coagulation in the blood of the new-born. Krüger found that this tendency existed in the sense that the coagulation occurred slowly. He thought that this was due to the diminished tendency of the leucocytes to undergo retrograde changes. He also found more iron in the blood at birth than after a lapse of fourteen days.

ERYTHROCYTES.—Normally, the red corpuscles in the fœtal blood are nucleated, at least in the early months; they are of the normoblast type (Plate V., 6, facing page 330). After the seventh month they diminish rapidly in number, and give place to the normal red corpuscle. There is some difference of opinion as to the frequency of the occurrence of the normoblasts in the last few months of intra-uterine life, but the best observers seem to agree that they are rather infrequent.

LEUCOCYTES.—Fischl, in a report of four cases, found up to the end of the seventh month very few eosinophiles, after this a large number, and then a diminution towards the end of full term; and the observations of Weiss agree with this. He found little variation in form and in size. There is a low percentage of lymphocytes up to the seventh month, the majority of the corpuscles consisting of the large mononuclear cells (Plate V., 2). After the seventh month, an increase of the former and a diminution of the latter occur as full term is approached. The transitional variety (Plate V., 2*a*) predominates at birth, and later gives place to the polynuclear (Plate V., 3 and 4, facing page 330).

Gundobin found that in the blood of premature infants the lymphocytes (Plate V., 1) were both relatively and absolutely increased, and that it was therefore a younger blood. He found also that the same changes occur in the leucocytes of the premature infant's blood as in that of the infant at full term, only that there is a more rapid diminution up to the third or fourth day, and that the leucocytes remain below the average a longer time, from ten days to three weeks, than they do in the blood at full term. When the development of the infant was slow, its blood was correspondingly slow in development, its red corpuscles were diminished, its hæmoglobin was diminished, there was an increase of leucocytes, and in this way a condition of anæmia with leucocytosis was produced.

Just as I have explained to you that to understand intelligently the iseased conditions of early life you must first acquire a knowledge of the ormal development of the infant and child during the different periods of heir existence, so I wish to impress upon you the importance of knowing hat exists normally in the blood of early life before you can appreciate he abnormal conditions. I shall therefore first describe what is found in he normal infant's and child's blood, comparing it with the adult's blood, nd then point out the variations caused by disease.

NORMAL BLOOD AT BIRTH.—AMOUNT.—Welcker states that the otal amount of blood at birth is one-nineteenth of the body-weight. His pinion is based on the examination of a poorly developed infant, in which he umbilical cord was ligatured immediately at birth.

Schuecking places the amount at one-fifteenth of the body-weight, from n examination of five full-term infants, without expressing the blood rom the placenta, and with immediate ligature of the cord. When the ord was tied later, and the so-called "reserve" blood was expressed from he placenta, the percentage rose to one-ninth. In adults the relation of he blood to the body-weight is stated to be one-thirteenth. All authors gree that there is a temporary gain in the amount of the blood when the ord is tied late.

REACTION.—The reaction of the blood at birth is always alkaline.

COLOR.—The color is found to be darker in the capillaries during the first few days than at any other time.

SPECIFIC GRAVITY.—At birth the specific gravity of the blood is about 1065, and this does not vary for the first few weeks. From this time up to the second year there is a constant diminution, decreasing in boys to as low as 1048 and in girls to 1050. It then gradually rises, till at the end of the first year it has reached the normal average of 1050 to 1058. The specific gravity seems to be uninfluenced by the number of red or white corpuscles, food, rest, exercise, or other causes, but depends directly upon the amount of hæmoglobin. As a whole, the specific gravity is, apart from physiological variations, very constant in the same individual, and remains for weeks and months the same. Hock and Schlesinger place the greatest twenty-four-hour variation at 0.00025. Let me here remind you that the appearance of the child's skin is not an index to the specific gravity of the blood or to the amount of the hæmoglobin. Children often appear anæmic without any especial alteration in either of these conditions.

Specific Gravity of the Blood-Serum.—Hock and Schlesinger's results are the most reliable. They estimated the specific gravity by a method of Hammerschlag's which has not yet been published. They found in young children that the physiological variations were much greater than in adults, and were often between 1026 and 1031. Adults, on the other hand, according to Hammerschlag, showed very little variation, perhaps from 1029 to 1031. Older children resemble adults. The deductions from these investigations seem to be, that any marked change in the specific gravity either of the blood or of the serum, aside from the action of drugs on the hæmoglobin, denotes a marked change in the whole organism and in the functions of the various organs.

HÆMOGLOBIN.—The hæmoglobin is found to be less firmly bound to the red corpuscle in the infant at term than it is in adults. It is, however, proportionately greater at birth than in adult life. The hæmoglobin, like the specific gravity, which, as I have told you, seems to be dependent upon it, reaches its maximum at birth. Starting at 100 or 104, it falls rapidly to its minimum in the first three weeks of life. (Hock, Schlesinger, Widowitz, Schmaltz, and Hammerschlag.) The lowest percentage that you will find varies from 55 to 96.5 per cent. From two weeks to six months it remains about the same, and then rises slowly.

ERYTHROCYTES (Plate V., 5, facing page 330).—So far we have been studying the blood as a whole. I shall now direct your attention to its more minute composition. Here, under the microscope, is a specimen showing the normal red corpuscles, as seen through a Leitz oil-immersion $\frac{1}{12}$ and an ocular No. 3.

All authors agree that there is a large number of these erythrocytes at birth, and also that an increase occurs in the first twenty-four hours. As regards the actual number in a cubic millimetre of blood there is much diversity of opinion. From the second day the erythrocytes begin normally to diminish, and fall eventually, according to Lépine, Gerard, and Schlemmer, to 5,000,000. These authors found the loss of body-weight during the first twenty-four hours to be accompanied by an increase of the red corpuscles. Lépine attributed the variations to changes in the blood-plasma, and not to a new formation or to a degeneration of the corpuscles.

Hayem always found a larger number at the moment of birth than in the mother's blood, and gives as an average 5,350,000. He states that tying the cord influenced the number, the average being perhaps half a million higher when the cord was tied late. This effect is temporary, however, as is shown by Schiff's experiments. When the infant's weight was lowest, Hayem found that the count reached its maximum. From this time a slow but constant diminution took place, and in the second week it was found to be about half a million less than at birth. He thought that the increase was due not alone to the loss of fluid, but also to the increased formation, because in maximal counts he found the corpuscles smaller, and considered them, therefore, younger.

Stierlin examined older children, and found the erythrocytes to be very similar to those found in adults. There appeared to be more red corpuscles in each cubic millimetre of the blood of boys than in that of girls, about 350,000 more.

Schiff seems to have done the most thorough work on the estimation of the erythrocytes. He calculated the total blood quantity as well as he was able, and based his results on this. He found the highest count on the first day of life, and a diminution in the next few days, with hourly variations;

but each succeeding day the count was lower. This shows the value of conducting experiments on the blood at the same hour of the day. The increase after birth is only seeming, according to Schiff, and is due to a loss of fluid and consequent concentration of the blood, because after the first feeding the counts are lower. He agrees with Lépine, except that he thinks the changes in the blood are not due to gain or loss in weight, but to the fluid taken into the system, and he showed that in a fasting infant, after several hours, there was an increase in the red corpuscles. Schiff places the average at birth at 5,800,000, and is unable to observe any influence of sex at this age.

Leucocytes.—The white corpuscles are more numerous at birth than in adults or in young children. As I have already stated regarding the red corpuscles, the counts of the white corpuscles vary according to the examiner; so that our knowledge of the exact figures which should represent these counts is by no means settled.

Schiff found the highest counts, in the first twenty-four hours following the first feeding, to be from 26,000 to 36,000 in a cubic millimetre. He never found the rapid diminution noted by Hayem at the end of a physiological loss of weight, nor did he find so low a count as 4000 to 6000 at this time. He called attention to the daily variation in consequence of digestion, which is of the utmost importance to bear in mind when examining pathological blood. He estimated that from the twelfth to the eighteenth day the average figures were from 12,000 to 13,000, and for older children 10,000.

Hayem found that in the first few days of life there were three or four times more leucocytes than in adult blood, and his estimated average was 18,000. His average of 5000 for adult blood is rather low. He found that this average of 18,000 persisted until the physiological loss of weight had ended, when it was rapidly reduced to from 14,000 to 12,000. At the time when the infant begins to gain in weight the count rises to from 19,000 to 23,000, and there remains constant for a few days. The daily variations in the early days of life are more marked than in adults.

Gundobin, in an examination of infants from ten days to a year old, found an average of 12,900, the variations being from 10,000 to 14,000. The adult variation he estimates to be from 7000 to 10,000.

Bouchut and Dubrisay found the average of a number of counts in children from two to fifteen years of age to be 6700.

Denis examined the blood of artificially fed and breast-fed infants. He found the diminution of the leucocytes occurring on the fourth day, and that it took place more rapidly in the breast-fed than in the artificially fed. He observed that the counts in infants were higher than in adults, and that an increase of the white corpuscles occurred in poorly nourished infants. He refers to the influence of food on the counts, and states that soon after feeding an increase in the leucocytes was observed.

Anna Bayer, a pupil of Denis, estimates the leucocytes of new-born infants and young children as between 16,000 and 23,000. In later child hood, up to the sixth year, she placed them at from 9000 to 10,000.

The cause of the leucocytosis of the new-born, according to Gundobi is a predominance of the "over-ripe" elements (neutrophiles, Plate V., 3 these cells forming from 60 to 80 per cent. of the total increase. H thinks this is due to a diminished activity of the retrograde metamorphos From the second day the process is more rapid, and from the seventh da to the tenth day the white corpuscles have reached their normal conditio which is found to be due to an absolute and relative increase of lymph cytes,—that is, the blood becomes younger.

Gundobin opposes Lépine's theory that the leucocytosis of the new-bor infant is due to a concentration of the blood, and also Schiff's theory th there is an increased flow of lymph from the tissues into the blood whe the child is hungry.

A fair average of the leucocytes in the blood of infants from six month to a year old is from 10,000 to 12,000. After the first two or three week and up to six months, it is found to be from 12,000 to 14,000. Combinin these observations as well as we can, I find the average figures regardin the number of the erythrocytes and leucocytes at different ages to be as have represented in this table (Table 85). These figures assume a loss o weight for forty-eight hours and then a gradual gain.

TABLE 85. (R. C. Cabot.)

Normal Average Number of Blood-Corpuscles at Different Ages in Cases where there wa Loss of Weight in the First Forty-Eight Hours.

Age.	Erythrocytes.	Leucocytes.
At birth	5,900,000	21,000 (26,000 to 36,000 after first feeding.)
End of 1st day	7–8,800,000	24,000
" 2d "	generally increased.	30,000
" 4th "	6,000,000	20,000
" 7th "	5,000,000	15,000
10th day		10–14,000
12th to 18th day		12,000
1st year		10,000
6th year and upwards		7,500

After a meal 30,000 leucocytes is never an abnormal count in infan under two years.

Nucleated Red Corpuscles (Plate V., 6, facing page 330).—Neuma and Kölliker found large numbers of nucleated red corpuscles at birth (er throblasts of Ehrlich, the "cellules rouges" of the French). Hayem, Lu Loos, Fischl, and other authors did not find these numerous erythrobla and say that they are found in large numbers in fœtal life only. The f which are found are usually of the normoblast type. After six months th are rarely or never found normally. They are considered to be the res of delayed function. Pathologically, their presence may be of considera importance. Ehrlich divided them into three kinds, depending on their size and on the staining property of the nucleus: (1) the *normoblast*, which is the size of a normal red corpuscle, and has a small, deeply-stained nucleus; (2) the *gigantoblast* or *megaloblast*, which is very much larger, perhaps three or four times, than the red corpuscle, and has a large, pale, or fragmented nucleus; and (3) the *microblast* or *poikiloblast*, which is a very rare form, and corresponds to the microcyte in size. The normoblast is the type commonly found.

LEUCOCYTES.—Five varieties of white corpuscles are found normally in human blood, and they have been classified in various ways. Ehrlich's classification is as follows:

1. **Small Mononuclear, or Lymphocytes** (Plate V., 1, facing page 330).—These are small, round in shape, about the size of a red corpuscle, and contain a large round nucleus, which usually takes an intense stain with all basic stains. The protoplasm is a narrow band encircling the nucleus, and at times is so narrow as not to be visible. It sometimes stains faintly with eosin, and sometimes does not stain at all. These lymphocytes often vary much in size, and at times are so large as to be indistinguishable from the large mononuclear variety.

2. **Large Mononuclear** (Plate V., 2, facing page 330).—These cells are considerably larger than the lymphocytes, often two or three times. They have a large oval or ovoid nucleus, which stains faintly, and a large amount of almost colorless protoplasm surrounding the nucleus and giving the cell very much the appearance of a vacuole. The protoplasm stains very faintly with eosin.

3. **Transitional Forms** (Plate V., 2*a*, facing page 330).—Among the large mononuclear cells there is found at times a transitional variety. Similar in other respects to the large mononuclear corpuscles, they differ in that the nucleus is undergoing transition. This is shown by a more or less deep indentation, which gives to the nucleus a saddle-bag or horseshoe shape.

4. **Polynuclear,** or more properly **Polymorphnuclear, Neutrophiles** (Plate V., 3, facing page 330).—These corpuscles are somewhat smaller than the large mononuclear, more round in shape, and with a peculiar polymorphous deeply-staining nucleus. At times the nuclei resemble the letters S, V, Z, E. When stained the nucleus often appears segmented; hence the name "polynuclear." The protoplasm is acidophilic; that is, it has an affinity for acid stains, and is filled more or less completely with fine granules, which are not very refractive and are stained by neutral stains; hence the name "neutrophile." These corpuscles are more contractile than the other varieties, and are the ones most frequently found in pus, as they have the faculty of passing easily through the walls of the vessels by means of their mobility.

The last three named varieties are generally considered to be the same corpuscle undergoing metamorphosis, during which process the protoplasm becomes opaque and is changed from basophilic to acidophilic. The opacity is due to the fine neutrophilic granules which have appeared in the protoplasm. This change is supposed to occur in the blood, and, according to Ehrlich, is due to some nutrient material present there. Possibly the corpuscles are better nourished in the blood than in the organs which are supposed to produce them.

The transitional conditions are supposed by Uskow to be either a degenerative or a ripening process, of which the lymphocytes represent the "young" or "unripe cell," the large mononuclear the "ripe" cell, and the polynuclear cells or neutrophiles the "old" or "over-ripe" cells.

5. **Myelocytes,** or large **Mononuclear Neutrophiles** (Plate V., 8*a*, facing page 330).—These are large round or ovoid neutrophilic cells which probably originate in the bone-marrow. They contain one, very seldom two, large round or slightly bent nuclei, which stain blue. The body of the cell, which forms a ring around the nucleus, is crowded with a quantity of fine neutrophilic granules. Myelocytes are rare in normal blood, but are much increased in some of the pathological states. (Klein.)

6. **Polynuclear Eosinophiles** (Plate V., 4, facing page 330).—These cells are generally about the size of neutrophiles, and have a nucleus, staining deeply, which is similar to that of the neutrophile in shape and in its apparent segmentation. The protoplasm is acidophilic, and is more or less completely filled with coarse generally round or ovoid highly refractive granules, which have an affinity for acid stains. The origin, significance, and composition of these cells have caused more discussion and research than those of any of the other varieties. Ehrlich states that the granules are not albuminoid, and concludes that their composition is of a complex nature. Weiss maintains that they are albuminoid, and bases his assertion on the results of micro-chemical experiments performed by himself and others. Ehrlich thought at one time that the only place of origin for these cells was the bone-marrow, and that their occurrence in the blood in large numbers signified chronic changes in the blood-making organs. Since then it has been satisfactorily demonstrated that they occur—pathologically, at any rate—in various secretions. Neusser found them in large numbers in the blood in certain skin affections. According to Canon, who verified this, the number was less dependent upon the disease itself than upon the amount of surface involved. They are frequent in the blood and bronchial secretion in asthma; also in the prostatic secretion under certain circumstances, and in the urine of septic nephritis. The number is normally very variable in the blood of infants and children, so that they have not the significance that they may have in the blood of adults. Weiss considers their increase as occurring entirely independently of the other leucocytes, and for this reason their comparative percentage has a doubtful value.

7. **Mononuclear Eosinophiles** (Plate V., 8*b*, facing page 330).—Very similar to the myelocytes are the so-called eosinophilic myelocytes. They differ from them in having in the cell protoplasm eosinophilic granules in place of neutrophilic granules.

8. **Broken Cells.**—In addition to the above-mentioned varieties, we find in some conditions of the blood polynuclear cells that have lost their regular outline and appear as though burst, with their granules scattered outside the cell-body. Their cause and significance are still matters of dispute.

GRANULES.—By reason of their affinity for certain staining reagents, Ehrlich was enabled to differentiate seven varieties of granules occurring in the cells of the blood, five of which occur in the human blood. The staining fluids are divided into acid, basic, and neutral stains. The latter are obtained by combining a basic with an acid stain in certain proportions.

This affinity for certain staining agents or groups of staining agents Ehrlich terms their *elective power;* the degree of intensity with which they stain he terms their *tinctorial power*. He considers that but one kind of granule ever occurs in the same cell, and then only in the protoplasm. He attributed the former of these phenomena to a specific secretory function of the protoplasm, and hence the term specific granulations which he applied to these granules. These granules differ in their reaction to staining fluids, in size, in shape, and in solubility. They are usually more or less round. Their size is about the same in each variety, but is markedly different in different varieties, the eosinophiles being the largest.

The lymph-glands do not produce any cells containing granules, and Ehrlich believes that each variety must have its own peculiar protoplasm. He looks upon the granules as the product of cell activity, which is sometimes a function of reserve material, and at other times is a process of elimination. He found their composition to be complex.

Ehrlich classified these five varieties of granules that are found in the white cells in human blood as follows:

1. **α Granules.**—*Eosinophilic.*—Stained by all acid stains. They are neither fat nor albumin. (This has since been denied by Weiss.) These granules are coarse, round, and highly refractive. The leucocytes containing them are present normally in the blood in small numbers.

2. **β Granules.**—These are fine round granules stained by acid and basic stains (amphophilic), and occur in the medullary cavity of human bones, and in many of the leucocytes of rabbits and guinea-pigs.

3. **γ Granules.**—This variety is basophilic, and represents the German "mastzellen-körnung." They are moderately coarse, round, and not very refractive. They are said by Ehrlich not to occur normally in the blood. Other authors, however, have found them in small numbers. They are found in bone-marrow and connective tissue. They also occur pathologically in the blood of leucæmia in varying numbers, and occupy more or less of the protoplasm of the large mononuclear cells. They are thought by most investigators to be pathognomonic of leucæmia when found in large numbers.

4. **δ Granules.**—These are basophilic, and are found in the mononuclear elements of human blood. The difference between this variety and

the "mastzellen" granules, both of which are basophilic, has not yet been described by Ehrlich.

5. ε **Granules.**—*Neutrophiles.*—These granules are stained by neutral stains, are very fine, are not refractive, and usually fill the protoplasm more or less completely of the polynuclear leucocytes with the exception of the eosinophiles. The nature of these granules is not known. This affinity for staining reagents is more than superficial, as a chemical reaction is supposed to occur. Weiss doubts whether the granules in the cells are the result of a specific cell function. The living cell is a very complex substance, with varied properties, morphological and chemical, and the granules may be formed in a number of ways and from chemically different substances to serve various purposes.

Percentages of Various Leucocytes in Normal Blood.—Estimates have been made by many observers of the percentages of the different leucocytes in normal blood. It is sufficient to note that the blood of infants differs from that of adults in that the blood of the latter contains from 60 to 75 per cent. of neutrophiles, the remaining 40 to 25 per cent. being made up of mononuclear cells, of which about 28 per cent. are lymphocytes; while in the infant the mononuclear cells, which include the lymphocytes and the large mononuclear cells, form the majority of the cells, perhaps two-thirds or three-quarters, and in very young infants the percentage is even higher. The following table (Table 86) illustrates what I have just said:

TABLE 86.

	Adults.	Infants.
Small mononuclear	24 to 30 per cent.	50 to 70 per cent.
Large mononuclear	3 to 6 "	6 to 14 "
Neutrophiles	60 to 75 "	28 to 40 "
Eosinophiles	1 to 2 "	½ to 10 "

Gundobin finds very little change from the above figures until the beginning of the third year, when the blood resembles more that of adults, the neutrophiles and mononuclear elements being present in about equal proportions. In children of eight or ten years he found very little difference from the blood of adults.

His conclusions are that infants' blood is (1) richer in white corpuscles; (2) richer in young form elements, the absolute and relative counts of the lymphocytes being three times as large as in the blood of adults, while the "over-ripe" elements, or neutrophiles, are half as many; (3) in infants the white corpuscles remain relatively longer in the "unripe" and in adults in the "over-ripe" stage.

Experiments have also been made to determine the constancy of the absolute number of white corpuscles and their relative percentages in healthy infants under different conditions. It is found that the longer the interval between the feedings the more marked is the increase in the white corpuscles during digestion. After two or three hours' fasting there is not much change in the blood; after five hours' interval there is always a leucocytosis averaging from two to four thousand. The cause is to be found in an absolute and relative increase of neutrophiles, the number of which corresponds to the increase. Morphologically, therefore, the blood is older.

The time of day, variations in temperature, and physical exertion seem to have no effect upon the number of the white corpuscles. Most authors place the normal percentage of eosinophiles between 2 and 10 per cent. It is safe to say that they may be somewhat increased, even considerably, in infants' blood without having the same significance as in adults' blood.

It may be of value to speak of certain sources of error in computing percentages which Weiss mentioned,—namely, that it is not enough to count the varieties of corpuscles of each kind, and thus estimate the percentage of each, because you are then dealing with comparative and not absolute figures. It will readily be seen that if a leucocytosis is present, and one variety of corpuscle is increased, it must make the others appear relatively diminished, whereas they may be absolutely normal or even increased. The following table of Weiss shows this plainly:

TABLE 87.

Counts.	Eosinophiles.	Total Leucocytes.	Percentages.
1.	300	10,000	3+
2.	300	20,000	1.5+
3.	600	40,000	1.5+
4.	600	10,000	6+

This table shows how little reliance can be placed on a comparative count in a given case, for the percentages show a marked variation without being any index as to whether an actual change in the number of eosinophiles has occurred or not. In the first count they are normal; in the second they are absolutely normal and relatively diminished; in the third they are absolutely increased and relatively diminished; and in the fourth they are absolutely and relatively increased. This explains to some extent the contradictory percentages which have been reported. In order, therefore, to estimate an absolute increase of any variety a possibly concurrent leucocytosis must be taken into account. Another source of error mentioned by the same author lies in the staining fluid. Where acid and alkaline solutions are combined for staining purposes, it is possible, accidentally, for the alkaline solution to be so strong that not only do the coarse granules stain, but also the fine neutrophiles. Both have a red color, and a person depending on the color alone might mistake the neutrophiles for eosinophiles. The size and refraction of the granules should therefore be observed in every case.

LECTURE XV.

THE PATHOLOGY OF THE BLOOD IN EARLY LIFE.

PREMATURE INFANTS — NEW-BORN — LEUCOCYTOSIS — LEUCÆMIA—OLIGOCYTHÆMIA—PRIMARY ANÆMIAS—CHLOROSIS—ANÆMIA PROGRESSIVA PERNICIOSA—ANÆMIA INFANTUM PSEUDO-LEUKÆMICA VON JAKSCH — SECONDARY ANÆMIAS—TREATMENT OF DISEASES OF THE BLOOD—CONGENITAL SYPHILIS—RHACHITIS.

FROM what I have in the previous lecture described to you concerning the elements of the normal blood, you will now be able to appreciate the conditions which occur in various diseases. So far as our present knowledge of the blood in early life goes, its general diseases may be disposed of quite briefly, only a few characteristic conditions from a diagnostic standpoint having as yet been discovered. Traces, however, of diseases which have caused changes in the blood are often observed for a long time, and may afford an estimate of the patient's condition. For instance, the hæmoglobin is often comparatively low after the red corpuscles have reached their normal number, and thus affords an index to the rate of improvement.

As I shall frequently refer to the expert work which has been done by Dr. Richard C. Cabot on this subject, I wish to acknowledge my appreciation of the careful manner in which he has verified my cases.

I also wish to speak of the great assistance which I have received from Dr. John Dane, through his laborious work on and masterly grasp of this special branch of diagnostic medicine.

PREMATURE INFANTS.—You may remember that in my lecture on Development I explained to you that premature infantile conditions are in one sense—namely, the developmental—closely allied to the pathological. It therefore seems proper to speak of the premature infant's blood before considering the abnormal conditions of the blood in early life.

Here in Ward R is an infant (Case 104) premature at about the eighth month.

BLOOD EXAMINATION 1. (Whitney and Wentworth.)

Premature Infant, 8 months, thriving.

	Feb. 6.	Feb. 9.	Feb. 15.	Feb. 27.
Erythrocytes	5,118,750	5,023,750	5,072,500	4,500,000
	(a few nucleated.)			
Hæmoglobin	101 per cent.	98 per cent.	101 per cent.	91 per cent.
Leucocytes	16,500	15,500	24,000	18,000
Small mononuclear .	19 per cent.	47 per cent.	61 per cent.	39 per cent.
Large " .	7 "	33 "	19 "	32 "
Polynuclear	74 "	15 "	20 "	19 "
Eosinophiles	0	5 "	?	10 "

You will notice the high relative percentage of the lymphocytes, which ou would expect in the early days of life. The percentage of polynuclear ells was extraordinary on February 6, especially when compared with the ount three days later. For an infant, this was a very marked neutrophilic leucocytosis, for which no cause could be ascertained.

THE NEW-BORN.—Gundobin thinks that it is proper to speak of the new-born infant's blood as pathological. He considers that the morphological changes which occur in the blood during the first few days of life are not accounted for by the ordinary physiological conditions; that the variations in the weight of the new-born and the quantitative and qualitative changes in the form-elements of the blood correspond, so far as they are caused by the same processes; that the probable cause of the morphological and the chemical differences between the new-born infant's blood and that of the nursing infant is to be found in the deviation from a normal tissue metamorphosis occurring in the new-born; finally, that the organism of the new-born infant shows very little power of resistance to pathological processes, and that the examination of the blood after Ehrlich's method shows better the length of the period of development usually designated by the term "new-born" than any other means.

I have here another infant (Case 114), fourteen months old, to show you.

It is apparently perfectly healthy, but a physical examination shows that its growth has een retarded, and that it really only represents the development of an infant about seven onths old, so far as its weight, teeth, and functions are concerned. The blood examina- n presents characteristics which correspond to the stage of its development rather than its age. Its blood therefore can be considered abnormal, but illustrative of an early ge of development.

BLOOD EXAMINATION 2. (Whitney and Wentworth.)

Infant 14 months. Development corresponds to 7 months.

Erythrocytes	4,928,750
Hæmoglobin	45 per cent.
Leucocytes	23,000
Small mononuclear	66 per cent.
Large "	17 "
Polynuclear	16 "
Eosinophiles	1 "

With a few exceptions, such as malaria, leucæmia, chlorosis, anæmia ogressiva perniciosa, and anæmia infantum pseudo-leukæmica von Jaksch, is hardly wise at present to attempt to classify changes in the elements of the blood, according to their origin, into primary and secondary seases. I shall therefore merely explain to you exactly what was found in y examinations of the blood in various diseases, with the hope that this rk may aid you in understanding the far more extensive investigations hich are being made in Europe.

There are certain changes in the blood which occur under varying conditions, both physiological and pathological. They are quite commonly met, and are found in many different diseases, whether the diseases are primary in the blood itself or are merely represented secondarily by the changes in the blood. These general changes may be divided into two broad classes, (1) leucocytosis and (2) oligocythæmia, and I think that you will better understand what I shall say concerning the blood in each disease if I first describe these general classes, with, so far as is possible, the especial diseases which belong to them.

LEUCOCYTOSIS.—The best definition of leucocytosis that I can give you is one that has been formulated by Dr. Richard C. Cabot. He says that "leucocytosis is the presence in the blood of an increased number of white cells of the same varieties morphologically as those in normal blood, a plurality, and generally an overwhelming plurality, being polynuclear." Physiologically, we find a leucocytosis after the ingestion of any proteid food. It is at its height about two hours after a meal, when the total number of leucocytes may be as great as from 13,000 to 30,000, according to the age of the child. Pathologically, a leucocytosis follows a considerable number of diseases, and seems in a general way to depend upon the amount of local reaction to which the disease gives rise. We find a pronounced leucocytosis in most fevers and in most septic processes. Von Limbeck, in his article on inflammatory leucocytosis, says that a leucocytosis not only accompanies an exudation, but "corresponds in degree to the number of cells in the exudation; that is, whether it is serous or purulent." Of the pyogenic bacteria he says that the staphylococcus seemed most productive of leucocytosis, especially the pyogenes aureus. It is not known why this should be so. In these cases the increase is almost wholly composed of the polynuclear neutrophiles, which may make up from 90 to 98 per cent. of the entire leucocyte count.

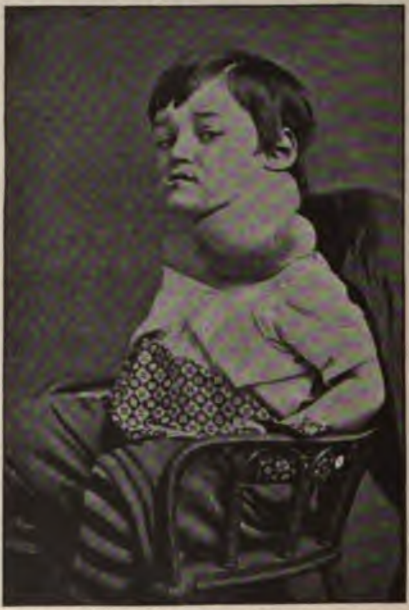
Although I shall have occasion, in showing you cases in the wards, to speak in detail of many of the diseases that give rise to a leucocytosis, I will now briefly enumerate them. Pneumonia shows generally a leucocytosis, and especially if the case is to have a favorable termination. In pneumonia the large increase in the number of leucocytes seems to follow closely the course of the pathological process, and the "blood crisis" is found to anticipate the "temperature crisis" by some hours. Pericarditis and endocarditis, advanced phthisis, pleuritis, erysipelas, acute rheumatism, purulent meningitis, pharyngitis, diphtheria, septicæmia, osteo-myelitis, scarlet fever, variola, some profound anæmias, whether primary or secondary, leucæmia, hemorrhage, malignant new growths, abscess of any kind, including appendicitis, and many skin diseases, are among the others that show leucocytosis. The diseases in which the leucocytes are approximately normal are malaria, tubercular meningitis, tubercular and serous peritonitis, influenza, measles, typhoid fever, and pulmonary phthisis unless there is a secondary infection by other bacteria. Comparing these two lists, you will see that there are some cases in which the leucocyte count may be of great importance to the physician in making a differential diagnosis. By its aid we may in some cases differentiate scarlet fever from measles, a purulent from a tubercular meningitis, and a beginning pneumonia from a tubercular meningitis or typhoid fever. Lastly, we may by the leucocyte count alone be able to decide between sepsis and malaria in a patient whose only symptoms are malaise and returning chills.

LEUCÆMIA.—The disease called leucæmia sometimes occurs in infancy and childhood. Klebs, von Jaksch, and Sänger describe congenital cases. On the whole, it is a rare disease in infancy, and when it occurs it is probably always a mixed form. A pure myelogenous form of leucæmia is very rare. The etiology of the disease is obscure. Cases have been reported which followed congenital syphilis and rhachitis. It is thought by some to be an infectious disease, but the evidence is insufficient. Von Limbeck thinks that it is a disease of the lymphatic system. Others say that any anæmia or Hodgkin's disease may progress to leucæmia under certain circumstances, as may also anæmia infantum pseudo-leukæmica. A number of cases are apparently primary. This is one of the few diseases which can be diagnosticated definitely from the blood-examination alone.

Speaking of the hæmatology of leucæmia, we find that it occurs in two distinct varieties, according as the lymph-glands or the spleen and bone-marrow have been most affected. I will begin with a short description of the latter, the spleno-myelogenous. The first and perhaps the most striking thing that you will notice in examining the blood is the great increase in the leucocytes. Von Jaksch reports a case in an eighteen-months infant where the figures were 1 to 18, and another in which the astonishing ratio of 1 to 2.5 was found. But a leucocytosis alone, even a profound one, does not make a leucæmia; it is the especial kind of leucocyte that you must depend upon, the so-called myelocytes, or "markzellen" of Ehrlich (Plate V., 8*a*, facing page 330). These cells, which are said never to be found in normal blood, are present in this disease in varying proportions up to 20 per cent. of the entire leucocyte count, or even higher. Associated with these there may be the eosinophilic markzellen (Plate V., 8*b*, facing page 330), which Rieder and others have held to be equally diagnostic; also the dwarf eosinophiles (Plate V., 4*b*, facing page 330), which differ only in size from the ordinary polynuclear eosinophilic cells. These three varieties of elements are found in great numbers in the marrow of the long bones, and thence are supposed to get into the blood. Of the forms of leucocytes with which you are familiar in normal blood you may find the polynuclear eosinophiles increased. Their variation was thought at one time to be of diagnostic importance, but it is now considered to be of no value. The polynuclear neutrophiles are normal, or frequently are relatively diminished and vary in size more than usual. The lymphocytes in pure spleno-myelogenous leucæmia are always diminished. Karyokinesis is marked in the leucocytes. The erythrocytes you will find reduced, but never so much so as in the primary or even the secondary anæmias. The percentage of hæmoglobin decreases proportionately with the number of red globules or slightly in advance of it. Lastly, nucleated red cells appear, mostly normoblasts, though megaloblasts are not very rare in children.

Turning now to the second or lymphatic variety, the blood-picture is very different, though quite as distinctive. In this the leucocytes are never so greatly increased, and seldom exceed the proportion of 1 to 15. The diagnosis rests upon the wonderful relative increase of the lymphocytes. These, as you know, should make 25 to 60 per cent. of the entire leucocyte count, according to the age of the child; but in this form of leucæmia 90 per cent. and over has been reported, even in adults. Relative to these, all the other leucocytes are diminished. The special cells, which I have told you are found in varying proportions in the spleno-myelogenous form, are rare; perhaps their occasional presence may be explained by a slight involvement of the bone-marrow, even in the purest lymphatic form.

CASE 115. (Damon and Cheever.)



Warren Museum, Harvard University. Lymphatic leucæmia. Boy, 8 years old.

This case (Case 115), the history and picture of which I have brought from the Warren Museum to show you, is that of a boy eight years old, reported by Dr. H. F. Damon. He had shown symptoms of enlarged cervical glands for a year, but his general health had been fairly good. He never had any pain in the glands, and was well enough to go to school. An examination of the blood showed that the relation of the leucocytes to the erythrocytes varied from 1 to 50 to 1 to 10.

Two months previous to the time when this picture was taken, the tumor had increased rapidly, and you see it is of considerable size, involving the entire left side of the neck. The boy complained at this time of headache, which probably was caused by pressure on the recurrent vessels of the neck. The mass of impacted glands had begun evidently to press upon the trachea, and on exertion the respiration was slightly interfered with. On palpation the tumor was found to consist of many lobules, which were to some extent movable, and appeared to be made up of an enlarged chain of lymphatic glands. It extended from near the middle line of the neck in front, back upon the edge of the trapezius on the left side, and above from the lobe of the ear and angle and body of the lower jaw down to and beneath the clavicle. The left shoulder was depressed by it. A number of enlarged cutaneous veins ran over it in various directions. As the tumor showed no signs of softening, but was steadily enlarging, it was deemed best to attempt its removal. The operation was performed by Professor D. W. Cheever. An incision was made from just below the ear to near the cricoid cartilage, through the skin and platysma, and disclosed a lobulated, hard, glandular mass, lying mainly beneath, and partly behind, the sterno-mastoid muscle. Contrary to expectation, it was found very adherent in all directions, and the lobules were bound together by strong fibrous tissue. Considerable time and care were requisite to divide the adhesions, which were too strong to yield to anything but the edge of the knife. It was found necessary to divide the sterno-mastoid, and to dissect aside the external jugular, which ran, somewhat displaced, over and through the tumor. The lower edge of the tumor extended beneath the clavicle, into and below the subclavian triangle. The base lay over the sheath of the carotid, which was necessarily exposed for about two inches. Continuous dissection was required, even to the last adhesion, for they could nowhere be made to yield.

The boy recovered in a few weeks, a large part of the wound healing by first intention.

The tumor was found to consist of a lobulated mass of hypertrophied lymphatic glands, firmly bound together by investing fibrous tissue.

Two years after the operation the child was alive and fairly well, although the glands on both sides of the neck were again found to be considerably enlarged, as were also those in the axillæ. The further history of the case is unknown, and the report is in many ways unsatisfactory, but the facts as stated are all that I could ascertain about it. There is no doubt in my mind that it was a case of leucæmia.

The second general class, which I have referred to as liable to occur in many diseases, is oligocythæmia.

OLIGOCYTHÆMIA.—The anæmias are of common occurrence in infancy and childhood. Our ordinary methods of examination are evidently insufficient to discover the causes of the anæmia. It seems as if in the future we must direct our attention to other methods of investigation, and especially to the examination of the blood-serum. It may be of interest to refer briefly to Maragliano's recent theory regarding the blood-serum and its action on the corpuscles. Maragliano's researches upon the blood-plasma have tended to show the various relations existing between the organs and the blood. Pronounced local pathological changes influence the composition of the blood-serum, so that in consequence of this the corpuscles later are destroyed. The length of time required to produce this result depends upon their resistance. These observations throw light on a number of clinical results, and on the dependence of the anæmias upon severe pathological disturbances. Maragliano found that the erythrocytes, when pathological conditions were present in the serum, were rapidly destroyed, whereas in healthy serum they remained almost intact. He examined the blood serum in various diseases, as, for instance, the essential anæmias of all grades, carcinoma, saturnismus, spleno-myelogenous and lymphatic leucæmia, purpura, cirrhosis of the liver, nephritis, pneumonia, typhoid fever, erysipelas, and tuberculosis. In all these diseases the serum has a destructive effect on the corpuscles as compared with normal serum, but with quantitative differences depending on two factors: (1) the vulnerability of the red corpuscles, and (2) the destructive power of t serum. If both of these factors work together, the effect produced is e treme. He is very cautious about explaining the cause, but denies a definite relation between the amount of albumin and the destructive pow of the serum, and rather inclines to the belief that the quantity of salts the serum has some influence. While the erythrocytes are being destroy however, the blood-making organs are undergoing an increased functio activity, and producing erythrocytes to supply the loss. Maragliano co cludes that different conditions of the serum produce in the red corpuscl all the appearances of necrobiosis, and can even destroy them. This giv an anatomical, physiological, and pathological basis for our belief in ind pendent diseases of the blood. This theory concerning the blood-serum at least plausible, and until it has been proved incorrect it may be accepte Perhaps this necrobiotic power of the serum, which depends on some pr vious disease, varies in different diseases and in different individuals. Ev if it is present in sufficient amount to cause anæmia in a given case, it ma not do so because of the resistance of the blood-corpuscles to its influenc The variation in these two factors—namely, the resistance of the red corpu cles and the destructive power of the serum—will account for the variatio in the degree of anæmia produced in different individuals having the sa disease.

We know that in distilled water the coloring matter of the red corpu cles is extracted from the stroma, but when a certain percentage of sodiu chloride is added to the water the integrity of the red corpuscles is pr served. That the erythrocytes are not normally destroyed by the serum i which they float seems to depend upon the presence of a sufficient quanti of salts in the serum. A serum in which there is just enough saline matt to preserve the red corpuscles has been called by Hamburger "isotonic But as an isotonic serum would easily lose its protective properties, owing its dilution after each meal, we generally find a higher salt percentage th is necessary to preserve the red corpuscles,—a condition designated by t term "hyperisotonic."

From experiments upon animals it has been proved that the serum po sesses powerful germicidal properties, which are easily destroyed by raisi the blood to a temperature of 55° C. (131° F.) for a short time or by e posing it to light. Still more singular is the fact that not only does t mixing of the serum of one animal with the blood of another of a differe species destroy its germicidal power, but also that the added serum acts a solvent for the red corpuscles and renders the white corpuscles inacti There is considerable reason to believe that immunity from a given disea depends upon the character of the serum; and Klemperer is now carryi on some interesting experiments with a view to producing immunity b serum inoculation.

In regard to what are usually looked upon as primary anæmias, we ca speak of such diseases as chlorosis, pernicious anæmia, and anæmia infantu pseudo-leukæmica (von Jaksch). By far the greater number of anæmias in early life are, however, of secondary origin. Hemorrhage, the acute infectious diseases, syphilis, rhachitis, new growths, intestinal affections, and diseases of the respiratory system, skin, and bones, are the ordinary causes of secondary anæmia. The degree of the anæmia depends upon the individual, upon the severity and length of the disease, and upon other causes which are as yet unknown. The secondary anæmias may be either of a mild or of a severe form, and may be accompanied or not by a greater or less degree of leucocytosis. The mild forms are usually spoken of as *anæmia chronica levis*, while the severe forms may be called *anæmia chronica gravis*. The blood in these cases shows a varying degree of oligocythæmia and oligochromæmia, with or without leucocytosis, and, if severe enough, poikilocytosis, microcytosis, and at times nucleated red corpuscles. The latter are the more frequent the younger the child, and generally occur during the first year. They are never very numerous in these cases, and are, as a rule, of the normoblast type.

PRIMARY ANÆMIAS.—Poor as the classification of the anæmias into primary and secondary may be, it will, I think, keep the subject more clearly before your minds if I follow this very imperfect division, which for purposes of simplicity it has seemed almost necessary to make. I shall therefore speak at once of the anæmias which are supposed to be primary and which I have just enumerated, wishing it, however, to be understood that I use the word *primary* only provisionally until further light is thrown upon this class of diseases.

Chlorosis.—Although it is still a matter of dispute whether chlorosis should be classed as one of the anæmias, it will simplify what I have to say on this subject if I speak of it as such.

Weiss doubts if chlorosis occurs in infants and young children, but the observations of Henoch and others tend to show that it does. The distinguishing characteristic of the disease is the very low percentage of hæmoglobin relatively to the nearly normal number of erythrocytes, which is in marked contrast to that found in other diseases, especially progressive pernicious anæmia. There is very little or no leucocytosis. Considerable variation in the size of the erythrocytes occurs, poikilocytes, microcytes, and macrocytes being often found.

In this connection I will refer you to some interesting work on the intestinal origin of chlorosis which has been done by Dr. Forchheimer, of Cincinnati, and to his original views and new definition of this disease.

This infant (Case 116), eighteen months old, has never had the symptoms of any special disease beyond a pallor of the nails, skin, lips, and mucous membrane of the gums, with loss of appetite and strength. This has lasted for about six months, and does not appear to depend on climate or habitation, as the infant has been during this period in a number of houses, both in the city and at the sea-shore. An examination of the blood showed that it was a case of chlorosis, the erythrocytes being somewhat reduced and there being a decided oligochromæmia.

BLOOD EXAMINATION 3. (Wentworth.)

Erythrocytes	4,427,500.
Hæmoglobin	35 per cent.

(The infant gradually improved under a course of treatment which was larg dietetic.)

The next case (Case 117) which I have to report is that of a female infant, eleven one-half months old. The pallor of the lips, gums, skin, and nails in this case w extreme, and was accompanied by loss of appetite, but no especial emaciation. The blo examination gave the following result:

BLOOD EXAMINATION 4. (Whitney and Wentworth.)

Erythrocytes	4,470,000
Hæmoglobin	30 per cent.
Leucocytes	25,000
Small mononuclear	45 per cent.
Large "	21 "
Polynuclear	30 "
Eosinophiles	3 "

The cause of the chlorosis was apparently arsenical poisoning from wa papers. The chlorosis was always extreme during the nine months of year when the infant was in its winter home, and was unaffected by tre ment, either dietetic or medicinal. During the three summer months t it was away from home it decidedly improved, but it immediately g worse on returning. The papers throughout the house in its winter ho were found to be dangerously arsenical, and on their removal the inf rapidly improved, and in a few weeks regained its healthy color, streng and appetite. If the chlorosis in this case was caused by arsenic, it m of course be classed with the secondary anæmias.

Both these cases illustrate the fact that extreme pallor does not ne sarily indicate a great reduction in the number of the erythrocytes.

The symptoms of this disease as met in infants are progressive los appetite and of strength, and extreme pallor of the skin and of the muc membrane, not accompanied, as a rule, by marked emaciation.

Anæmia Progressiva Perniciosa.—I shall next speak of a v severe form of primary anæmia, the prognosis of which is so serious t it is called *anæmia perniciosa*.

Biermer in 1868 described a disease under the above title, and said t it developed apparently without any cause and by a gradually increasi and constantly progressing anæmia caused death. The pathological and a tomical changes consisted in a great diminution in the amount of blood in the organs, with marked fatty degeneration of the heart, blood-vessels, liv and kidneys. Capillary hemorrhages were frequently found. Since then mass of literature has been accumulating on the subject. Various auth have observed cases in infancy and in childhood. Most of the cases, ho ever, were over a year old, and it is certainly not a common disease of ea childhood. The description of the disease does not differ materially fr that of adults. There is an apparently spontaneous beginning in most cases. Klebs and Frankenhäuser thought that they found certain micrococci in the blood, but this has not been proved. Cases have been recorded which have developed as a result of congenital syphilis, and there are other cases in which the presence of such intestinal parasites as the anchylostoma duodenalis and the bothriocephalus latus have appeared to be followed by it. Cases have also apparently followed repeated hemorrhages. The majority, however, occurred without any discoverable cause.

The blood in pernicious anæmia is thin and light-colored, and all the formed elements are markedly decreased. The enormous diminution of erythrocytes, which is more marked than in any other disease, even in the highest grade of simple secondary anæmia, the relatively high hæmoglobin percentage, due to the large amount of hæmoglobin in each corpuscle, and the presence of megaloblasts in large numbers, are considered to be diagnostic of this disease. Poikilocytosis is usually pronounced. Microcytes and macrocytes are common. There is generally a diminution in the number of leucocytes, the prevailing type being mononuclear, but at times we find a distinct leucocytosis. It has been pointed out by von Jaksch that the degree of leucocytosis is never so great as in anæmia infantum pseudoleukæmica. Eosinophilic cells are, as a rule, present in unusual numbers. Myelocytes in small numbers are not infrequently found. Clinically the disease does not differ from that of the adult. It is the severest type of all the anæmias, and all the cases have proved fatal, except those in which intestinal parasites were found to be the cause and were removed before the disease had become fully established.

This infant (Case 118), six months old, is apparently a case of pernicious anæmia, although the blood examination does not entirely establish the diagnosis. It entered the hospital when it was five and one-half months old, with the history of having been fed on a variety of patent foods from the time of its birth. On physical examination nothing abnormal was found in the abdomen or thorax, and it was not especially atrophic. Its weight was 2841 grammes (6¼ pounds). It now weighs 2915 grammes (6⅜ pounds). Since entering the hospital it has failed to respond to treatment of any kind, whether dietetic or medicinal, and has become more and more anæmic. You will notice the extreme pallor of the mucous membrane of the gums and of the entire skin. The following is the report of the examination of the blood:

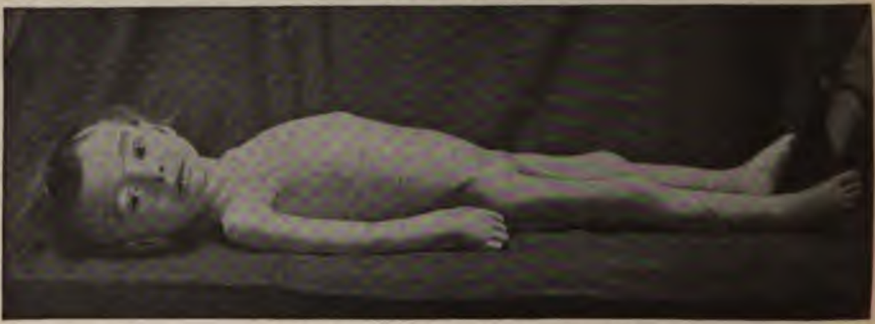
BLOOD EXAMINATION 5. (Whitney and Wentworth.)

Erythrocytes	2,937,500
Hæmoglobin	35 per cent.
Leucocytes	5,500
Small mononuclear	55 per cent.
Large "	10 "
Polynuclear	35 "
Eosinophiles	5 "

The infant continued to fail progressively, and died about two months later. The temperature and pulse were practically normal through the whole course of the disease, and nothing abnormal was at any time detected in the thorax or abdomen. There was no autopsy. A few days before death there appeared extensive hemorrhages under the skin of the abdomen.)

This infant (Case 119), seventeen months old, presents the typical appearance of a pernicious anæmia. On entering the hospital it weighed 5925 grammes (13.03 pounds), and has been progressively losing, until this morning its weight was only 5798 grammes (12.75 pounds). It is emaciated and has had but little appetite, but it has evinced a desire to eat any dirt that it can lay its hands on. A physical examination reveals nothing abnormal, such as thoracic disease, enlarged spleen, or enlarged lymph-glands. The skin has the transparent rather waxy appearance (well represented in Plate V., facing page 330, Anæmia Infantum Pseudo-Leukæmica von Jaksch) which occurs in anæmias of the highest grade.

Case 119.



Anæmia perniciosa. Female, 17 months old.

She is rather apathetic, in fact, almost dull, and can be handled and examined without any apparent discomfort. The result of the blood examination is very significant:

BLOOD EXAMINATION 6. (Wentworth.)

Erythrocytes	1,022,500
Hæmoglobin	17 per cent.
Leucocytes	16,000

The next case (Case 120), an infant nine months old, was seen by me in consultation with Dr. C. P. Putnam, who has kindly provided me with its previous history. The infant was healthy at birth, and up to the time of its present sickness had never had any disease. For several months it had progressively grown pale, and its appetite had decidedly lessened. It had not, however, lost materially in weight, but had grown weak physically, and its mental hebetude had been so noticeable that a suspicion had arisen that it was lacking in cerebral development. On inspection the infant seemed moderately fat, but the muscles were soft, and the skin was of an extremely pale and waxen tinge. It was evidently very weak. On physical examination nothing abnormal was detected about the head, thorax, or abdomen. All the organs seemed to be of natural size. An examination of the blood, made by Dr. Dane, resulted as follows:

BLOOD EXAMINATION 7. (Dane.)

Erythrocytes	1,571,000
Hæmoglobin	22 per cent.
Leucocytes	19,100
Small mononuclear	42 per cent.
Large "	18 "
Polynuclear	40 "
Eosinophiles	0 "

You will notice the great reduction in the number of red corpuscles, the relatively large percentage of hæmoglobin, and the slight increase of white cells. The differential count gives us no special information in regard to the cause or character of the disease. (The child died a few days later without showing any other symptoms.)

The next case is the fourth in which the clinical history and the great oligocythæmia seemed to point towards anæmia perniciosa as the most probable disease, but no elaborate blood examination was made.

A female infant (Case 121) entered my wards at the Children's Hospital on the 16th day of April. She was then nine months old. Nothing abnormal was detected in the lungs or heart, and there was no appreciable enlargement of the liver, spleen, or lymph-glands. The pulse varied from 120 to 140, and the temperature from 36.7° C. (98.06° F.) to 37.8° C. (100.04° F.). The respiration was from 44 to 68. There were hemorrhagic spots on the ankles and head for a few days, but these soon passed away, and nothing abnormal was detected except extreme pallor of the skin, progressive loss of appetite, emaciation, and quick respiration. The erythrocytes were reduced to 785,000, and there was marked poikilocytosis. There was a slight amount of albumin in the urine. The infant grew rapidly worse on April 22, and died in the evening. There was no autopsy.

Anæmia Infantum Pseudo-Leukæmica (von Jaksch).—I shall now speak of a form of chronic primary anæmia where, in order to make a differential diagnosis, we must consider the etiology and physical signs as well as the blood examination.

Von Jaksch, in 1889 and 1890, was the first to describe this disease and give it this title. Since then it has been the subject of much investigation and contention. Von Jaksch based his diagnosis on the following points: that it was a disease of infancy, characterized by marked oligocythæmia, oligochromæmia, considerable permanent leucocytosis, marked splenic enlargement, at times enlarged lymph-glands, only moderate or slight enlargement of the liver, and clinically to be differentiated from leucæmia by the disproportion existing between the size of the liver and the spleen. The more favorable prognosis is also an aid in the diagnosis. About the same time Hayem described a similar disease in a child, and noted the presence of numerous nucleated red corpuscles. Von Jaksch had noticed them, but had mistaken them for leucocytes having erythrocytes inside of them. Hayem noted especially that many of the nucleated red corpuscles were undergoing mitosis. This had never been observed before in the circulating blood.

Luzet verified Hayem's observations. He described this as a disease of early infancy, and emphasized the chronic course, the intense anæmia, and the large size of the spleen and the liver without enlargement of the lymph-glands. He only found a slight leucocytosis, in which the eosinophiles were quite numerous. He considers the large number of nucleated red corpuscles, many of them showing mitosis, as especially important for diagnosis. This condition he has not found so marked in any other disease of the blood. He considers this one of the rare affections of infancy, as according to his statistics it was met only once in fifteen hundred cases of anæmia, and he thinks that it does not occur after two years of age. The effects of sex, temperament, habitation, heredity, and climate are not known.

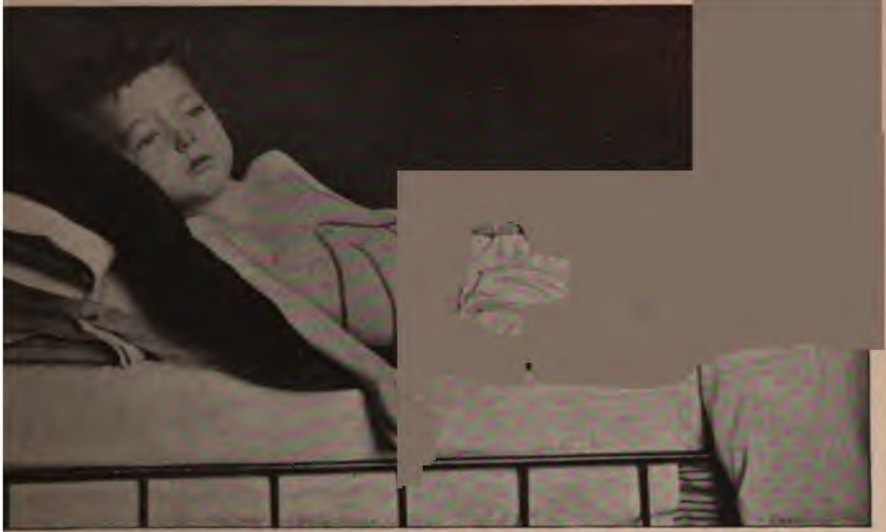
He thinks that rhachitis and syphilis, which at times produce anæmia, with enlarged spleen, do not cause anæmia infantum pseudo-leukæmica. Loos, Weiss, Somma, and others have written a great deal about this disease. Some of them consider it an infectious disease. Most of them deny that it has any connection with malaria, syphilis, and the digestive disturbances, and only occasionally mention its connection with rhachitis.

As a result of my investigations of a considerable number of cases of anæmia of every grade in young infants, it seems to me that we have arrived at a degree of knowledge which justifies us in making a diagnosis, in certain cases, of anæmia infantum pseudo-leukæmica von Jaksch, and I shall presently show you some cases illustrative of this disease. We are dealing with a disease of infancy characterized by a chronic course, rather rare occurrence, and etiological obscurity. The clinical symptoms are those of a grave anæmia, with a "waxen" appearance of the skin. The child may be somewhat atrophied, but is often fairly nourished. There is always considerable splenic enlargement, with only moderate or slight enlargement of the liver. The lymph-glands are generally somewhat enlarged, but never form packets. The blood is characterized by marked oligocythæmia and oligochromæmia, together with a leucocytosis which is often considerable. Nucleated red corpuscles of all types are very numerous, and many of them are found to be undergoing mitosis in their nuclei. Poikilocytosis is marked. The polychromatophilic condition of the red corpuscles, mentioned by Alt and Weiss, may occur. The eosinophilic cells vary in number, and at times are much increased. They also vary considerably in size. Occasionally small numbers of the large mononuclear neutrophiles and the eosinophilic "markzellen" of Müller and Rieder are found. (Klein.)

The course of the disease varies. Von Jaksch lays stress on the more favorable prognosis as compared with leucæmia. All of my cases have been fatal without any apparent complication, and even if the disease remains stationary for a time the risk from intercurrent disease is great. Four cases which apparently can be classed as representing this disease have occurred in my practice. The first case (Case 122) you will remember seeing in my wards at the Children's Hospital, and may be described as follows:

A boy, three years of age; had never had any disease, with the exception of a questionable malaria, from which he had entirely recovered two years previously. The father said that since his second year he had looked pale, and that a physician was consulted about him eight months before he entered the hospital. He entered my wards on October 4. The child, as you will remember, was of a waxen color, well shown in Plate V. (facing page 330), and the mucous membrane of the lips and nails was nearly white, with a livid tinge. The skin was almost translucent. There was not much emaciation. The spleen was considerably enlarged, and could easily be felt about two inches below the border of the ribs. The liver was slightly enlarged and could be felt upon palpation. The glands were enlarged the size of peas in the neck, axillæ, and groins. On percussion the heart showed no largement. A loud systolic murmur was heard over all the cardiac orifices. The action the heart was very rapid, but regular. Its impulse was in the fifth interspace inside of e mammary line. Auscultation and percussion of the lungs showed that they were noral, with the exception of some sibilant râles. The respirations were 30 to 44 in a minute.

CASE 122.



næmia infantum pseudo-leukæmica von Jaksch. Male, 3 years old. Lower border of ribs, enlarged liver, and spleen outlined in black.

he temperature at entrance was 38.3° C. (101° F.), and afterwards varied from 39.5° C. 103.8° F.) to about 38.3° C. (101° F.). The pulse varied from 125 to 150. Diarrhœa was resent when the child entered the hospital, and at first there were four to six very offensive movements daily. For three or four days preceding death the movements were more frequent, but were not so offensive, and contained mucus. Vomiting occurred at times. The infant was treated with modified milk, bismuth, and stimulants. An examination of the blood, October 13, resulted as follows:

BLOOD EXAMINATION 8.

Erythrocytes	1,295,000
Hæmoglobin	15 per cent.
Leucocytes	64,500

There were numerous poikilocytes, microcytes, and megalocytes. A number of the corpuscles were pale, and many of them contained very little hæmoglobin.

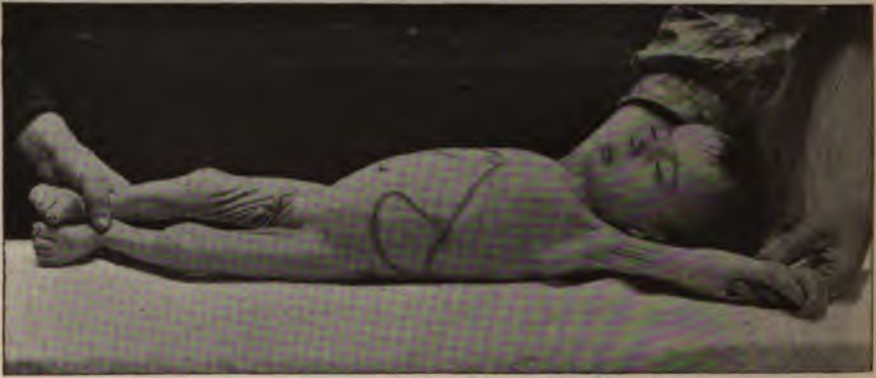
The polymorphous character of the blood was very marked. There were numerous *nucleated red corpuscles* (Plate V., 6, facing page 330), chiefly of the normoblast type, and in many of them the nuclei were undergoing subdivision. The *eosinophiles* were absolutely and relatively increased. None of the "markzellen," characteristic of leucæmia, were present, and the leucocytes were largely of the polynuclear variety. The child died October 20, and a partial autopsy was obtained.

A microscopic examination showed no evidence of leucæmia in the liver, spleen, kidneys, or lymph-glands. There were no evidences of syphilis or rhachitis, nor of any inflammation which could have caused the leucocytosis.

My second case (Case 123) of this disease I happen to have here in the wards to-day to show you. It is a male infant, eleven months old.

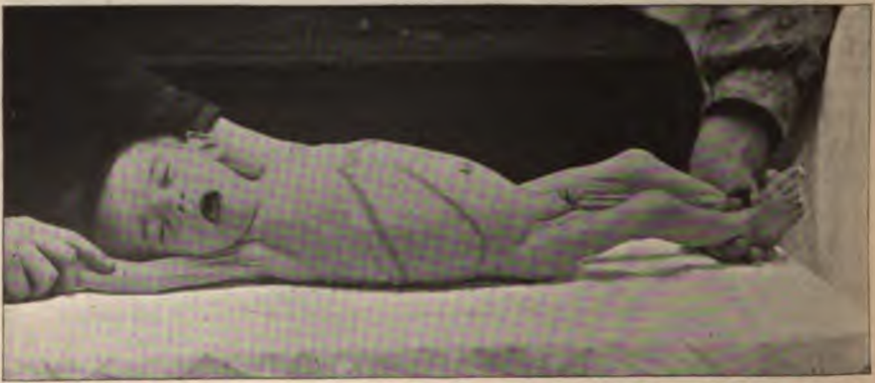
The previous history of the infant has not been ascertained, as the parents have disappeared. You see that he is poorly developed and somewhat emaciated. The skin has a waxen color, and the mucous membrane of the lips and gums is almost colorless. The dejections are frequent; they contain partially digested blood, and have so offensive an odor that disinfectants are constantly required in the room. The cervical lymph-glands and those in the groins are slightly enlarged. I find nothing abnormal on examining the heart and lungs. Palpation of the abdomen reveals a large tumor of firm consistency, beginning under the lower border of the ribs in the left axillary line, and extending towards and considerably below the umbilicus. This tumor is evidently the spleen, and you see I have outlined its edge in black. On the right side of the abdomen the edge of the liver can be felt just below the ribs. I have outlined this edge in black. There appears to be no tenderness of the bones or enlargement of the epiphyses such as would occur in rhachitis, and there is no evidence of syphilis. The following examination of the blood has just been made by Dr. Wentworth:

CASE 123.



Anæmia infantum pseudo-leukæmica von Jaksch. Male, 11 months old. Left lower border of ribs, ensiform cartilage, and enlarged spleen marked in black.

CASE 123.



Anæmia infantum pseudo-leukæmica von Jaksch. Male, 11 months old, crying. Right lower border of ribs, ensiform cartilage, and enlarged liver marked in black.

BLOOD EXAMINATION 9. (Wentworth.)

Erythrocytes	1,311,250
Hæmoglobin	20 per cent.
Leucocytes	116,500
Small mononuclear	46 per cent.
Large "	34 "
Polynuclear	16 "
Eosinophiles	4 "

A drop of the blood, you see, is watery and of a pale red color, but the corpuscles themselves you will observe on this slide under the microscope are not markedly pale. The erythrocytes vary much in size and shape, *poikilocytes*, *microcytes*, and *macrocytes* all being present. In fact, the *polymorphous* character of the blood is very pronounced, the normoblast type of the erythrocytes predominating. Mitoses are very frequent, and show all varieties of subdivision, many of the erythrocytes having two nuclei, others, being in process of subdivision, showing three and four segments. Many of the nuclei also lie eccentrically in the cells. The leucocytes are very variable in this case, but the mononuclear type prevails. The eosinophiles are relatively and absolutely increased, but are somewhat smaller than usual, and are polynuclear.

(The infant failed rapidly and died. No autopsy was obtained.)

My third case in the series is one in which the blood examination was so unsatisfactory that there is a possibility of my being mistaken in my opinion that it should be placed under the heading of anæmia infantum pseudoleukæmica. It would seem, however, from the history, and from the physical examination, that it can better be considered a case of this disease than of secondary anæmia.

A male infant (Case 124), twelve months old, entered the hospital August 15, with the following history. The mother was healthy, the father was said to be tubercular. There were three other living children said to be healthy, and one child, a boy seven years old, was said to have died from some disease of the brain. The infant was healthy at birth, was nursed by its mother and throve until it was five months old, when patent foods of various kinds were given to it, and it was nursed irregularly. It then began to have digestive disturbances. On examination it presented so typical a picture of the two cases (Cases 122, 123) which I have just described as representing anæmia infantum pseudoleukæmica that I have had this colored sketch made of it. (Plate V., facing page 330.) You will notice the intense pallor of the entire skin, which has the "waxen" color in a pronounced degree. The transparent ears are very noticeable, and, as you see, I have mapped out the border of the enlarged spleen in black. The inguinal glands were slightly enlarged, and the liver was scarcely perceptible beneath the margin of the ribs, but the spleen was enormously enlarged, extending down into the left inguinal region as far as the crest of the ilium. There were no other enlarged glands detected. The abdomen was rather distended, and the infant was not especially emaciated. It had had convulsions from time to time since it was five months old. There was no evidence of rhachitis or of syphilis. An analysis of the mother's milk made on August 25 was as follows:

ANALYSIS 56.

Fat	1.91
Milk-sugar	6.45
Proteids	2.66
Mineral matter	0.17
Total solids	11.19
Water	88.81
	100.00

The examination of the blood gave the following results:

BLOOD EXAMINATION 10. (Whitney and Wentworth.)

I. August 25. II. October 17. III. November 9. IV. December 19.

I.	Erythrocytes	1,585,000
		(All of large size and normal red color.)
	Hæmoglobin	30 per cent.
	Leucocytes . . (The estimate was too doubtful and unsatisfactory to report.)	
	Small mononuclear	61 per cent.
	Large "	23 "
	Polynuclear	14 "
	Eosinophiles	2 "
	Megaloblasts and normoblasts in moderate numbers.	
II.	Erythrocytes	3,215,000
	Hæmoglobin	46 per cent.
III.	Erythrocytes	3,300,000
	Hæmoglobin	45 per cent.
	Leucocytes	
	Small mononuclear	58 "
	Large "	45 "
	Polynuclear	3 "
	Eosinophiles	3 "
IV.	Erythrocytes	3,925,000
	Hæmoglobin	40 per cent.

(Treatment of various kinds, both medicinal and dietetic, appeared to have no effect upon the patient's general condition, and when last heard from it was growing progressively weaker and more anæmic.)

I am fully aware that without the count of the total leucocytes the diagnosis is not proved. There should have been found a decided leucocytosis, which I shall assume to have been the case, as all the other characteristics of the disease were present.

The fourth case which I shall speak of as one of anæmia infantum pseudo-leukæmica occurred some years ago in my practice, and, as no reliable examination of the blood was made, I cannot, of course, accept the diagnosis as proved. It was, however, so interesting that it is well to put it on record, as it may in the future be valuable in comparison with cases having similar clinical symptoms, especially as an autopsy was obtained.

The infant (Case 125), a male, was first seen and examined by me when it was four months old. The parents were healthy, and there was one older child, also healthy. There was no history of hereditary disease in the family. Their home was in a comfortable country house, well built, with good drainage, an unpolluted water-supply, and no arsenic in the papers, curtains, or furniture-coverings. The house was built on a considerable elevation, and was not in a malarial district. The infant was healthy at birth, and weighed 3750 grammes (8¼ pounds); it was nursed for a short time, and was then fed with various artificial foods. It soon began to show digestive disturbance and to grow pale. It lost somewhat in weight, had a gradually lessening appetite, and at times vomited.

On physical examination nothing abnormal was found in the thorax or abdomen. There were no enlarged glands. The symptoms were entirely those of functional indigestion, and under a proper regulation of the diet it improved somewhat for a time, and there was a gain in weight.

Two months later I again saw the infant, and, with the exception that the pallor of the skin had much increased, nothing abnormal was discovered. The infant was brought to the Children's Hospital to be under my care when it was eight months old. I then found that it had a much enlarged spleen. The liver and lymph-glands were not enlarged. There was a slight albuminuria. The erythrocytes were reported to be diminished and the leucocytes increased. Its weight was 6704 grammes (14¾ pounds). A few hemorrhagic spots were reported to have been seen on its legs and thorax before entering the hospital, but they were not present on entrance. The infant began to fail soon after coming to the hospital, and died a week later, the spleen having decreased in size. On the day of its death it became very restless and cried a great deal, putting its hands to its head. Nothing abnormal was found on examination of the ears. Slight œdema was detected at the base of both lungs a few hours before death, and it finally died rather suddenly. The following is the report of the autopsy made by Dr. W. F. Whitney:

The body was that of an apparently well-nourished infant, and externally the only remarkable feature was the extreme pallor.

The lungs were normal and retracted.

The heart was of normal size and shape and without any malformation. A microscopic section showed an occasional granular fibre. The spleen was slightly enlarged, firm, and somewhat pale. Microscopic examination failed to show any deviation from the normal structure. The stomach and intestines presented nothing abnormal. The liver was of normal size, its consistency was firm, and its appearance was marked on section by a pale whitish color, which was everywhere present, and had no relation to any part of the lobules. Microscopic examination showed that the liver-cells were separated by large spaces, looking at first like dilated capillaries, filled with small cells similar to leucocytes. The appearance was very similar to that of a fœtal liver of the fifth month. Chemical tests failed to show the presence of any free iron in the liver-cells. The kidneys and other organs presented nothing abnormal.

Secondary Anæmias.—The secondary anæmias are so numerous and arise from so many different causes that an exhaustive discussion of them would hardly be practicable in a clinical lecture. You must bear in mind what I have already said regarding them: first, that almost every anæmia which we meet with is secondary,—that is, that it arises somewhere outside of the blood-making organs; second, that in almost every disease of any organ a secondary anæmia is liable to arise, and is of a high or a low grade according to the severity of the disease. The changes in the form-elements of the blood which are found in these secondary conditions are simply the constant occurrence of oligocythæmia and oligochromæmia, the presence or absence of leucocytosis, and the absence of the other characteristics which are supposed to belong to chlorosis, anæmia perniciosa, and anæmia infantum pseudo-leukæmica von Jaksch. Where the leucocytosis is great, the anæmia is usually one of the graver forms, and in these grave anæmias the leucocytes are found to vary from 14,000 to 54,000. You may remember this infant (Case 126, page 366), four months old, which I examined before you in the wards of the Infants' Hospital six weeks ago. It represented at that time what I shall later describe to you as a moderate grade of infantile atrophy. It was much emaciated, and the interference with the normal activity of the intestinal absorbents was seriously affecting its nutrition. It was pale, but did not have the "waxen" pallor which I have described in previous cases.

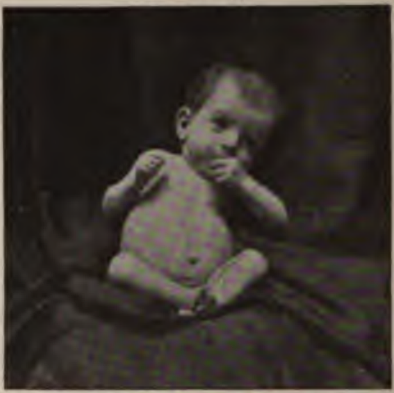
A blood examination at that time gave the following result, which was simply that of a moderate grade of anæmia:

BLOOD EXAMINATION 11. (Wentworth.)

Erythrocytes	3,006,250
Hæmoglobin	40 per cent.
Leucocytes	11,500

The treatment was by food adapted to the disabled condition of the absorbents,—namely, a low percentage of fat with a rather high percentage of sugar and a moderately high percentage of proteids. You see what a marked change has occurred in the appearance of the infant, which has grown fat and is no longer anæmic.

CASE 126.



Male, 4 months old. Infantile atrophy of medium grade, with moderate anæmia.

TREATMENT.—With the exception of the case last spoken of, you will notice that up to the present time I have said nothing whatever as to the treatment of these diseases of the blood. I have done this purposely in order to impress upon you that in infants and young children these diseases depend, so far as I can ascertain, almost entirely upon some interference with the nutrition. It is very rarely that I give drugs in any form in these diseases. The treatment of anæmia perniciosa and anæmia infantum pseudoleukæmica von Jaksch, either with or without iron, arsenic, or other drugs, is well known to be ineffectual. On the other hand, the treatment of chlorosis and the secondary anæmias has, in my experience, been followed usually by complete recovery. This treatment has been, first, to remove the cause, whether it be the inhalation or ingestion of poisons, such as arsenic or impure air and improper food; second, to adapt the percentages of the food so as to meet the requirements of the special disease or results of that disease, in order that the infant's nutrition may be thus restored to a state of equilibrium, and the effects of the disease may be eradicated. From this stand-point you will understand that it would be impracticable to enter into the subject of treatment in detail in speaking of the blood as a whole.

The treatment of all these diseases of the blood is merely that of the especial disease which causes the blood-changes, and, as I have just said, is well illustrated by the treatment of this case (Case 126) of anæmia secondary to infantile atrophy. If you thoroughly understand the principle which underlies the treatment and subsequent recovery of this case, you will appreciate the truth of what I have just said, and will be prepared to treat intelligently all the cases which I have already described to you.

The more severe types of secondary anæmia are of great interest and importance in the study of infants and of children. As the grade of the anæmia becomes higher the specific gravity of the blood becomes somewhat lower. In addition to this there is more variation in the size and shape of the erythrocytes. There is poikilocytosis and microcytosis. A few nucleated erythrocytes, generally of the normoblast type, are found. They are, however, not very numerous. The same causes give rise to these grave forms as to the milder forms of anæmia. The terms "syphilitic anæmia" and "rhachitic anæmia" are misnomers, as there are no characteristic blood-changes in these anæmias.

CONGENITAL SYPHILIS, WITH ENLARGED SPLEEN.—I will now show you a case of congenital syphilis which has been under my care for some time, and in which a number of careful blood examinations have been made. It represents very well the grave secondary anæmia which at times accompanies syphilis; but, as I have already told you, these examinations of the blood show nothing characteristic of syphilis, but merely an ordinary secondary anæmia.

This infant (Case 127) is three months old, and is being nursed by its mother.

CASE 127.

Male, 3 months old. Congenital syphilis. Grave secondary anæmia. Lower border of ribs, fifth rib, and enlarged spleen marked in black.

It was healthy at birth, and remained so until it was three weeks old, when it showed marked syphilitic lesions, which have since become very characteristic. I shall not here enter into a full description of the case, as I shall show it to you again in a few days in connection with some other cases illustrating my lecture on congenital syphilis. The infant, as you see, is fairly well nourished. You will notice the "waxen" pallor of the skin, so characteristic of the higher grades of grave anæmias. There is a moderate enlargement of the liver, which on palpation is found to be hard and somewhat tender. The inguinal glands are slightly enlarged. The post-aural glands are enlarged. The spleen is much enlarged, and extends, as I have indicated with the black line, from the fifth rib to the left inguinal region. It has, as you see, a peculiar tongue-shaped outline. It is hard, but is not tender. I can detect no other glandular enlargements. The examination of the blood gives the following results:

BLOOD EXAMINATION 12. (Wentworth.)

	Nov. 17.	Nov. 20.
Erythrocytes	3,387,000	3,300,000
Hæmoglobin	47 per cent.	45 per cent.
Leucocytes	20,000	20,000

There is a considerable variation in the size of the erythrocytes, which are pale in color. There is poikilocytosis in a moderate degree; there are also some microcytes and megalocytes. The mononuclear elements predominate (about three-quarters). The eosinophiles are not numerous.

RHACHITIC ANÆMIA.—Rhachitis is so commonly met in early life after the first six months, both alone and in connection with other diseases, that I think it will be well to tell you what is known about the blood before speaking of the separate blood examinations which I have had made in a number of different cases. In this class of cases there is a complete independence of the specific gravity, as influenced by the course of the disease, except when it is complicated by anæmia. When this occurs the specific gravity falls, and it invariably rises as recovery from the rhachitis takes place. Unless this disease is accompanied by a secondary anæmia, the blood is practically normal. Hock and Schlesinger found that if the secondary anæmia was moderate in intensity, and diarrhœa and vomiting occurred, it simply made the anæmia more acute. The majority of the leucocytes were found to be mononuclear and about the size of the erythrocytes. There is a moderate permanent leucocytosis in most of these cases, and at times the mononuclear leucocytes seem to be the most numerous forms.

RHACHITIC ANÆMIA WITHOUT SPLENIC ENLARGEMENT.—This infant[1] (Case 128), a female, seven months and three weeks old, has just been brought to the hospital for treatment. The enlarged epiphyses of the wrist and ankles, the rhachitic rosary, and the other symptoms which so commonly occur in infants fed on patent foods, indicate that this is a case of moderate rhachitis. The infant is pale and poorly nourished. The blood examination gives the following result:

BLOOD EXAMINATION 13. (Whitney and Wentworth.)

Erythrocytes	4,492,000 (occasionally nucleated)
Hæmoglobin	70 per cent.
Leucocytes	22,000
Small mononuclear	33 per cent.
Large "	32 "
Polynuclear	35 "

RHACHITIC ANÆMIA WITH SPLENIC ENLARGEMENT.—This case, ich I have under treatment in the wards, is a very interesting illustion of rhachitis with a secondary anæmia of high grade, accompanied enlargement of the spleen.

The child is three years old, and, as you see, is fairly well nourished. (Case 129.)

CASE 129.

Male, 3 years old. Rhachitis, with enlarged spleen.

It has, however, enlarged epiphyses, a rhachitic rosary, the square rhachitic head, and rked bowing of the legs. On physical examination I find no indication of enlargement the liver or glands. The spleen is very much enlarged, and I have indicated the posi of its outline and its notch, as you see, in black. The blood examination has just been de, and gives the following figures:

BLOOD EXAMINATION 14. (Wentworth.)

Erythrocytes	2,686,250
Hæmoglobin	35 per cent.
Leucocytes	13,000

Poikilocytes and marked pallor of the corpuscles were present.

LECTURE XVI.

THE BLOOD IN INDIVIDUAL DISEASES.

TYPHOID FEVER—SCARLET FEVER—MEASLES—VARIOLA—DIPHTHERIA—PNEUMONIA—BRONCHO-PNEUMONIA—PNEUMONIA AND EMPYEMA—EMPYEMA—MILIARY TUBERCULOSIS—TUBERCULAR MENINGITIS—HYDROCEPHALUS—CHOREA—NEPHRITIS—TUBERCULAR PERITONITIS—INFANTILE ATROPHY—PERIOSTITIS—SCORBUTUS—ICTERUS NEONATORUM—SCLEREMA NEONATORUM.

A NUMBER of observations have been made on the blood of children where a condition of fever was present, and a few regarding the specific gravity of the blood in connection with a heightened temperature.

Widowitz found in five-eighths of the cases examined during fever that the hæmoglobin was higher than in the post-febrile period. The remaining three-eighths of the cases had other complications. Regarding the diminution in the number of erythrocytes and the percentage of hæmoglobin after fever, he explains it either as an actual diminution of hæmoglobin or a dilution of the blood by absorption of fluid from the tissues.

Schiff, who has made the most reliable and methodical experiments on this subject, differs from Widowitz in some points. He found a diminution of erythrocytes during the fever and an increase afterwards, and in long-continued fever this was modified somewhat, so that the absolute count was lower. This he considers due to a diminished production, and so a condition of anæmia is gradually produced. He considers the diminution of the erythrocytes in acute fever to be partially due to an increased degeneration of the red corpuscles, and also to the increased metabolism, and not to diminished production. He could not perceive any connection between the normal daily variation of the temperature and the blood-count. He found that the hæmoglobin was diminished at the beginning of the fever, together with the red corpuscles, but that later it was even more marked than the diminution of the red corpuscles, especially when the fever was long continued. He noted cases in which the red corpuscles increased later, but the hæmoglobin remained diminished, or even sank lower.

Regarding the leucocytes in fever, Schiff considers that they do not follow the course of the fever, as regards increase and diminution, except at the beginning, when there is an increase. Some other authors consider that the leucocyte count is not affected by the temperature alone, but that when fever is accompanied by local suppuration the leucocytosis is much more marked. This agrees with the results obtained in adults.

I shall now show you a number of cases in the wards representing different diseases, in each of which a blood examination has been made lately.

TYPHOID FEVER.—Arnheim found a striking diminution in the amount of hæmoglobin after defervescence had occurred, and in spite of an increase in the number of the erythrocytes. In this disease we know that the leucocytes are usually diminished in number. This also occurs in malaria. In the early stages the erythrocytes are increased, as is also the hæmoglobin. In the later stages a condition of anæmia may occur, producing a diminution of the red corpuscles and hæmoglobin.

Here is a girl, eight years old (Case 130), with the clinical symptoms of typhoid fever. The examination of the blood gives the following result:

BLOOD EXAMINATION 15. (Wentworth.)

Erythrocytes	4,602,500
Hæmoglobin	60 per cent.
Leucocytes	7,000

The next case (Case 131) is also one of typhoid fever, in a boy six years old, and the result of the blood examination is as follows:

BLOOD EXAMINATION 16. (Whitney and Wentworth.)

Erythrocytes	5,496,250
Hæmoglobin	64 per cent.
Leucocytes	7,000
Small mononuclear	14 per cent.
Large "	20 "
Polynuclear	66 "

As I shall not take you into the contagious wards this morning, I think it will be well, before passing on to the other patients, to remind you in a few words of what we should be likely to find on examining the blood of children with scarlet fever, measles, variola, or diphtheria.

SCARLET FEVER.—Widowitz divides the cases of scarlet fever systematically into three groups: *a*, those with a mild course and without complications; *b*, those in which nephritis occurs as a complication; and *c*, those with a malignant course. All three from the beginning showed a high percentage of hæmoglobin, which in uncomplicated cases diminished with the disease, and rose again later without reaching the former high percentage. In the cases of nephritis there was a rapid fall of the hæmoglobin. The malignant cases showed no constant relation. As above mentioned, leucocytosis was generally present, even in the stage of incubation.

MEASLES.—Arnheim found in uncomplicated cases no especial changes in the hæmoglobin. He found slight variations, but less than in scarlet fever, and in convalescence the hæmoglobin often reached the high percentage found in the efflorescent stage of the disease. Von Limbeck, Pick, and Rieder found no leucocytosis in uncomplicated cases of measles, and thought this fact of value in the diagnosis from scarlet fever.

VARIOLA.—Arnheim found the hæmoglobin diminished at the beginning of the disease. After the formation of pustules and during their exsiccation, he found an increase of the hæmoglobin, with diminution of the erythrocytes. Where complicating suppuration occurred, both the erythrocytes and the hæmoglobin remained for a long time abnormally diminished.

Hayem found in "variola confluens" that the erythrocytes were diminished to two million; in the stage of eruption they were normal, and in the stage of suppuration, in consequence of the concentration of the blood, they were increased. Two weeks after the fall of the temperature they were normal.

R. Pick reports forty-two cases examined by him in which he found no leucocytosis, except in the stage of suppuration or in some complication like pneumonia. The temperature, the severity of the disease, or even a fatal termination, unless complicated as above, produced no leucocytosis.

DIPHTHERIA.—Bouchut and Dubrisay found in severe septicæmic forms of diphtheria an increase of leucocytes, increasing and diminishing with the severity of the process. The mild cases showed no leucocytosis, which fact, according to these authors, has a prognostic value. Von Limbeck found always a marked leucocytosis, and it was greatest in the severest cases.

PNEUMONIA.—The leucocytosis is generally very marked, coming on from six to twelve hours before the physical signs of pneumonia show themselves, and in the same way the temperature crisis of the pneumonia is sometimes preceded by a crisis in the number of the leucocytes of about the same length of time. This, of course, is of value in prognosis. There have been some cases recorded (generally fatal ones) in which the leucocytosis did not occur. This may possibly have been dependent upon the nature of the infection. Von Limbeck's experiments upon dogs seem to show that Friedländer's bacillus caused a marked leucocytosis, whereas Fraenkel's diplococcus caused scarcely any. The leucocytosis is said to be higher in children than in adults in pneumonia.

Here in this next bed (Case 132) is an infant eight months old with the characteristic clinical symptoms and physical signs of a fibrinous pneumonia, involving the whole of the left lower lobe of the lung. As the case is one of undoubted pneumonia without complications, the blood examination which has just been made is of unusual interest:

BLOOD EXAMINATION 17. (Whitney and Wentworth.)

Erythrocytes	4,818,750
Hæmoglobin	54 per cent.
Leucocytes	40,000
Small mononuclear	51 per cent.
Large "	21 "
Polynuclear	27 "
Eosinophiles	1 "

The small percentage of the polynuclear cells is very unusual in a case of this kind. They are generally much increased, and their small percentage, though partially accounted for by the age of the infant, cannot be entirely explained in this way.

The next case that I have to show you (Case 133) is also one of pure fibrinous pneumonia, in a boy three and one-half years old. Three examinations of the blood have been made in this case. The first one was made eighteen hours after the crisis had occurred, the second one forty-five hours after the crisis, and the third one has just been made to-day, which is the tenth day since the crisis occurred.

BLOOD EXAMINATION 18. (Whitney and Wentworth.)

(*After crisis.*)

	I. 18 hours.	II. 45 hours.	III. 10 days.
Erythrocytes	4,598,750	4,849,166	About the same as before.
Hæmoglobin	52 per cent.	58 per cent.	Not taken.
Leucocytes	24,500	29,000	17,500
Small mononuclear		28 per cent.	21 per cent.
Large "		18 "	11 "
Polynuclear		51 "	68 "
Eosinophiles		3 "	

At the time that the second examination was made the temperature was normal. The percentage of polynuclear cells in this case would be very small if the patient were an adult, but for a child of this age they show, as would be expected, a moderate increase. To-day, with a normal temperature and with resolution completed, we find, as we should expect, a decided lessening of the leucocytosis. Dr. Cabot's observations have convinced him that the so-called blood crisis occurs in only a certain percentage of cases of pneumonia, and that a blood lysis is more common.

BRONCHO-PNEUMONIA.—The next case (Case 134) is one of broncho-pneumonia occurring in a rhachitic child four years old.

BLOOD EXAMINATION 19. (Whitney and Wentworth.)

Erythrocytes	4,286,250
Hæmoglobin	53 per cent.
Leucocytes	54,000
Small mononuclear	18 per cent.
Large "	11 "
Polynuclear	71 "

The pneumonia was marked by certain circumscribed patches of dulness in both backs. It ran the usual course of broncho-pneumonia, and resulted in complete recovery.

PNEUMONIA AND EMPYEMA.—The next case (Case 135) is that of a boy thirteen years old, who has had a marked fibrinous pneumonia running its usual course, and now has an empyema as a complication. He has been aspirated, and streptococci were found in the pus. The result of the blood examination is very significant.

BLOOD EXAMINATION 20. (Whitney and Wentworth.)

Erythrocytes	3,513,750
Hæmoglobin	43 per cent.
Leucocytes	45,000
Small mononuclear	8 per cent.
Large "	5 "
Polynuclear	86 "
Eosinophiles	1 "

On comparing this case with the two cases of fibrinous pneumonia which I have just shown you, you will note how much larger the percentage of polynuclear cells is than where the pneumonia was uncomplicated.

EMPYEMA.—This next case (Case 136), a boy twenty months old, is one of empyema. The blood examination was made yesterday.

BLOOD EXAMINATION 21. (Whitney and Wentworth.)

Erythrocytes	4,393,750
Hæmoglobin	49 per cent.
Leucocytes	28,000
Small mononuclear	9 per cent.
Large “	16 “
Polynuclear	74 “
Eosinophiles	1 “

(This infant had the radical operation for empyema performed on it, and ultimately recovered completely.)

Here in this next bed is another case of empyema (Case 187), ten years old, in which the blood count was made this morning.

BLOOD EXAMINATION 22. (Whitney and Wentworth.)

Erythrocytes	4,355,000
Hæmoglobin	60 per cent.
Leucocytes	66,000
Small mononuclear	7 per cent.
Large “	8 “
Polynuclear	85 “

(This child was operated upon and recovered completely.)

MILIARY TUBERCULOSIS.—You will remember the male infant twenty-five months old (Case 188) which I examined before you in the ward yesterday, and in which there was a question whether it was a case of simple starvation or one of general miliary tuberculosis with some complication. The blood examination resulted as follows:

BLOOD EXAMINATION 23. (Whitney and Wentworth.)

Erythrocytes	5,567,500
Hæmoglobin	66 per cent.
Leucocytes	29,500

The autopsy this morning showed a general miliary tuberculosis of all the organs, and an absence of pneumonia.

Miliary tuberculosis in adults shows no leucocytosis, and the increase of the leucocytes in this case is but moderate, and might be due entirely to starvation.

TUBERCULAR MENINGITIS.—Here is an interesting case of cerebral disease (Case 189) in a male infant. The clinical symptoms and general aspect of the child are those of tubercular meningitis. The blood examination, however, shows that some complication is in all probability present.

BLOOD EXAMINATION 24. (Whitney and Wentworth.)

Erythrocytes	4,541,250
Hæmoglobin	68 per cent.
Leucocytes	38,000
Small mononuclear	22 per cent.
Large “	20 “
Polynuclear	58 “

In considering this case I must remind you that the bacillus of tuberculosis is not a pyogenic organism. As I can find no lesion in any of the organs to account for the increase in the leucocytes, a large proportion of which are polynuclear neutrophiles, we that the original miliary inflammation was followed by a secondary infection producing organism.

ant passed through the various typical stages of tubercular meningitis and topsy was obtained.)

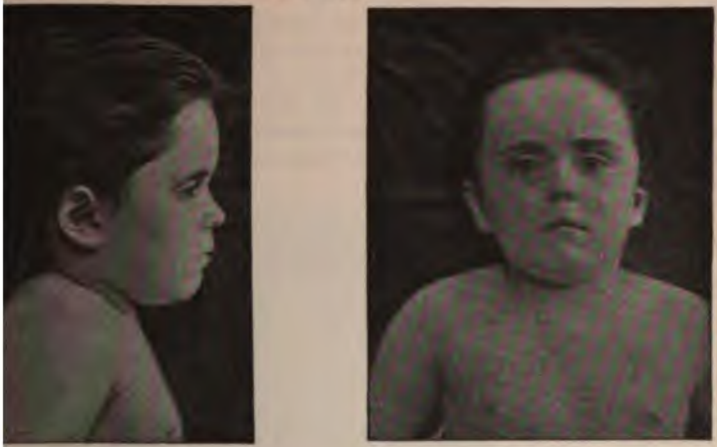
ment that the leucocytosis which is at times found in tubercular meningitis ome complication is well illustrated in the case (Case 140) of the little girl old who was shown to you a few days ago as a case of tubercular meningitis. ymptoms were very typical from the beginning to the end of the disease, but amination, as I explained to you at that time, led me to believe that some was present.

BLOOD EXAMINATION 25. (Wentworth.)

ocytes	5,298,750
globin	68 per cent.
cytes	37,500

psy showed the case to be one of tubercular meningitis, represented by solitary he brain without any purulent exudation. There was, however, found in the ppendicitis, which accounted for the leucocytosis.

OCEPHALUS.—This little girl (Case 141), six years old, is a marked case alus. The history of the noticeable enlargement of the head corresponds to drocephalic appearance of the child.



CASE 141.

esult of the blood examination of this case I cannot explain the high pere polynuclear cells.

BLOOD EXAMINATION 26. (Whitney and Wentworth.)

ocytes	5,675,000
globin	80 per cent.
cytes	19,000
nall mononuclear	4 per cent.
arge "	8 "
olynuclear	83 "
osinophiles	5 "

t case (Case 142), a boy two years and ten months old, is apparently also one alus, but of slight degree. The blood examination resulted as follows:

BLOOD EXAMINATION 27. (Whitney and Wentworth.)

Erythrocytes	4,492,500
Hæmoglobin	72 per cent.
Leucocytes	20,500
Small mononuclear	20 per cent.
Large "	20 "
Polynuclear	58 "
Eosinophiles	2 "

The cause of this leucocytosis is not known. The examination of the lungs and the heart was negative; the head measured twenty-six inches; there was protrusion of the eyes, as well as mental disturbance. The child remained in the hospital, and showed continued improvement until complete recovery some months later.

CHOREA.—I shall now show you a case (Case 143) of chorea of a severe type, but without complications. The child, a boy eight years of age, can scarcely swallow, and is unable to speak, stand, or walk. The choreiform movements are, as you see, constant. The result of the blood examination is as follows:

BLOOD EXAMINATION 28. (Wentworth.)

Erythrocytes	5,222,500
Hæmoglobin	60 per cent.
Leucocytes	19,000

There has at times been a faint hæmic murmur over the base of the heart, but this h been very transient and has now passed away.

NEPHRITIS.—I have here to show you two cases (Cases 144, 145) of renal dise The examinations of blood made in renal disease in children have not been very extensi or satisfactory, but in general the specific gravity of the blood is quite low, on account o the loss of albumin in the blood serum. The specific gravity of the serum is much diminished, 1022 to 1023. Klein, in a series of observations upon the blood in the nephriti of scarlet fever, has found an increase of eosinophiles in favorable cases, and an absence o them in fatal cases.

ACUTE NEPHRITIS.—This first case (Case 144), a boy six years old, was one o acute nephritis. The urine at present, however, only shows an active hyperæmia of t kidney. The blood examination gives the following results:

BLOOD EXAMINATION 29. (Whitney and Wentworth.)

Erythrocytes	3,481,250
Hæmoglobin	51 per cent.
Leucocytes	32,500
Small mononuclear	8 per cent.
Large "	10 "
Polynuclear	80 "
Eosinophiles	2 "

The percentage of the eosinophiles, you see, is no greater than normal, although t case seems to be tending towards recovery.

CHRONIC NEPHRITIS.—The other case (Case 145), a girl nine and one-half ye old, is one of chronic parenchymatous nephritis.

BLOOD EXAMINATION 30. (Whitney and Wentworth.)

Erythrocytes	4,355,000
Hæmoglobin	60 per cent.
Leucocytes	33,000
Small mononuclear	36 per cent.
Large "	4 "
Polynuclear	60 "

unately, the percentage of eosinophiles in this case was not recorded. The size ocyte count is remarkable.

ERCULAR PERITONITIS.—I have here two cases of tubercular peri- which the diagnosis has been verified by laparotomy. The blood examinations before the operations were performed.

rst case (Case 146) was one of an infant eighteen months old, and the blood n resulted as follows:

BLOOD EXAMINATION 31. (Whitney and Wentworth.)

throcytes	4,970,000
noglobin	48 per cent.
ocytes	19,000
Small mononuclear	19 per cent.
Large "	18 "
Polynuclear	73 "

ase, as well as the first one, followed the rule of an absence of leucocytosis in s, for at this age the leucocyte count may be as high as 19,000 to 20,000 under al conditions.

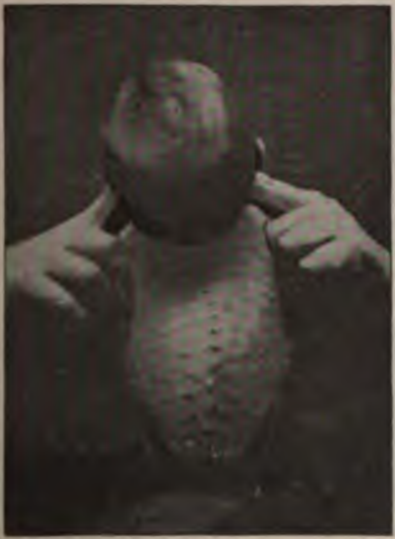
cond case (Case 147) is a boy nine years old.

BLOOD EXAMINATION 32. (Whitney and Wentworth.)

throcytes	4,792,500
noglobin	55 per cent.
cocytes	7,500
Small mononuclear	18 per cent.
Large "	31 "
Polynuclear	54 "

ANTILE ATROPHY.—The next case (Case 148), eleven months old, is one ile atrophy. The extreme emaciation of this infant is well seen in looking at its

Case 148.



Infantile atrophy. Female, 11 months old.

here there is an almost entire absence of adipose tissue, so that the vertebræ and the be studied as though on the dissected skeleton.

The result of the blood examination in this case is as follows:

BLOOD EXAMINATION 33. (Wentworth.)

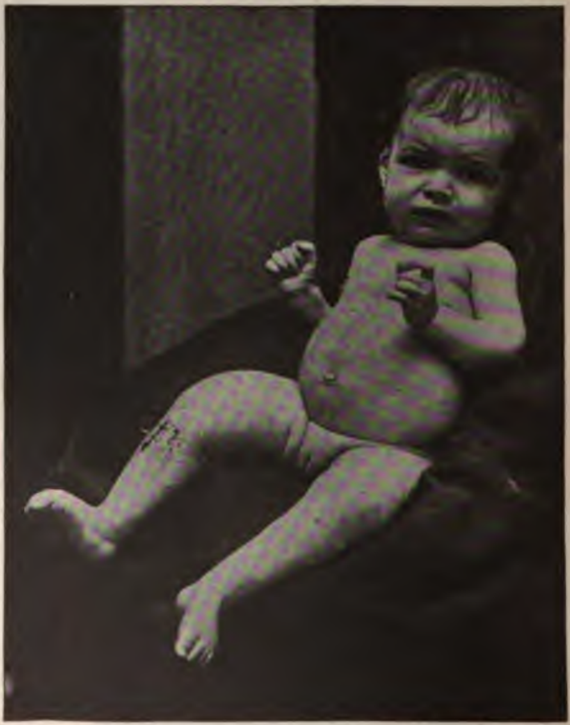
Erythrocytes	4,738,750
Hæmoglobin	76 per cent.
Leucocytes	21,000

This count was made after the infant had been under treatment for over three and a half months, so that we cannot take it as typical of the early stages of the disease.

Guffer found a gradual diminution of erythrocytes and an increase in leucocytes in these cases of infantile atrophy, which he referred to the accompanying anæmia. Parrot found that a diminution of red corpuscles constantly went on until death, and that the increase in the leucocytes corresponded to the severity of the disease. Schiff made some experiments proving the analogy between these cases with loss of fluid and cases in which fluids were withheld, both causing concentration of the blood.

PERIOSTITIS.—In order to show you of what great importance a careful examination of the blood may be in determining the diagnosis in obscure cases, I will report to you the following case (Case 149):

CASE 149.



Female infant, 15 months old. Periostitis of both legs.

Many of you will remember seeing the infant at the Children's Hospital, where it was brought to be treated for a persistent and painful swelling of the right thigh. It was at that time fifteen months old, and the pain had been so severe that it had lost much

n this examination was that the growth removed was not large enough for a posi-
iosis, but that it simulated very closely an osteo-sarcoma. A few days later
icision was made in the right tibia, which can be seen in this photograph taken
ly after the operation.

her piece of the growth was removed, and on examination was reported by the
t to be probably an osteo-sarcoma. The infant had been in the hospital for
days, and the swelling had steadily increased, while its general condition had
orse. The question of amputation was considered, but at this time a blood count
, which so strongly pointed toward the absence of a malignant growth that it was
wiser to postpone the operation and wait for further developments.

BLOOD EXAMINATION 34. (Whitney.)

icocytes.

Small mononuclear	46 per cent.
Large "	18 "
Polynuclear	36 "

ignificance of this differentiation of the leucocytes lay in the small percentage of
iclear variety, which should have been found increased if the disease of the bone
a new growth, such as is represented by osteo-sarcoma. Somewhat later, but
blood examination had been finished, the left thigh was also incised, owing to a
of trouble in that location, and a piece of periosteum covering the left femur was
This was also reported as a probable osteo-sarcoma, and the infant was dis-
om the hospital as a hopeless case, and was taken home to die. The subsequent
this case is of extreme interest, in reference to the value of blood examinations,
fant soon began to improve, the swelling was absorbed, and, although the infant
n walking, it is now, after an interval of some months, well and strong, and
o appearance of disability in the legs. The growth was probably a sluggish
of an unusual type, which simulated sarcoma very closely. The case is a
e.

RBUTUS.—Nothing distinctive has as yet been found in the blood
tions which have been made in cases of infantile scorbutus.

ERUS NEONATORUM.—The simple benign form of icterus
um, which I have described to you in an earlier lecture, is practi-

LECTURE XVII.

PARASITES OF THE BLOOD.—LITERATURE OF THE BLOOD IN EARLY LIFE.

As in other parts of the economy, so in the blood are found parasites, which may be of the vegetable or of the animal kingdom.

Of the vegetable parasites, such as (1) Moulds, (2) Yeasts (Saccharomycetes), and (3) Fission-fungi (Schizomycetes, Bacteria), the latter (Fission-fungi) are the only ones which would be likely to occur in the blood of early life, and even they do not especially concern us in our discussion of the blood.

Of the animal parasites (Hæmatozoa) we find two classes, (1) Protozoa and (2) Vermes. The former class (Protozoa) is the only one with which I have had any experience, and I shall therefore confine my remarks to the micro-organisms of malaria.

MALARIA.—The term malaria should be limited to a definite disease in which we know there is a specific infectious origin. This specific infection is primarily shown in the blood in the form of certain micro-organisms which, like the amœba coli, belong to the class of protozoa, and inhabit the blood of the infected individual. We must, however, understand that in the specific micro-organisms of malaria we have not as yet proved the three conditions required to show that a given disease is caused by a specific micro-organism. These three conditions, as formulated by Koch, are as follows:

(1) The presence of the organisms in all cases of the disease and in such distribution as will explain the lesions.

(2) The isolation of the organism in pure culture.

(3) The reproduction of the disease by inoculation with the isolated organisms.

When, as has been said by Welch, all these conditions have been fulfilled, there will be no doubt that the disease has been caused by the especial organism. In regard to malaria, therefore, you see that only the first of Koch's three required conditions is present. The micro-organism of malaria has not been found in any other part of the body than the blood, and malaria may therefore justly be said to be a disease of the blood. It has no known means of exit from the body, and its mode of entrance has not been definitely determined. The germs of this parasite may be contained in the blood-plasma, or in the substance of the erythrocytes. The name plasmodium has been given to the germ found in the red blood-disks. According to Thompson, in acute paludism (malarial fever) the plasmodium

olorless and transparent, and at the height of their development
ndergo segmentation. This amœboid form of the parasite is the
ommonly found in what is designated as the tertian variety of mal-
nd is the most common of all the known forms of the parasite of
ia.

addition to these amœboid forms, crescentic shapes of the germ,
ing to the investigations of Laveran, are common in the blood of
types of paludism, irregular forms of the disease, and malarial
ia. Like the amœboid forms, they are transparent and colorless,
for the pigment-granules which they contain in their centres. They
ger than the amœboid forms, are much more rare, and are much less
l by the action of quinine.

uncilman describes flagellate bodies as being most commonly found
od which has been aspirated from the spleen; and in acute cases of
a they may sometimes appear in other situations. They exhibit from
o eight vibrating cilia.

is still a matter of dispute whether the plasmodium malariæ is poly-
ous and thus may produce the different types of malaria, of which I
resently speak, or whether there are certain distinctly separate organ-
which the name plasmodium malariæ is applied.

ere is no doubt that two distinct forms of parasites of malaria can be
sticated by the appearance of the plasmodium in the blood, and that
wo forms can be separated clinically.

lgi is the investigator who has most clearly shown that there is more
ne parasite of malaria, while Laveran is the exponent of the poly-
ous theory.

ETHOD OF EXAMINATION.—The technique of the examination of
ood for the purpose of detecting the plasmodium malariæ is very
. I shall describe the method which has been used more largely
ildren than any other, and which has been found satisfactory by
oplik, of New York, whose work on the blood of malaria in early

cover-glasses are then stained in a very dilute solution of methylene-blue. Eosin is not used, as some varieties decolorize the blue and thus introduce an element of uncertainty. The blood is heated at a temperature above the boiling-point (120° C.) on the plate. The variety of dye is important, as some blue does not stain. Grübler's blue powder, soluble in alcohol, has proved to be satisfactory. A few drops of the saturated solution of this blue in alcohol are added to 30 c.c. (1 ounce) of water. The cover-glasses should not be deeply stained, as certain appearances may, under these circumstances, be lost. They are to be repeatedly washed in water and then dried in the air without heating, as heat decolorizes them. In this way the blood-cell is well hardened, and its protoplasm and hæmoglobin stain more certainly than when hardened with alcohol, sublimate, or osmic acid. Other specimens, again, may be stained by Ehrlich's aniline method to study the different appearances. The erythrocytes of malarial cases, when stained in this way, show the plasmodium in blue and the protoplasm in yellowish green or colorless rings, if there is anæmia. If the Ehrlich dyes are used, aurantia, orange G, and others (preferably the solution in glycerin of eosin, indulin, and aurantia), the plasmodium does not stain, but the hæmoglobin of the erythrocytes is stained in shades of varying intensity.

As in every case of pronounced malaria, whether in early life or in adults, the characteristic feature of the disease is a paroxysm, we naturally should first examine the blood at a time when this paroxysm is taking place, and from this point study the changes which the parasite shows in the intervals between the paroxysms.

Golgi was the first observer who actually described and differentiated the more common forms of paludism, and his observations coincide practically with those which have been made since. I shall, therefore, describe, as observed by Golgi, the main features of the changes in the blood which are caused by the development of the plasmodium, and such features as will explain the resulting symptoms of malaria and will thus be of clinical importance. These changes in the plasmodium have been so well described by Dr. Thayer, of Baltimore, that I shall quote what has been said by this admirable investigator. It will, however, be necessary first to explain certain terms which, having been used in connection with malaria, and having become established before the specific parasite of malaria was known, are really more adapted to the symptoms of the disease, and are hence given more prominence than is in accordance with our present knowledge of it.

The prominent symptom of malaria being the paroxysm, earlier authors naturally classified malaria according to the time when the paroxysms appeared, using the term quotidian where they occurred with intervals of twenty-four hours, tertian where they occurred with intervals of forty-eight hours, and quartan where they occurred with intervals of seventy-two hours. The term tertian is somewhat misleading, unless we understand

oxysms. The *remittent* form is characterized by the presence of
less fever of a continued type which does not cease between the
ms. You will presently see that these terms should not be used as
ations of distinct types of malaria, as the conditions which they rep-
nay, according to chance, appear in any of the types, and are merely
by a variation in the behavior of the parasite.

ve examine the blood from a tertian case where there is a decided
of twenty-four hours between the paroxysms, we find that just
e paroxysm some of the erythrocytes will contain small, round,
s bodies, which appear to have a slight depression in the centre,
en stained in dry specimens show a pale central area with a dark
ry.

hese bodies, examined in the fresh specimen, show active amœboid
ents. A few hours later the organism will be found to have in-
somewhat in size and to contain a few fine brownish pigment-
s which dance actively under the eye, the motion probably being
undulating movements in the protoplasm. On the day between the
sms the bodies will be found to have half filled the erythrocytes.
re still actively amœboid, and the number of pigment-granules is
rably increased. The erythrocyte at this stage will be seen to be a
rger than its unaffected neighbors, and to be considerably decolorized.
day of the paroxysm the organism is found to have entirely filled
nost to have destroyed the erythrocyte, which is represented only by
pale rim about the full-grown parasite, if indeed it has not entirely
eared. The pigment-granules may show at this stage a very active
, but the amœboid movements of the organism, as a whole, are but
marked. At the time of the paroxysm a change takes place. The
nt gathers together in a more or less solid clump, usually in the
of the erythrocyte, while the rest of the protoplasm looks somewhat
ar, and shows a suggestion of lines radiating outward from the
This appearance gradually changes, the lines becoming more dis-

by these fresh segments has never been actually observed, the evidence that this occurs is so strong that we can safely accept it as a fact. Besides these forms, we see not infrequently small or large extra-cellular pigment bodies, —that is, organisms resembling exactly those within the erythrocytes, except that they are free in the blood-current. These may be seen at times to break up into several smaller bodies, while at other times they may show a long tail-like non-motile process containing sometimes a few pigment-granules. They are probably organisms which have escaped from the erythrocytes, or full-grown bodies which have broken up. They are considered to be a degenerative form."

At times we find the flagellate bodies which I have already referred to as described by Councilman.

According to Thayer, the characteristics of this form of organism, which is observed in tertian fever alone, are so marked that with a little study of the parasites one can make a definite diagnosis of the type of fever from an examination of the blood alone. He also observes that the quartan fever is not common in this country, but that where he has seen it the organisms differ distinctly from the tertian parasite, and their appearance coincides exactly with that described by Golgi. For instance, the first stage, of the quartan organism is similar to that observed in the tertian, except that the amœboid movements are not so active; as the body develops the rods and clumps of pigment are larger and darker than those which appear in the tertian form, while the amœboid movement of the organism is relatively slight. The full-grown quartan forms are materially smaller than those found in the tertian, while the erythrocytes, instead of being expanded and decolorized, appear at times shrunken about the body and of a somewhat deeper old-brass color (Messingfarber). Thayer also states that in the quartan form the segmentation of the organism is into from six to ten different parts, instead of from twenty to thirty, as is seen in the tertian form.

Although Marchiafava and Celli have described an organism which they assert causes a definite form of paludism represented by the paroxysm occurring at intervals of twenty-four hours, this has not been corroborated by other investigators. We are not justified, therefore, in assuming that there is an especial parasite which causes a distinct disease represented by the term quotidian. In like manner, we do not at present recognize that there is a separate parasite which may cause the symptoms of remittent fever, unless it shall be proved to be the æstivo-autumnal. I shall therefore confine my remarks to the two forms of disease represented by intervals in the paroxysms of forty-eight hours and seventy-two hours.

It is evident from what I have already told you concerning the changes which the plasmodium malariæ undergoes in the process of its development in the erythrocytes that it causes the different symptoms which arise in malaria by its action in the different stages of its development. We see also that the segmentation of the organism is always coincident with the paroxysms, and that the interval between the paroxysms is characterized by a distinct and early stage of development of the parasites.

Koplik has made so especial a study of malaria as it appears in early life that I shall quote freely from his writings on this subject.

In pure types of paludism, either tertian or quartan, one generation of the plasmodium will be found to predominate. In those cases of tertian where the paroxysms are found to be of daily occurrence, several generations of parasites, each with a different cycle of development, will be found in the blood. The same observation will be found to be true where irregular types of fever with the tertian parasite are carefully examined, and also where the blood in quartan fevers is examined. If more than one generation of parasites exists in the blood in a tertian case, the fever may become quotidian, with daily paroxysms due to the ripening of distinct sets of parasites on different days, each set of parasites taking forty-eight hours to mature. In like manner, in cases of quartan fever, through the ripening of distinct sets of parasites on different days, different combinations occur, according to the number of sets of parasites. Thus, while in the form in which there is only one parasite the intervals between the paroxysms are seventy-two hours, in that in which there are two parasites there may be an interval between the paroxysms of only forty-eight hours, and where there are three parasites there may be an interval of only twenty-four hours, thus representing the quotidian chills described by Mannaberg. This will be more clear to you if you examine this table (Table 88), which I have arranged for the purpose of definitely explaining the different types of paludism as they are now understood by the most recent investigators.

TABLE 88.

The Principal Combinations of Paroxysms caused by the Plasmodium Malariæ.

	Intervals.	1st day.	2d day.	3d day.	4th day.
TERTIAN.					
Pure tertian . . . (One parasite.)	48 hours.	Paroxysm.	No paroxysm.	Paroxysm.	No paroxysm.
Double tertian . . (Two parasites. Quotidian.)	24 hours.	Paroxysm.	Paroxysm.	Paroxysm.	Paroxysm.
QUARTAN.					
Pure quartan . . . (One parasite.)	72 hours.	Paroxysm.	No paroxysm.	No paroxysm.	Paroxysm.
Double quartan . . (Two parasites.)	48 hours.	Paroxysm.	Paroxysm.	No paroxysm.	Paroxysm.
Triple quartan . . (Three parasites. Quotidian.)	24 hours.	Paroxysm.	Paroxysm.	Paroxysm.	Paroxysm.

The table, as you see, explains how the different intervals in the paroxysms are caused by the development of the parasite on different days. It will therefore be easy for you to understand that it is according as the parasite happens to develop that we have a regular or an irregular periodicity.

Thus, it may happen that we have two parasites, and these two parasites may develop on the same day, but at different hours. In this case, supposing that they are of the tertian type, two paroxysms may occur on the same day, followed by an interval of forty-eight hours from the time of the full development of each of the parasites until this development occurs again. In this way different broods of parasites may cause an almost infinite variety of symptoms. Again, we must recognize that it is probably true that it is only when the broods of the parasites are especially large in number that a pronounced paroxysm is produced, because if the brood is small in number and insignificant it may cause only a greater or less rise of temperature in place of a pronounced paroxysm. You see that in this way we can probably explain those different forms which have been designated as remittent fever. That is, on the intervening day, when there is no paroxysm, but only a continuous heightening of temperature, it may be that the broods have developed only sufficiently to produce fever and not a paroxysm, and we shall probably in the future, by a more extended study of this parasite in all its phases and under all circumstances, be able to show that it is a variation in numbers as well as in the kind of the parasite which causes these distinct differences in the symptoms of malaria.

It has been noticed that the administration of quinine tends to interfere with the regularity of the time of the paroxysm, and in this way other variations may occur. It has also been noticed that if the paroxysm comes earlier in the day than it has been doing, the disease is apt to be of a severe type and to be growing worse, while if the interval is lengthened and the attack is found to come at a later hour in the day than usual, it is a sign that the disease is amenable to treatment, is of a benign character, and is tending towards recovery.

The tertian form is the one which is by far the most common in this country, and the one which is most influenced by the administration of quinine, the other form, represented by the quartan, being peculiarly difficult to manage with quinine. In young infants the tertian form in its quotidian variety is met with most commonly. In older children, in my experience, it is the pure tertian that is most common. It will be noticed, by glancing at the table (Table 88, page 385), that the quartan form of paludism can never represent by its intervals and paroxysms the pure tertian form.

PATHOLOGY.—There are no especial differences between the pathological lesions found in the malaria of children and those which occur in adults. I shall, therefore, not dwell on this part of the subject, but shall merely state what Thayer has said concerning this disease.

In *acute* cases of malarial fever, on examination with the microscope, the *cerebral* capillaries are found to be crowded with malarial parasites. There is usually a marked granular degeneration of the endothelium of the vessels.

The *spleen* is always enlarged. The capsule is tense. The parenchyma is cyanotic, of a slaty-gray color, and almost diffluent. The pulp of the leen is found to contain enormous numbers of red blood-corpuscles, many which contain parasites. It also contains numerous large white elements ch in protoplasm, with usually a single bladder-like nucleus and at times arse granulations. These elements are commonly laden with pigment, hich at times has the same arrangement as it has in the body of the parate itself. There may be free pigmentation in the intercellular spaces of e pulp. The small mononuclear elements and the lymphocytes of the llicles never contain pigment. The capillaries are usually filled with e plasmodia, while the splenic veins show relatively few, though they lways contain large cells enclosing pigment or the remains of red blood-orpuscles.

The *liver* has usually a slaty-gray color. The capillaries are filled with eucocytes, which contain numerous pigmented bodies. Relatively few plasmodia are found in the blood-corpuscles in the vessels.

The *lungs* show in their capillaries numerous cells containing pigment clumps and well-preserved parasites, although it is unusual to find pigment in the endothelial cells, in the capillaries, and in the smaller veins.

In the areas of broncho-pneumonia which may occur, polynuclear leucocytes are often found, while the large pigmented cells take no part apparently in the active inflammatory process.

The vessels of the *kidneys* contain relatively few organisms. The glomeruli may be considerably pigmented. There may be marked degeneration of the epithelium of the capsule, and at times changes in the parenchyma, especially areas of necrosis of the epithelium of the convoluted tubules. The other *viscera* show no special characteristic changes, except, at times, that of melanosis.

In the more *chronic* form of malaria the *anæmia* is usually particularly marked. The *spleen* is always enlarged and very firm. There is marked thickening of the capsule, which is often adherent to the neighboring tissue. On section the spleen is generally of a dark brownish-gray color, the fibrous tissue throughout the organ being greatly thickened. The *liver* is considerably enlarged, and usually has a grayish-brown or slaty color. At times there is a considerable increase in the connective tissue. The *kidneys* show no particularly characteristic changes, though there may be considerable pigmentation. The pigment is most marked about the blood-vessels and the Malpighian bodies, and sometimes in the region of the convoluted tubules.

There are no characteristic changes in the other *organs*, except the slaty-grayish pigmentation.

DIAGNOSIS.—Malaria as it occurs in early life is far more difficult to diagnosticate by its symptoms than where the disease runs the typical course usually seen in the adult. It is the most protean disease which we are called upon to deal with in young children, and it simulates so closely almost every other disease we are likely to meet with that we should always be on our guard, and allow the possibility of the existence of the plasmodium malariæ in making a diagnosis in a doubtful case where a periodicity is noticed in the symptoms.

The only rational method of determining that we are dealing with a cas of malaria is the examination of the blood, which at once settles the questio if the plasmodium be found.

SYMPTOMS.—The symptoms of malaria as it occurs in infants and i young children are much more varied and far more uncertain than thos which we are accustomed to meet with in adults.

The younger the individual the more likely are the pronounced chill to be replaced by some other symptom, such as vomiting, delirium, and co vulsions. The paroxysms come more frequently in children than in adult and in young children a condition of apathy and somnolence, sometimes wit fever, and sometimes accompanied by coldness of the extremities and a co lapsed condition, very commonly replaces the chill of the adult. The symptoms, representing the onset of the disease, may often disappear as t disease becomes established, and in their place we may meet with the sym toms of some other disease, such as bronchitis, torticollis, and many oth affections. The symptoms of these other diseases will often continue an be very intractable until quinine is given, when they will disappear, an thus we shall be led to believe that we have been dealing with one of t masked and misleading manifestations of the plasmodium malariæ. (Vi Case 269, page 610.)

My experience with malaria in young children is so similar to that o Dr. Holt, of New York, who has written more fully on the symptoms o malaria in early life than any one else of whom I know, that I shall quo from his writings on this subject.

The susceptibility of the nervous and respiratory systems in youn children to produce variations in the form and type of malaria is most mi leading in regard to diagnosis, the symptoms referable to a particular org often completely overshadowing the real disease, malaria, and producing a entirely new clinical picture. The symptoms often are so indefinite and t disease frequently comes on so insidiously that the physician does not s the case until it has made considerable progress and the diagnosis thus much obscured.

In addition to the other symptoms of which I have already spoke severe pain in the head and sometimes in the epigastric region is met wit In the form in which the invasion is gradual, the prominent symptoms a anæmia, loss of appetite, and frontal headache of moderate type. T spleen in the majority of cases is found to be enlarged, but the well-know difficulty of detecting an enlarged spleen in young children makes it po sible that in many cases there is enlargement of the spleen without o being able to detect such enlargement by percussion or palpation.

The time and character of the onset of the disease and of its paroxys are very irregular, so much so, indeed, that it would not be practicable dwell upon the exact differences which occur from those in the adu

Splenic and hepatic tenderness, and pains in the back, extremities, and neck, are occasionally observed, and general cutaneous hyperæsthesia is at times noticed. As the capsule of the spleen is less resistant in young children than in adults, the organ seems to enlarge more rapidly, and also to subside more quickly, in children than in adults.

The condition of the intestinal tract varies as much as do the other symptoms. Sometimes constipation is present, and sometimes diarrhœa, the latter being the more prominent the younger the child.

Dr. Holt's observations on the pulmonary symptoms occurring during attacks of malaria are so interesting and important that they should be recorded. Bronchitis was found to be the most frequent of all the complications occurring in the course of malaria, and again and again proved to be intractable until its malarial origin was discovered. Certain acute cases appeared to be pulmonary congestions analogous in their pathology to the congestions of the spleen and the liver. The pulmonary symptoms in these cases were quite uniform and characteristic. The invasion was acute and the temperature high, ranging from 40° C. to 41.1° C. (104° to 106° F.). The respirations were very rapid, in three or four cases reaching 100 in a minute, and resembling the superficial breathing of lobar rather than the labored breathing of lobular pneumonia. The face was often cyanotic, and the pulse varied from 160 to 200 per minute. In one or two cases there was marked drowsiness. The physical signs were usually a slight increase of vocal fremitus and slight dulness on percussion. The respirations were always high-pitched and sometimes broncho-vesicular. Vocal resonance was exaggerated, and there were sonorous râles and occasionally coarse and fine mucous râles. These signs were sometimes general in both lungs, but were usually most marked behind and towards the apices. They were at times found to be confined to a single lung and once to a single lobe. When first seen they were diagnosticated as cases of pneumonia, but their subsequent progress and termination convinced Dr. Holt that they were temporary manifestations of malaria, for patients who were seen in the afternoon with these symptoms would be found the following morning running about the house with a normal pulse and respiration, and with only the signs of an insignificant bronchial catarrh in the chest. These attacks would recur on the following days until quinine was administered. Marked splenic enlargement was detected in these cases.

Pneumonia, both lobar and lobular, was occasionally found as a complication of malaria.

Spasmodic asthma of malarial origin was seen in some cases. These attacks were accompanied frequently by marked splenic enlargement, and were promptly relieved by antiperiodics.

PROGNOSIS.—The prognosis of malaria in children is good, provided that the child is removed from the malarial district and is treated with quinine. Relapses occur, even after long intervals of apparent immunity, and the disease can recur a number of times.

When a child has been once attacked by the plasmodium malariæ, it seems to be peculiarly vulnerable to a second attack of the organism.

TREATMENT.—Quinine is the only drug which can be relied upon to eradicate the plasmodium malariæ from the blood, and is the only medicine for this purpose which I shall mention.

It may be given to an infant under six months in doses of 0.03 gramme (½ grain); at one year the dose may be 0.06 gramme (1 grain), at two years it may be 0.12 gramme (2 grains), and it can be increased up to 0.3 or 0.36 gramme (5 to 6 grains) at five and six years. There is little danger of giving too large doses of quinine to children, as they tolerate the drug very well.

The manner of administering quinine is rendered somewhat difficult on account of the bitter taste of the drug and its insolubility in water. In very young infants, and in fact in the first six or eight months of life, it is well to try the effect of suppositories. In older infants and in children it can usually be successfully concealed in a small amount of chocolate cream.

The time for the administration of the quinine does not have to be regulated so carefully as in the adult. The dose can often be given with effect three or four times in the twenty-four hours. It is commonly given immediately after a paroxysm. I have been in the habit of giving it about eight or ten hours before the paroxysm is expected. It is well to continue the treatment with quinine for some weeks after the paroxysms have ceased, as the symptoms often return if the quinine is omitted at once.

The anæmia which always accompanies the disease to a pronounced degree should be treated with doses of arsenite of potash, or with some mild form of iron, such as the saccharated carbonate or the tartrate of iron and potash.

These prescriptions, varied to suit the individual, are what I am in the habit of using in cases of malaria:

PRESCRIPTION 42.

For an Infant under Six Months.

Metric.	Gramma.	*Apothecary.*	
℞ Quiniæ sulphatis	0.36	℞ Quiniæ sulphatis	gr. vi;
Olei theobromæ	11.25	Olei theobromæ	ʒiii.
M.		M.	
Ft. suppos. no. 12.		Ft. suppos. no. 12.	

S.—One suppository to be used every 6 hours.

PRESCRIPTION 43.

Metric.	Gramma.	*Apothecary.*	
℞ Ferri carbonatis saccharati	0.90	℞ Ferri carbonatis saccharati	gr. xv.
Ft. pulv. no. 15.		Ft. pulv. no. 15.	

S.—For an infant under 6 months, 1 powder every 8 hours.
For an infant from 6 to 12 months, 1 powder every 6 hours.
For an infant from 12 to 18 months, 1 powder every 4 hours.
For a child of 3 years, 3 powders every 8 hours.
For a child of 6 years, 3 powders every 6 hours.
For a child of 12 years, 3 powders every 4 hours.

PRESCRIPTION 44.

Metric.	Gramma.			*Apothecary.*	
Ferri et potassii tartratis	3	00	℞	Ferri et potassii tartratis	℈iiss;
Glycerini	18	75		Glycerini	ʒv;
Aq. destil.	ad 90	00		Aq. destil.	ad ℥iii.
M.				M.	

S.—For a child 2 years old, 2 c.c., or ½ drachm, once in 8 hours.
For a child 4 years old, 4 c.c., or 1 drachm, once in 8 hours.
For a child 8 years old, 4 c.c., or 1 drachm, once in 6 hours.
For a child 12 years old, 8 c.c., or 2 drachms, once in 8 hours.

PRESCRIPTION 45.

For a Child Two Years old.

Metric.	Gramma.			*Apothecary.*	
Liq. potassii arsenitis	0	96	℞	Liq. potassii arsenitis	♏xvi;
Aq. destil.	ad 120	00		Aq. destil.	ad ℥iv.
M.				M.	

S.—4 c.c., or 1 drachm, to be given every 8 hours.

In my experience, malaria may occur at any age.

Dr. Dane has recently mentioned to me a case (Case 150) of probable malaria (the blood was not examined) in an infant a few days old.

The infant's mother had malaria during her pregnancy, and some of the manifestations of the disease appeared ten days before the birth of the infant. The infant from the earliest days of its life showed symptoms of severe digestive disturbance, characterized by vomiting and diarrhœa, and far beyond what could be accounted for by the lack of equilibrium of the function of the mother's mammary gland.

Dr. Dane made a careful physical examination, but failed to detect anything abnormal in its thorax or abdomen.

Observations of the temperature in this case, taken both in the axilla and in the rectum, showed that it was of an irregular type, varying from 37.2° C. to 38.3° C. (99° F. to 101° F.) rectal, and that at times in the latter part of the day it rose to 39.4° C. to 40° C. (103° F. to 104° F.) axillary.

Every day at about 1 A.M. there was a paroxysm, represented by cyanosis, coldness of the entire skin, both of the body and of the extremities, collapse, and somnolence. These attacks, beginning at the seventh day of life, lasted until the twelfth day, when quinine in 0.03 gramme (½ grain) doses, given in suppositories and administered every two hours for seven doses, at once and completely checked the paroxysms.

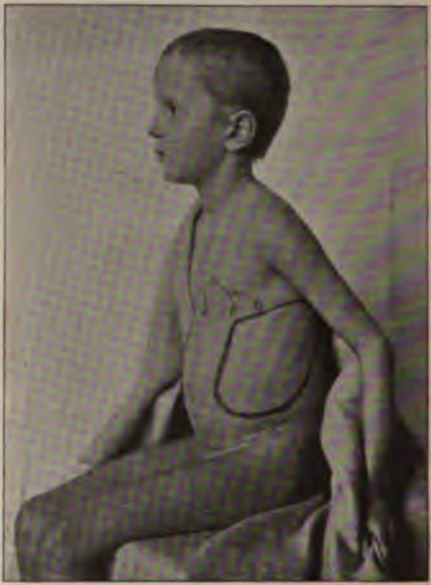
From this time the attacks entirely disappeared, the food was well digested, and the infant seemed perfectly well.

I have here in the wards to-day two cases (Cases 151 and 152) of malaria to show you.

One is this boy (Case 151), nine years old, who was admitted to my service on the 13th day of February.

He lived in a malarial district until one year ago. He had a slight cough, anorexia, malaise, night-sweats, and rapid loss of flesh for several weeks. The movements of the bowels were rather irregular. According to his mother's report, he had never before had any symptoms of malaria. On examining the child you will see that he is pale and emaciated. On physical examination you will find that there is resonance over both lungs, and on auscultation you will hear a few moist râles and an occasional sibilant râle. The area of cardiac dulness and the sounds of the heart are normal. T not enlarged, but the spleen, as you see, is very much increased in size, and I ha the limits of its enlargement in black. You see that the upper border rises the sixth rib in the axillary line, and extends down into the left inguinal re examination of the urine shows it to be normal.

Case 151.



Boy, 9 years old. Enlarged spleen. Plasmodium malariæ found in blood.

This is a case which represents the tertian form of malaria. The child had a chill until 3 P.M. two days after entering the hospital. The chill lasted hour, and was followed by sweating. A paroxysm of some kind, represented chill or by a decided rise in temperature with chilly sensations, occurred on the 21st, 23d, 25th, 27th, and 29th of February, March 2, and March 4, and on Ma was a decided rigor at 4 P.M. On March 8 the paroxysm occurred in the morni past twelve. On the morning of March 10 the paroxysm occurred at abou eleven, and was followed by marked sweating. Between the paroxysms the peared to be very well. He has had a fair appetite, and has gradually gained and strength.

On March 10, immediately after the paroxysm, the blood was examined by worth, and the plasmodium malariæ was found. A specimen of the blood, Wentworth has prepared to show you, is under this microscope (Plate V You will see the clusters of pigment in the erythrocytes in the various stages of opment of the parasites.

Here is the result of the examination of the blood:

BLOOD EXAMINATION 35. (Wentworth.)

Erythrocytes	2,935,0
Hæmoglobin	36 per c
Leucocytes	25,500
Small mononuclear	17 per c
Large "	27 "
Polynuclear	56 "
Eosinophiles	

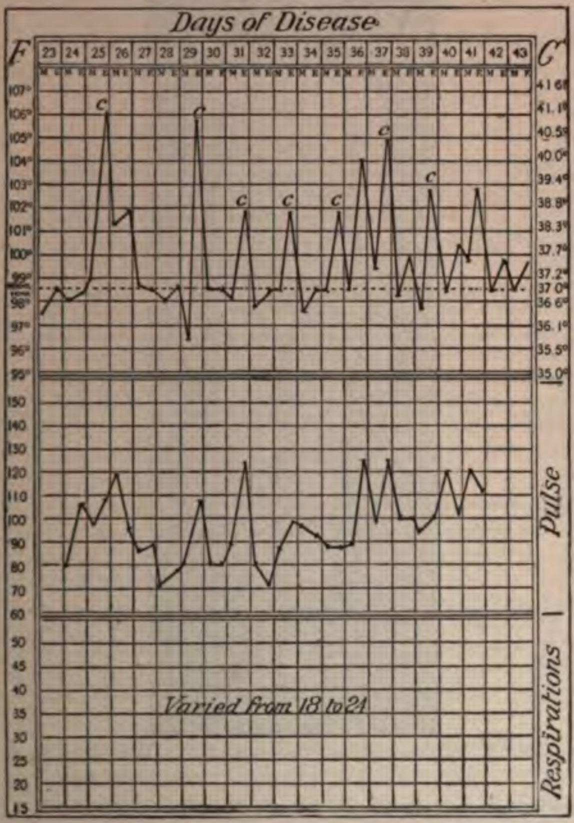
large number of the erythrocytes contained the plasmodium malariæ.

he large number of leucocytes pointed towards some complication, but none was at me discovered.

he chills continued on March 12, 14, and 16. On March 17 0.36 gramme (6 grains) inine were given six hours before the paroxysm was expected to return. On March re was no paroxysm. The quinine was given regularly three or four times a day for al days, and the paroxysms have not returned.

Here is the chart (Chart 6) representing the temperature and pulse of this case. The representing the disease are necessarily only approximate for the first twenty-two , and he is supposed to have entered the hospital on the twenty-third day of the dis- The first chill occurred on the twenty-fifth day, as is shown in the chart.

CHART 6.



Tertian form of malaria. (C means chill.)

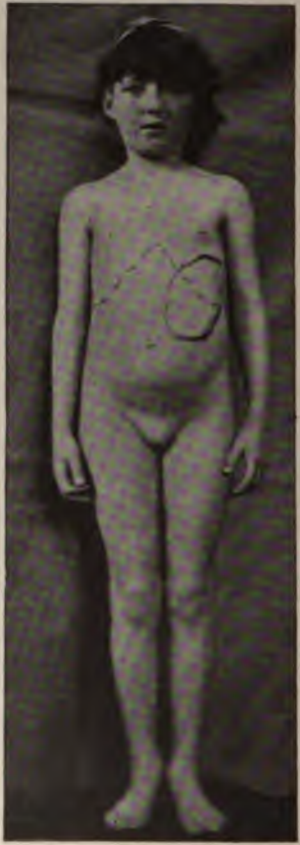
e subsequent history of this case was that the quinine was omitted, the chills did rn, the spleen recovered its normal size, the anæmia disappeared, and the child it, and left the hospital in good condition.)

Here is the second case (Case 152) of malaria to which I have referred.

A girl, nine years old, who entered the hospital also on the 13th of the month.

She represents, in contradistinction to the tertian form of malaria seen in the boy, a of the double tertian (quotidian) form. She has been living in a malarial district, but never had any previous symptoms of malaria, although a sister living in the same house has been affected by the disease. Four weeks before entering the hospital s had an attack of vomiting, nausea, and headache, without any apparent cause for the These symptoms occurred at intervals for two weeks, when she began to have chills occ ring every day at about 5 P.M. These chills continued, with the exception of four da until her entrance to the hospital.

CASE 152.



Girl, 9 years old. Enlarged spleen. Plasmodium malariæ found in the blood.

On examination you see that she is fairly developed and is very anæmic. On physi examination moist râles are heard over the bases of the lungs behind. The heart shows increase in the area of dulness, but there is a soft systolic murmur over the whole præco This murmur is most intense over the pulmonic area. The pulmonic second sound i accentuated. The murmur is heard in the jugular veins. An examination of the abdo shows it to be soft and tympanitic. The liver is enlarged, so that it extends 2.5 cm. (1 i below the border of the ribs. The edge of the spleen is plainly felt, and the percus dulness extends downward to the level of the umbilicus and upward as far as the s rib. I have designated it, as you see, by a black line. The urine is high-colored and a specific gravity of 1025, but is otherwise normal.

On the day of entering the hospital (the 13th) the child's temperature was raised, there was no chill. On the following day, the 14th, there was a chill at 4 P.M. On 15th there was a marked chill, with a considerable rise of temperature.

Immediately after the paroxysm an examination of the blood was made by Dr. W worth, with the following result:

BLOOD EXAMINATION 36. (Wentworth.)

throcytes	2,396,250
moglobin	30 per cent.
cocytes	5,000

smodium malariæ present.

noted that the splenic enlargement was greatest during the chill.

e 16th there was a chill, and the temperature rose to 40.6° C. (105.2° F.), the attained during the course of the disease.

e 17th and 18th the chills recurred.

e 18th 0.36 gramme (6 grains) of sulphate of quinine were given at 12.30 P.M.

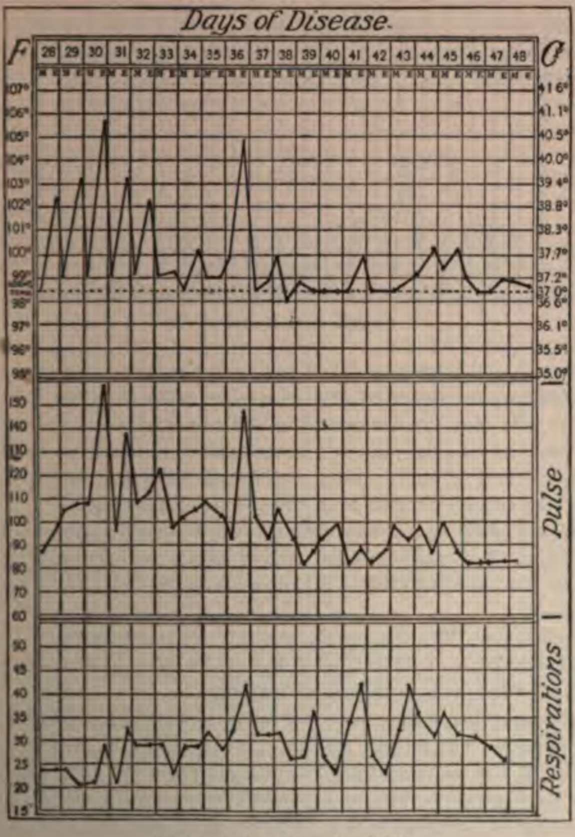
e 19th there was no rise in the temperature, and no quinine was given.

e 20th and 21st there were no chills, but a slight rise of temperature, and 0.12 grains) of quinine were given four times daily.

y, the 22d, she has just had a chill, and the temperature is 40.5° C. (105° F.).

s the chart of this case.

CHART 7.

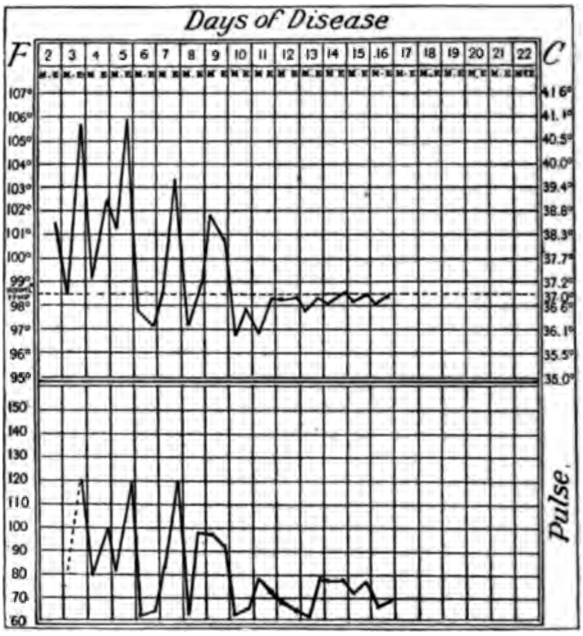


Double tertian form of malaria.

subsequent history of this case was as follows. 0.6 gramme (10 grains) of quinine n in the course of each twenty-four hours for the next sixteen days, the spleen growing smaller. As the temperature was still irregular, the quinine was then to 0.72 gramme (12 grains). The temperature remained normal for three days, was again slightly raised and irregular. Two weeks later the quinine was omitted, and the spleen was found to be normal in size. Ten days later the child left the hospital in good condition.)

I have here to show you the chart (Chart 8) of the temperature and pulse of a boy (Case 153) nine and one-half years old, who was under my care with malaria.

CHART 8.



Tertian form of malaria.

He had been well and strong, and had not been living in a malarial district, but had spent a few days in the early part of May in a place where malaria had been known to occur occasionally. On May 7, after returning to his home, he complained of feeling tired and dizzy. On the following day, May 8, he complained of headache and of feeling chilly. He had no appetite, and in the evening was found to have a temperature of 38.6° C. (101.5° F.). He had two movements from the bowels on that day.

On the next day, May 9, his temperature at 7 A.M. was normal. At 8.45 A.M. he complained of nausea, of headache, and of feeling chilly. He had no appetite. His temperature at 1 P.M. was 40.8° C. (105.5° F.), and his pulse 120. He appeared to be very nervous and irritable.

On the following day, May 10, he felt perfectly well and bright, had no headache, a good appetite, a temperature of 37.2° C. (99° F.), and a pulse of 80. He continued to feel well until 7.30 P.M., when he complained of headache.

On May 11 his morning temperature was found to be 38.3° C. (101° F.) and his pulse 80. He had no appetite, was restless and nervous, but slept for two or three hours. His temperature at 8 P.M. was 41° C. (105.8° F.) and his pulse was 120. At 10 o'clock, after having a sponge bath given to him at a temperature of 35° C. (95° F.), his temperature fell to 38.3° C. (101° F.). He slept well during the night, and perspired freely. The spleen was found to be somewhat enlarged on this day, and nothing else abnormal was discovered on physical examination.

On the morning of May 12 the temperature was 36.5° C. (97.8° F.) and the pulse was 60. He felt perfectly well and bright, and had a good appetite. The movements of the bowels were rather loose.

On May 13 the morning temperature was normal and his pulse was 60. He felt well and bright until noon, when he had a rigor lasting twenty minutes. After the rigor he was sleepy and tired, and at 3 P.M. the temperature was 40.5° C. (105° F.) and the pulse 120. At 6 P.M. he felt perfectly well again, his appetite returned, his temperature fell to 38.6° C. (101.8° F.) and his pulse to 100.

On the following day, May 14, he felt perfectly well, had a good appetite, and at 7 A.M. had a temperature of 36.1° C. (97° F.). On this day he had 0.06 gramme (1 grain) of quinine given to him three times a day.

On the following day, May 15, the record was that he had passed a quiet night, and that he woke early and seemed nervous. 0.3 gramme (5 grains) of quinine were given to him at 6 A.M. His temperature at 7 A.M. was 37.1° C. (98.9° F.). At 9 o'clock he began to grow sleepy; at 9.30 his temperature was 38.6° C. (101.6° F.), and at noon it was 41° C. (105.5° F.). At 7.30 A.M. his pulse was 82, at 9.30 A.M. 100, and at noon 120. At the time that he was having the high temperature his urine was large in amount and pale in color. At other times it was normal.

0.12 gramme (2 grains) of quinine were given on the following day, May 16, when the record was that he had passed a quiet night and that he waked at 2 A.M., seeming to be exhausted and complaining of feeling weak. His temperature was 35.5° C. (96° F.) and his pulse 48 and very weak. Thirty drops of brandy were given to him, and his pulse soon rose to 75 and was of a better character. He then slept until 7 A.M. At 7.30 A.M. his temperature was 35.8° C. (96.6° F.) and his pulse was 60. He appeared to feel bright and well all day, had a good appetite, and for the first time had a normal movement of the bowels. The temperature in the evening was 36.6° C. (98° F.) and the pulse was 60. At 7.30 P.M. he complained of slight pain in the bowels.

On the following day, May 17, 0.36 gramme (6 grains) of quinine were given at 5.15 A.M. His temperature remained normal all day, and his pulse varied from 70 to 80. He felt a little sleepy at noon, but his skin was natural. The bowels were moved regularly, and there were no abnormal symptoms.

On the following day, May 18, he was given 0.36 gramme (6 grains) of quinine at 5.30 A.M. He was perfectly well and bright all day, and had more appetite. He was given one grain of quinine three times during the day in addition to the 0.36 gramme (6 grains) at 5.30 A.M.

On the following day, May 20, he was out of bed and dressed all day, feeling perfectly well.

From this time until the 27th he continued to take 0.3–0.6 gramme (5–10 grains) of quinine during twenty-four hours, and he has since been perfectly well, with no recurrence of the malarial symptoms.

(No examination of the blood was made.)

I have also here to report to you the records of two infants who apparently were suffering from the effects of the plasmodium malariæ, although no examination of their blood was made.

The first one (Case 154) was one year and ten months old. This infant had lived in a malarial district until within a few weeks of the time when I saw him in Boston.

The history which was given to me by his mother was that for several weeks he had had attacks, represented by a chill or chilly sensations, occurring every day at about noon. These attacks had recurred for about a week or ten days before I saw him. In connection with the chill and the fever the infant usually became unconscious, and its feet and hands were cold and clammy.

0.06 gramme (1 grain) of quinine was given to the infant on the 29th of April, and on the following day none of the usual manifestations occurred at noon, but at about 4.30 P.M. he had a chill and a slight rise of temperature, but was not unconscious. 0.03 gramme ($\frac{1}{2}$ grain) of quinine was then given, and on the following day, April 30, 0.06 gramme (1 grain) of quinine at 10.30 A.M. On this day there was a decided chill, and the rectal temperature rose to 40.5° C. (105° F.). During the attack the child breathed rapidly; its feet, hands, and nose became cold, and it was practically unconscious for some minutes until its circulation was restored by injections of warm water and brandy. 0.03 gramme ($\frac{1}{2}$ grain) of quinine was then given three times during the twenty-four hours. On the following day none of these abnormal symptoms occurred. On the next day 0.03 gramme (½ grain) of quinine was given in the morning and again at night, and this dose was continued for a few days.

From this time the symptoms of malaria entirely disappeared, the infant grew less and less emaciated, became stronger, had a good appetite, and continued to thrive.

No enlargement of the spleen was detected in this case.

The next infant (Case 156) was nineteen months old, and was brought from a decidedly malarial district.

It had previously been well until three weeks before it was brought to be treated for the following symptoms. At the time when its bath was given to it, which was between 11 and 12 in the morning, it had symptoms characterized by drowsiness and cyanosis, and it would fall asleep, and after about half an hour would wake up bright and well. These attacks, though short in duration, were very alarming and apparently serious, as, although the infant did not have any pain or convulsions, it could not be roused while in the attacks, and became so blue and cold that it was feared that it might die in one of them. At the time of the attacks the rectal temperature varied somewhat, but was usually about 38.3° C. (101° F.).

The treatment of this case was with sulphate of quinine, sometimes given by the mouth and sometimes by means of rectal suppositories. After the administration of the quinine for four or five days the attacks entirely ceased and did not return. The infant from that time continued to thrive.

This table (Table 89) contains references to most of the important articles which up to the present time have been published on the blood. You must remember, however, that it is not a general literature of the blood, but only that of an early period of development. It is the source from which I have drawn most of my information in the endeavor which I am making to elucidate the subject for you, and in this way I acknowledge what I have received from other authors.

TABLE 89.

1. ALT UND WEISS	Anæmia Infantilis Pseudo-Leukæmica. Centralblatt für die Med. Wissenschaft, 1892, Nos. 24 u. 25.
2. ANDREESEN	Ueber die Ursachen der Schwankungen im Verhältnisse der rothen Blutkörperchen zum Plasma. *Dissert.* Dorpat, 1888.
3. ARNHEIM UND WIDOWITZ	Scarlatina. Morbilli.
4. BAGINSKY	Archiv für Kinderheilk., Bd. xiii., 1891.
5. BAYER	Ueber die Zahlenverhältnisse der rothen und weissen Zellen im Blute von Neugeborenen und Säuglingen. *Dissert.* Bern, 1881.
6. BOTKIN	Beitrag zur pathologischen Anatomie der Milz bei Pneumonia Crouposa. *Dissert.* St. Petersburg, 1892.
7. BOUCHUT ET DUBRISAY	Gazette Médicale de Paris, 1878.
8. CADET	Etude physiologique des Eléments figurés du Sang. *Dissert.* Paris, 1881.
9. CANON	Ueber eosinophile Zellen und Mastzellen im Blute Gesunder und Kranker. Deutsche Med. Wochenschrift, 1892, No. 10.
10. COHNSTEIN UND ZUNTZ	Pflüger's Archiv, Bd. xxxiv., 1884.
11. DAVIDOFF	Untersuchungen über die Beziehungen des Darm-Epithels zum lymphoiden Gewebe. Archiv für mikroskopische Anatomie, Bd. xxix., 1887.

TABLE 89.—*Continued.*

12.	Demme	17. und 18. Bericht des Berner Kinderspitals, 1880 und 1881.
13.	Demme	Zwei Fälle von perniciöser Anämie. Jahresber. a. d. Berner Kindersp., No. 28.
14.	Denis	Recherches expérimentales sur le Sang, Paris, 1830.
15.	Dupérie	Sur les Variations physiologiques dans l'Etat anatomique du Sang. Thèse de Paris, 1878.
16.	Ehrlich	Farbenanalytische Untersuchungen zur Histologie und Klinik des Blutes. Berlin, 1891. I. Theil.
17.	Einhorn	Ueber das Verhalten der Lymphdrüsen zu den weissen Blutkörperchen. *I. D.* Berlin, 1884.
18.	Engelsen	Virchow's Jahresbericht, 1884.
19.	Escherich	Ein Fall von perniciöser Anämie. Wien. Klin. Wochensch., 1892.
20.	Fano	Lo Sperimentale, 1880.
21.	Fischl	Der gegenwärtige Stand der Lehre von kindlichem Blute. Sammelreferat. Prager Med. Wochenschrift, No. 12 u. f., 1892.
22.	Fischl	Zur Histologie des kindlichen Blutes. Zeitschrift für Heilkunde, 1892.
23.	Flemming	Zellsubstanz, Zellkern, Zelltheilung.
24.	Gabritschewsky	Grundriss der norm. und patholog. Morphologie des Blutes, 1891.
25.	Guffer	Revue Mensuelle, 1876.
26.	Gundobin	Ueber die Morphologie und Pathologie des Blutes bei Kindern. Jahrb. f. Kinderheilk., Bd. xxxv., 1893.
27.	Halla	Ueber den Hämoglobingehalt des Blutes und die quantitativen Verhältnisse der rothen und weissen Blutkörperchen bei acute fieberhafte Krankheiten. Zeitschrift f. Heilk., 1893, Bd. iv.
28.	Hammeder	Centralblatt für Gynäkologie, 1879.
29.	Hammerschlag	Ueber das Verhalten des spec. Gewichtes des Blutes und Krankheiten. Wien. Klin. Wochensch., 1891, und Centralblatt für Klin. Med., 1891, No. 44.
30.	Hayem	Du Sang et de ses Altérations anatomiques, Paris, 1889.
31.	Hayem	L'Anémie des Nourrissons. Gazette des Hôpitaux, 1889, No. 30.
32.	Hetaguroff	Pathologische Anatomie des Blutes bei Unterleibstyphus. *Dissert.* St. Petersburg, 1891.
33.	Hock und Schlesinger	Blutuntersuchungen bei Kindern. Vorläufige Mittheilungen. Centralblatt für Klin. Med., 1891.
34.	Hock und Schlesinger	Hämatologische Studien. Franz Deuticke, Leipzig und Wien, 1892.
35.	Von Jaksch	Ueber Leukämie und Leukocytose im Kindersalter.
36.	Von Jaksch	Ueber Diagnose und Therapie der Erkrankungen des Blutes. Prager Med. Wochensch., 1890, Nos. 22, 23, 31, 33.
37.	Lloyd Jones	Journal of Physiology, vol. viii. Part 1, 1887.
38.	Kikodse	Pathologische Anatomie des Blutes bei Pneumonia Crouposa. *Dissert.* St. Petersburg, 1890.
39.	Klein	Centralblatt für Med. Wissensch., 1872.

TABLE 89.—*Continued.*

40. KLEIN	Untersuchungen über Formelemente des Blutes u ihre Bedeutung für die praktische Medicin, 18
41. KÖLLIKER	Ueber die Blutkörper eines menschlichen Embry Zeitschr. für rat. Med., 1846, No. 4.
42. KOTSCHETKOFF	Morphologische Veränderungen des Blutes b Scharlach. *Dissert.* St. Petersburg, 1891.
43. KRÜGER	Ueber das Verhalten des fötalen Blutes im Mome der Geburt. *Dissert.* Dorpat, 1886.
44. LAACHE	Quoted by Reinert.
45. LEICHTENSTERN	Untersuchungen über der Hämoglobingehalt, Lei zig, 1878.
46. LÉPINE	Comptes-rendus de la Société de Biologie, 1876.
47. VON LIMBECK	Zeitschrift für Heilkunde, 1890.
48. VON LIMBECK	Ueber entzündliche Leucocytose. Wiener Me Presse, No. 43, 1889.
49. VON LIMBECK	Grundriss einer Klin. Pathologie des Blutes. Jen 1892.
50. LITTEN	Zur Pathologie des Blutes. Berl. Klin. Wochensch 1883 und 1889.
51. J. LOOS	Die Anämie bei hereditärer Syphilis. Vorläuf Mittheilung. Wien. Klin. Wochensch., No. 2 1892.
52. J. LOOS	Ueber das Vorkommen kernhaltiger röther Blu zellen bei Kindern. Wien. Klin. Wochensch No. 2, 1891.
53. LUZET	Etude sur les Anémies de la première Enfanc *Dissert.* Paris, 1891.
54. MANDYBUR	Vorkommen und diagnostische Bedeutung der ox philen und basophilen Leucocyten im Sputum Wien. Med. Wochenschrift, Nos. 7 bis 9, 1892.
55. MARAGLIANO	Beitrag zur Pathologie des Blutes. Berliner Kli Wochensch., No. 31, 1892.
56. MOLESCHOTT	Wien. Med. Wochensch., 1854, No. 8.
57. MONTI UND BERGGRÜN	Die chronische Anämie im Kindersalter. Leipzi (Vogel), 1892.
58. MÜLLER	Klinische Beobachtungen zur Verdauungsleukoc tose. Zeitschr. für Heilk., 1890.
59. MÜLLER	Ueber Mitose an eosinophilen Zellen. Archiv f Exp. Path. und Pharm., 1891.
60. MÜLLER	Ueber Leukämie. Deutsches Arch. für Klin. Med Bd. xlviii. Heft i. u. ii.
61. MÜLLER UND RIEDER	Ueber das Vorkommen und die klinische Bedeutun der eosinophilen Zellen im circulirenden Blute d Menschen. Deutsches Arch. für Klin. Med., B xlviii. Heft i. u. ii.
62. H. MÜLLER	Die progressive perniciöse Anämie. Zurich, 1877.
63. MÜLLER	Blutbildung. Sitzungs-Bericht der Akad. d Wiss., 1889.
64. NASSE	Untersuchungen zur Physiologie und Patholog des Blutes. Wagner's Handwörterb., Bd. i. S 138.
65. NAUNYN	Ueber den Hämoglobingehalt des Blutes bei de verschiedenen Krankheiten. Corresp.-Blatt f Schweizer Aerzte, 1872, No. 14.

TABLE 89.—*Continued.*

EUMANN	Centralblatt für Med. Wissensch., 1869, und Archiv d. Heilk., Bd. x.
EUSSER	Klinische hämatologische Mittheilungen. Wien. Klin. Wochensch., Nos. 3 und 4, 1892.
TTO	*Inaug. Dissert.* Halle a. S., 1893.
ANUM	Die Blutmenge neugeborener Hunde und das Verhalten ihrer Blutbestandtheile verglichen mit denen der Mutter und älterer Geschwister. Virchow's Archiv, Bd. xxix.
ARROT	L'Athrepsie. Paris, 1877.
PICK	Untersuchungen über die quantitativen Verhältnisse der Blutkörperchen bei Variola. Archiv für Dermat. u. Syph., Bd. xxv., 1893.
OPOFF	Pathologische Anatomie des Blutes und der blutbereitenden Organe unter dem Einflusse der Hämoglobinurie hervorrufenden Substanzen. *Dissert.* St. Petersburg, 1892.
REYER	Physiologie des Embryo, 1886.
UINCKE	Ueber perniciöse Anämie. Volkmann's Samml. Klin. Vorträge, No. 100.
UINCKE	Weitere Beobachtungen über perniciöse Anämie. Deutsches Arch. für Klin. Med., Bd. xx.
EINERT	Die Zählung der Blutkörperchen. Leipzig (Vogel), 1891.
EDER	Beiträge zur Kenntniss der Leukocytose. Leipzig (Vogel), 1892.
ENGER	Archiv für Gynäkologie, Bd. xxxiii., 1888.
HLI	Archiv für Gynäkologie, Bd. xxxiii. S. 181.
HAFFER	Beiträge zur Histologie der menschlichen Organe. Sitzungs-Bericht der Akad. der Wissenschaften, Band c., Wien, 1891.
HERENZISS	Untersuchungen über das fötale Blut. *Dissert.* Dorpat, 1888.
HIFF	Ueber das quantitative Verhalten der Blutkörperchen und des Hämoglobins bei neugeborenen Kindern, etc. Zeitschrift für Heilkunde, Bd. xi., 1890.
HMALZ	Zur Untersuchung des spec. Gewichtes des menschlichen Blutes. Deutsches Arch. für Klin. Med., Bd. xlvii.
. SCHMIDT	Demonstration mikroskopischer Präparate zur Pathologie des Asthma. Verhandl. des XI. Internat. Congresses für innere Med.
HUECKING	Berliner Klin. Wochenschr., 1879.
HWARTZE	Ueber eosinophile Zellen. *I. D.* Berlin, 1880.
NATOR	Berliner Klin. Wochenschr., 1882, No. 35.
LBERMANN	Zur Hämatologie des Neugeborenen. Jahrb. für Kinderh., Bd. xxvi. Heft ii.
SOMMA	Anæmia Splenica Infantilis. Allgemeine Wiener Med. Zeitung, 1891.
RENSON	Jahresbericht über die Fortschritte der Physiologie und Anatomie, S. 192–197.
ERLIN	Deutsches Archiv für Klin. Med., 1889.
OJE	Ueber Leucämie und Pseudoleucämie. Berl. Klin. Wochenschr., 1892, No. 12.

TABLE 89.—*Continued.*

93.	Tschistowitsch	Bolnitschnaja Gazette, Botkin, 1890.
94.	Tümas	Gazetta, Botkin, 1885.
95.	Uskoff	Blut als Gewebe, 1890.
96.	Weiss	Das Vorkommen und die Bedeutung der eosinophil Zellen und ihre Beziehungen zur Bioblaste theorie Altmanns. Wiener Med. Presse, N 41–44, 1891.
97.	Weiss	Beiträge zur histologischen und mikrochemisch Kenntnisse des Blutes. Wien, 1892, Alfr Hölder.
98.	Weiss	Die Wechselbeziehungen des Blutes zu den Organe etc. Jahrbuch für Kinderheilk., Band xxx 1893.
99.	Welcker	Prager Vierteljahrschrift, IV. Jahrgang, 1854.
100.	Widowitz	Hämoglobingehalt gesunder und kranker Kind Jahrb. für Kinderheilk., 1888.
101.	Wiskemann	Zeitschrift für Biologie, Bd. xii., 1876.
102.	Woino-Oransky	Beiträge zur Morphologie des Blutes der Neug borenen. *Dissert.* St. Petersburg, 1892.

DIVISION VIII.

DISEASES OF THE NEW-BORN.

LECTURE XVIII.

MATERNAL IMPRESSIONS.—THE HEAD.—THE NECK.

UNDER the designation "diseases of the new-born" I shall now describe to you a series of cases which occur so early in life that they are most conveniently placed in a class by themselves. I shall not attempt to dwell upon all the conditions which are met with either at birth or within the first few weeks of life. That would require more space and time than the scope of these lectures will allow.

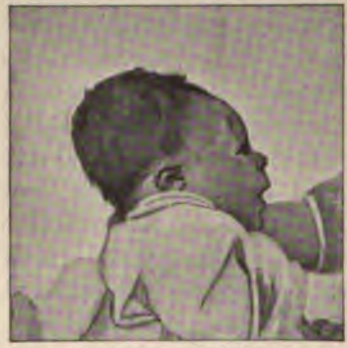
The diseases which we speak of as "diseases of the new-born" are distinct from those which are acquired later in life, in that they represent in almost every case an arrest of the normal development which should occur during intra-uterine life. This I have already referred to in my introductory lecture (Lecture I., page 19), and I only wish to impress again upon your minds that a stage of development which is normal at a certain period of intra-uterine life becomes abnormal if it persists to a later period, and that this persistence of an early stage of development constitutes in the great majority of cases what is known as congenital malformation. Such a failure of development may be the result of intra-uterine inflammation, which, either by crippling the various functions or by arresting the normal intra-uterine growth, produces a condition of disease at birth. In many cases, however, the causes are so obscure as to elude our usual methods of examination. "Diseases of the new-born" may also be made to include certain abnormal conditions which arise immediately after birth or in the early days of life.

Although many of these affections must pass into the hands of the surgeon for treatment, yet it is very important for the medical practitioner to be able to recognize at once their true nature and their significance. I shall, therefore, in this lecture attempt in a few words to tell you of some of the more common surgical affections of the new-born, as well as of those that are of a purely medical nature. In speaking of these diseases I shall, for the purpose of simplicity, classify them into diseases of the head and neck, diseases of the trunk, diseases of the extremities, and general diseases.

MATERNAL IMPRESSIONS.—A few words should be said concerning the subject of maternal impressions. For many years there has been accumulating a considerable amount of evidence showing that a violent mental impression made upon a woman who is at the time carrying a child may be followed by a physical or mental defect in the child which bears a striking relation in character to the impression made upon the mother. Thus, Sir Walter Scott narrates that King James the First could not endure the sight of a drawn sword. This feeling has been attributed by those who believe in maternal impressions to the terror which his mother experienced at witnessing the murder of Rizzio. Still more numerous are the facts adduced to prove that bodily defects, such as harelip, club-foot, and hairy mole, may be caused by strong impressions of pain or terror experienced by the mother at the time when the fœtus is in a certain stage of intra-uterine development. Interesting as these instances are, I think it is the general belief that nothing more has been proved than that they depend on a coincidence. The final decision on this obscure subject must rest on future investigation, and may cause us to guard a woman during her pregnancy from all unpleasant impressions with far more care than we do at present.

THE HEAD.—The normal average head at birth may be misshapen from various causes. Of the conditions which may cause unusual appearances, I shall refer merely to the most common. One of these conditions is called *caput succedaneum*, a case of which I have here to show you.

CASE 156.



Caput succedaneum. Male, 2 hours old.

CAPUT SUCCEDANEUM.—This infant (Case 156) is a male, two hours old, presents a swelling over the right parietal bone extending back to the occiput and causing an irregular tumor and a great increase in the antero-posterior diameter of the head. You will notice that the tumor does not fluctuate.

The presentation was occiput left anterior, and no instruments were used. You see that the swelling corresponds to the place where there was the least pressure,—that is, the presenting part. It is needless to say that this caput succedaneum requires no treatment, as it gradually disappears of itself by absorption in a few days. It is simply a swelling of the scalp caused by a passive congestion with extravasation of blood and lymph into the connective tissue external to the pericranium.

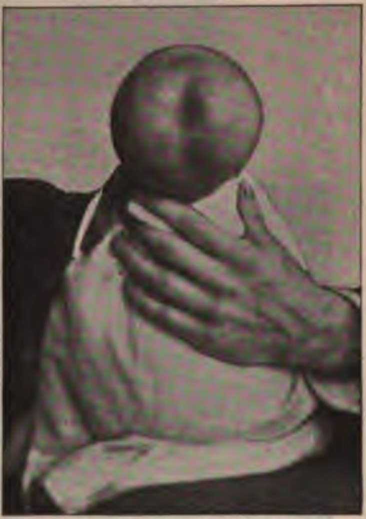
Caput succedaneum must be carefully distinguished from another swelling of the scalp, *cephalhæmatoma*, which may occur in connection with it, and which appears as the caput succedaneum disappears.

CEPHALHÆMATOMA.—During labor a hemorrhage may take place from the blood-vessels of the head which gives rise to a tumor in one of hree situations: (1) between the occipito-frontalis aponeurosis and the peri-steum; (2) between the periosteum and the skull; or (3) between the skull nd the dura mater. The first two are known as external cephalhæmatoma, he last as internal cephalhæmatoma. The cause cannot be entirely pressure ver the presenting part, as they have been found in breech presentations.

External Cephalhæmatoma.—By far the most common form is that in vhich the tumor has formed between the skull and the periosteum. It hows itself as an irregular circular swelling over a parietal bone, and gives on palpation a distinct feeling of fluctuation. The skin over it is not discolored or reddened. In those that have existed for a few days a bony wall can be felt surrounding the tumor, the edges of which give a crackling sensation under the finger. In this stage it may strongly suggest a fluid tumor coming through a circular hole in the skull.

The case (Case 157) which I have here to show you to-day is one of double cephalhæmatoma of the external variety; that is, it is an extravasation of blood under the pericranium. Its base, corresponding to the denuded bone, is oval or circular. You will notice the bulging tumors on each side of the sagittal suture with a deep sulcus between them. On palpation you will get fluctuation, and on feeling the circumference of the tumor an elevation and crackling sensation as though you were touching fine crystals of ice on the edge of water which is beginning to freeze.

Case 157.



Double cephalhæmatoma. Infant, 4 days old.

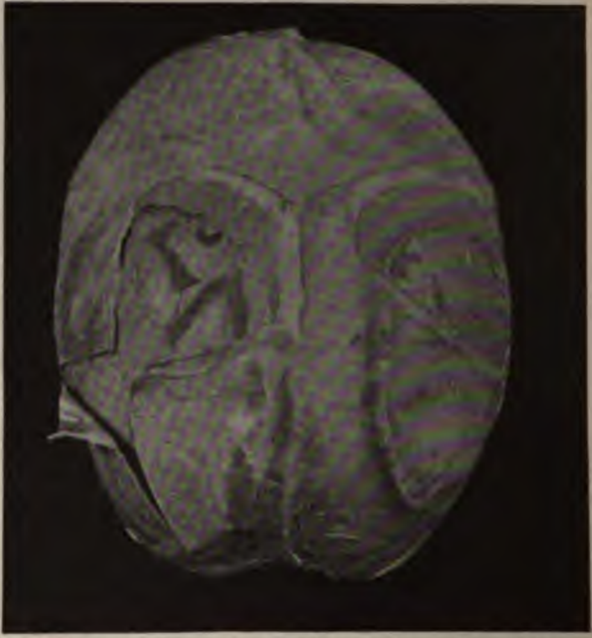
Cephalhæmatoma is distinguished from caput succedaneum by its sharp limitation to one of the parietal bones, by its fluctuation, and, if seen late, by its surrounding bony wall. It can be diagnosticated positively by the withdrawal of some of the fluid by a hypodermic syringe. Another condition which may simulate it somewhat is a depressed fracture. The differential diagnosis from this latter condition can best be made by remembering the fact that the resistant rim of the cephalhæmatoma is raised above the level of the surrounding bone, and is somewhat compressible, while on the inside it can be felt to slope evenly towards a fluctuating centre. In fracture no such arrangement occurs.

I shall now call your attention to this preparation (Fig. 86, page 406) of a double external cephalhæmatoma from the Warren Museum.

You see on the left side of the skull (the right side of the picture) the integument has been nearly removed, showing a raised bony rim.

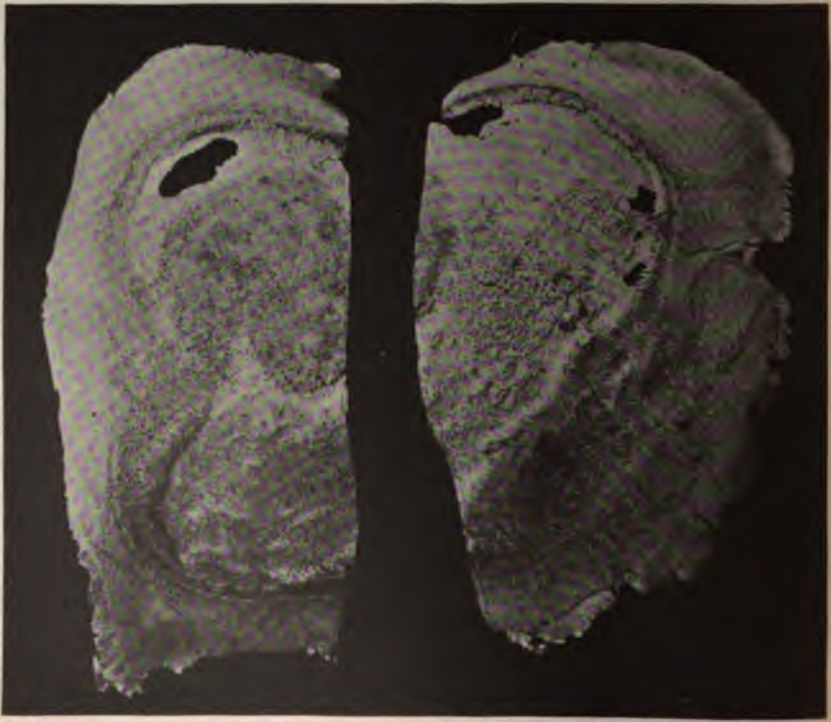
On the right side of the skull (the left side of the picture) the integument has been cut off and partially deflected, showing the cavity which contained the effused blood.

FIG. 86.



Double external cephalhæmatoma. Both parietal bones. Warren Museum, Harvard University.

FIG. 87.



External cephalhæmatoma. Parietal bone dissected. Warren Museum, Harvard University.

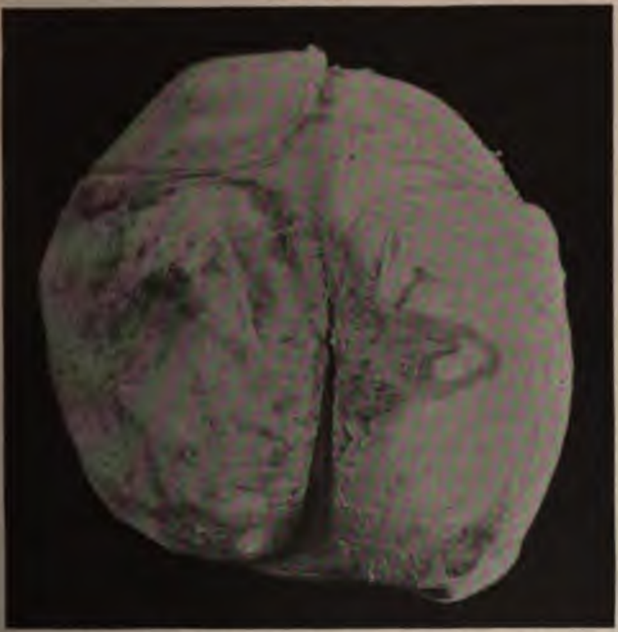
The next specimen (Fig. 87) is a parietal bone dissected so as to show the condition of the bone in a case of external cephalhæmatoma.

This specimen shows well the raised rim and the porous condition of the bone underlying the tumor. In two or three places the bone substance has entirely disappeared.

Internal Cephalhæmatoma.—Internal cephalhæmatoma is situated between the inner surface of the skull and the dura mater, and is rare. It is at times found in connection with the external variety.

The prognosis in this class of cases is bad. They are usually fatal, and there is no known treatment which can save them. I have here to show you the preparation (Fig. 88) of a skull taken from a case (Case 158) of internal and external cephalhæmatoma.

FIG. 88.



Internal and external cephalhæmatoma. Warren Museum, Harvard University.

The specimen was taken from an infant which was born at the Lying-in Hospital. Its death was caused by a large cerebral hemorrhage resulting from the internal cephalhæmatoma. It shows only the external cephalhæmatoma, which occupies the left parietal and occipital regions (shown on left of picture). Corresponding to this external cephalhæmatoma was a large effusion of blood occupying a space about 2.5 cm. (1 inch) in diameter, and lying between the dura mater and the brain substance, which was compressed by it.

MENINGOCELE.—By the term meningocele is understood a protrusion of some part of the membranes of the brain through a hole left in the cranial wall by defective ossification. In some instances this is caused by an intra-uterine hydrocephalus. These tumors generally contain some of the cerebro-spinal fluid in the bag of membrane. Such fluid can often be reduced into the skull by gentle pressure, but at the risk of bringing on symptoms of cerebral disturbance.

This case (Case 159) shows a small meningocele above the left ear about 2.5 cm. (1 inch) in diameter.

Some fluid was withdrawn from it by an aspirating needle, and the contents of the sac proved to be serous without cells. The sac refilled after tapping. No more extensive operation on it has so far been undertaken.

Case 159.

Meningocele. Female, 3 years old.

The history of this case is that the child is rhachitic. It had a fall some time ago and struck its head. Nothing abnormal was noticed about the child previous to the fall, but since the accident this swelling appeared above and behind the ear. The swelling increases in size when the child cries, is soft, fluctuating, and not tender. The knee-jerks and sensation are normal. The ophthalmoscopic examination discloses nothing abnormal.

A much more serious condition is shown in the meningocele of this infant (Case 160).

It is a male, and was two weeks old when operated upon. Behind its left ear was an irregular tumor about 7.5 cm. (3 inches) long. The ear was pushed forward, and appeared to be growing from the tumor. The labor was normal, and the infant at birth was perfectly healthy and well formed, except for the tumor, which was congenital. On examination the tumor was found to be fluctuating and translucent. There were large veins on its surface. Pressure on the tumor caused no symptoms. No impulse could be felt on crying, nor did pressure cause any cerebral symptoms. On aspirating it, 45 c.c. (1½ ounces) of a clear reddish fluid were withdrawn. This fluid contained red blood-corpuscles and a few endothelial cells. No unfavorable symptoms followed the aspiration. After the withdrawal of the fluid two openings could be felt, the anterior probably connecting with the external auditory meatus and the posterior with the anterior fontanelle. The tumor was increasing in size so rapidly that an operation was decided upon. On removing it an opening in the skull large enough to admit two fingers was found.

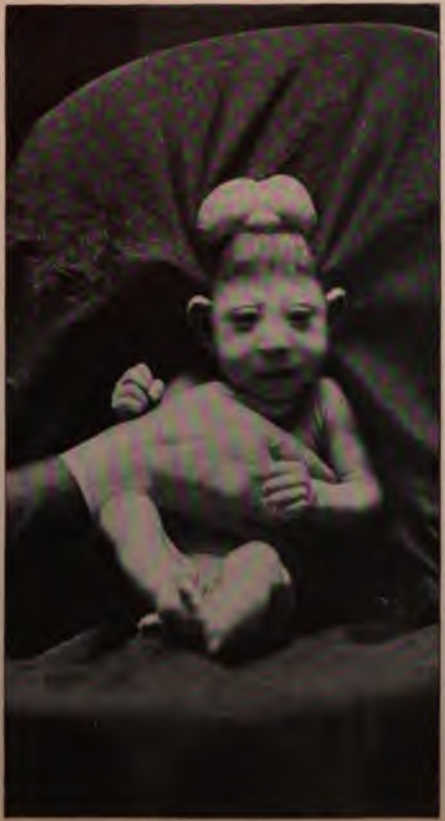
The child made a rapid recovery from the operation, and now has only a scar behind the ear. There were no cerebral symptoms. During convalescence and up to the present time the child has seemed to be mentally bright.

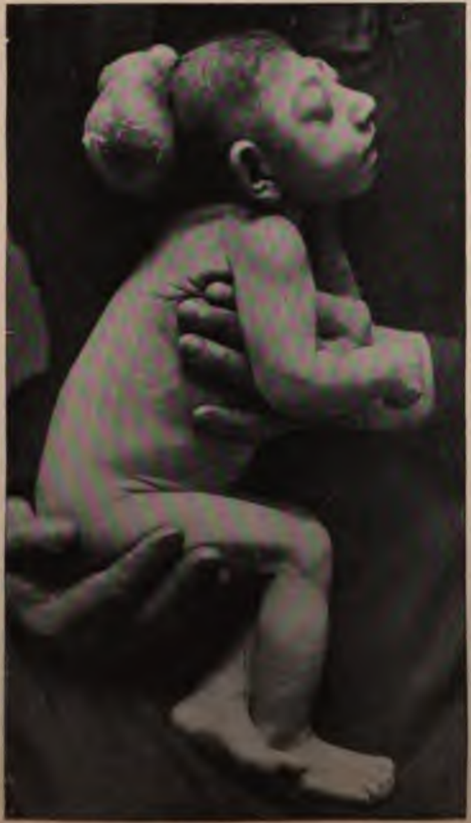
ENCEPHALOCELE.—Still more common than the pure meningocele is that condition in which the hernia contains some of the cerebral substance as well as the membranes. This condition is called encephalocele; or if, as is often the case, it contains a portion of a dilated ventricle, so that the tumor is filled with cerebro-spinal fluid, it is known as hydro-encephalocele or as hydro-encephalo-meningocele.

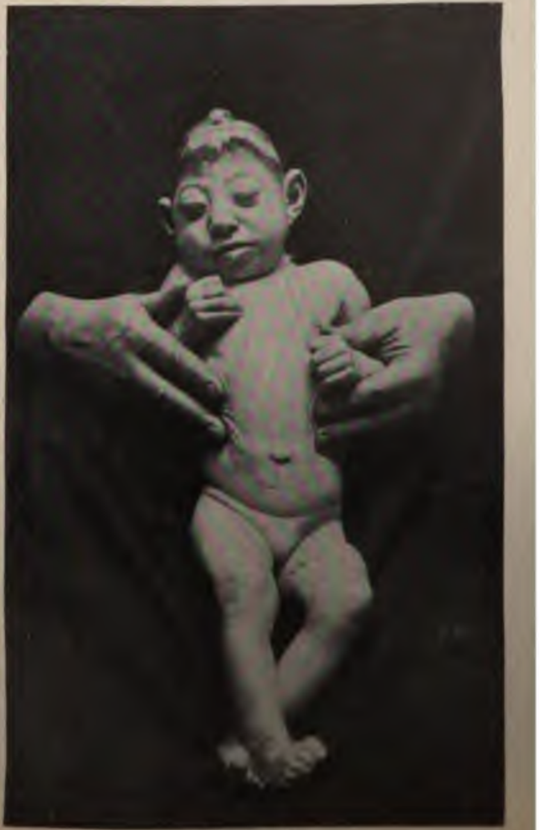
Here are some photographs of a remarkable case (Case 161) of hydro-encephalocele which was treated by Dr. Lovett in the hospital.

The infant from the time of its birth had tonic and clonic convulsions, occurring usually as often as once in three hours. It was brought to the hospital when it was two months old. It was well formed in every way, except that it had a tumor on the back of its head which was at least one-third as large as its skull. The tumor was only partly covered with skin, the upper part of it being a thin translucent membrane. It communicated with

CASE 161.


Before operation.


Female, 2 months old. Hydro-encephalocele.


After operation.

ain through a large square hole in the back of the skull. The tumor fluctuated y and appeared to be a multilocular cyst, for when it was aspirated only a part of the ned fluid could be withdrawn.

he tumor was removed by Dr. Lovett and the wound sewed up tightly. The cyst und to contain a viscous fluid with slight flakes in it which proved to be particles of al substance.

he convulsions immediately became less frequent, and ultimately on treatment with de of potash disappeared almost entirely.

he infant in other respects was very little affected by the operation, and recovered y. After remaining in the hospital two weeks it was taken to its home, where it died months later of some intercurrent affection.

egarding these tumors in general, it is enough to say that you should with suspicion any fluctuating swelling that seems to have a deep hment in the neighborhood of one of the cranial sutures. The most uent seat of these tumors is in the occipital region and at the root of nose. Their treatment has not proved very successful. Some few may lily decrease of themselves and ossification may block up the abnormal ing. Pressure and the injection of Morton's fluid have both been , and in some cases have been attended with success. At present the ative plan of treatment is considered the best. Without interference tendency is usually towards rupture of the hernia, convulsions, and h.

NENCEPHALIA.—As you have been taught in your course on embry- y, the cerebro-spinal system is formed from the medullary tube, which is e by the infolding of epiblast along the medullary groove: if the for- on of the medullary tube is for any reason incomplete, or if the dorsal of the tube is destroyed, the cerebrum or part of the cerebral axis will in rudimentary. According to the amount of interference with the lopment we may find more or less of the brain remaining in a rudi- tary condition, and thus producing greater or less degrees of what is d anencephalia. Total anencephalia is rare. Partial anencephalia is h more common. These cases are not of especial interest to us, as it is ptional for them to live beyond a few days.

ONGENITAL HYDROCEPHALUS.—One of the more common malfor- ons of the head is a hydrocephalic condition at birth. It is called enital hydrocephalus, and I shall describe it in a later lecture (Lecture X., page 634), on diseases of the brain.

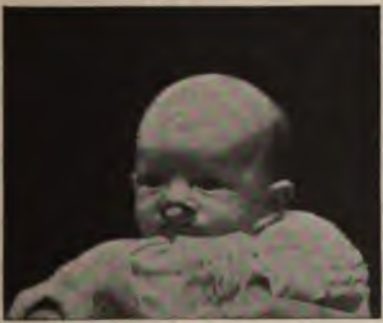
HARELIP.—The clearest way in which I can describe to you the malfor- on which I am now to consider is to remind you in a few words of the ner in which the parts around the mouth of the embryo are formed. can then see at a glance how a failure of any part of the process in development of that region will give rise to the several defects known ingle or double harelip and cleft palate. At first the fore-gut of the ryo does not communicate with the outside, but ends blindly under the rior region of the hind-brain. Over the end of the fore-gut curve the l-brain and fore-brain, causing a prominence on the ventral surface of the embryo. As the heart develops, another prominence is formed below the end of the fore-gut, and between these two prominences a wide shallow pit is found. At the bottom of this pit there is but a single velum, which separates the end of the fore-gut and the primitive mouth or stomodæum; later the velum is broken through and the two cavities form one canal. Above, this primitive mouth is bounded by the fronto-nasal process. Below, the boundary is made by the first visceral or mandibular arch, which has grown around the fore-gut from each side and has joined in the middle in front. The sides of the upper part of the buccal cavity are made by the maxillary processes, which growing from the base of the mandibular arch fill up the gap between it and the fronto-nasal process. The sides of the mouth are completed by the formation of the cheek-plates. The beginning of what in later life is to become the organ of smell is in the form of two small depressions, called the olfactory pits, in the sides of the fronto-nasal process, and immediately underlying the fore-brain. In the process of development these pits deepen and are partially surrounded by a semicircular ridge. The thickened inner edge of each olfactory pit now grows downward into the oral cavity, forming the mesial nasal process, and ends in a bulbous enlargement called the globular process. The mesial processes then grow backward along the roof of the stomodæum, forming the nasal laminæ. The lower portion of the fronto-nasal process, which is originally situated between the olfactory pits, and includes the globular processes, gives rise to the intermaxillary region, the middle part of the lip and the lower part of the nasal septum and the portion of the fronto-nasal process between them. The bridge and point of the nose are formed by a pushing out of that part of the fronto-nasal process which lies immediately above. So far we have been following the development of the internal rims of the olfactory pits. The external rims grow also, but less rapidly, and project downward as the lateral nasal processes. From them are formed the alæ of the nose. They begin by curling around the lower part of the nasal pits, but soon meet and coalesce with the maxillary processes of the mandibular arch, which you will remember I described to you as growing around each side of the roof of the primitive mouth. The lateral nasal processes and the maxillary processes eventually join in front with the intermaxillary process, and the union of all these makes the upper boundary of the mouth and shuts it off from the anterior nares. Behind this anterior bridge the nose continues to communicate freely with the mouth. Finally the palatine processes grow like two shelves from the inside border of each of the maxillary processes. These by their union with each other in the middle line and with the nasal septum complete the division of the nose and mouth. The median union of the palate begins in front by the eighth week and is completed by the thirteenth week of intra-uterine life. From what I have said you can easily picture how an arrest of this process would result in several kinds of deformity. If the maxillary process on one or both sides fails to unite with the intermaxillary, a cleft will remain open in the contour of er lip on one or both sides of the intermaxillary bone, and hence we ve single or double harelip as the case may be. If the cleft extends le distance from mouth to nostril it is called complete, but if the is not reached by the opening it is called partial harelip. If there lure of the palatine processes to join, one or both nostrils will open roof of the mouth as well as into the pharynx, and we shall have the nation known as cleft palate. This may be a huge chasm running le length of the roof of the mouth, or may be only a small opening, ing but a bifurcation of the tip of the uvula may be left to show normal process of development has not gone on to completion. An ing and as yet unpublished observation on the persistence of an early on of development in the lip has been made to me by Professor C. ot, of the Harvard Medical School. If you examine the mouths of of men, you will be struck with the fact that in some of them the ntour of the upper lip appears broken by two rounded masses, each the size of a pea, situated side by side nearly in the middle line. are the remains of the two globular processes which have failed to terated in the formation of the intermaxillary region.

ides their unsightly appearance, which always causes the mother concern, these malformations may so interfere with the infant's the breast as to render sucking impossible and make it necessary the infant with a spoon.

ave here to show you a typical case (Case 162) of double harelip uncomplicated by late.

u will notice the large size of the intermaxillary bone, which protrudes considerably the margin of the lips and is somewhat twisted upon itself. This alteration of the of the intermaxillary bone may cause the teeth that grow from it to appear in very l places, as, for instance, protruding from the nostril.

CASE 162.



Double harelip.

r. J. C. Warren, who has examined the case, will now tell you what deas are as to the proper time for and the method of operating on ip:

The operation for the cure of the deformity of harelip consists in removing the edges of the cleft with the knife or sharp scissors, and in bringing the portions of the lip together by sutures.

"The cut may be made so that the lower edge of the wound will project slightly, so as to avoid an indentation of the border of the lip when cicatrization has taken place. This may be accomplished by making a slightly curved or V-shaped cut in each margin of the cleft. When there is double harelip, the portions of the lip adherent to the intermaxillary bone should be refreshed, leaving a V-shaped flap hanging from the septum of the nose. The wound when brought together then forms a Y.

"The sutures which are usually applied produce almost invariably unsightly scars, owing to the traction which is exerted when the child cries. I have therefore devised a plan by means of which external scars are avoided. This consists in passing a fine wire through the cleft at the margin of the alæ of the nose and forcing the parts into apposition by a perforated shot, which is then clamped to the wire. In the case of single harelip the wire passes through the ala of the side affected and the septum. One of the shots is therefore concealed in the nostril of the other side. My other sutures are of silk, and are so taken that the knots are tied in the mouth and the rest of the suture is buried in the deeper portions of the lip. Three such sutures are usually sufficient to hold the lip firmly. A few very fine sutures such as are used for intestinal sutures may be applied on the exposed surface to make the coaptation of the edges of the wound complete. A band of crêpe lisse fastened to the cheeks by collodion removes the strain sufficiently to enable healing to take place promptly. The wire should remain in place for ten days, but the fine sutures should be removed in two or three days and the remaining sutures at the end of a week.

"These operations should be performed during the early weeks of life, as the growth of the facial muscles is not then sufficient to interfere with the healing of the wound."

The method of feeding these cases is important. Various devices have been used to promote the power of sucking, which is so much interfered with by the connection between the nasal and buccal cavities. Rubber nipples of peculiar shapes have been used, with the idea of artificially closing the opening in the hard palate while the infant is being fed. I have always preferred, however, to have the infant fed by the spoon, and not to have it suck at all until after it has been operated upon and the wound entirely healed. In this way we avoid the irritation upon the floor of the nasal cavity which would be caused by the introduction of rubber nipples or any other apparatus. The infants, as a rule, have no trouble whatever in swallowing milk introduced into their mouths by means of a spoon.

The method of feeding premature infants by means of Dr. Breck's tube (Fig. 42, page 313) is also a rational way of feeding cases of harelip, provided that the infant does not insist on sucking.

CLEFT PALATE.—In speaking of harelip I have described most of the conditions occurring in cleft palate. The difficulty of feeding, if the cleft s the hard as well as the soft palate, is very great, and must be met manner just described. The difficulty in articulation and the ant sound of the voice are reasons which lead the parents to demand eatment. We should wait a longer time before operating than in harelip, as it is seldom wise to operate upon this deformity before d is three years old.

operation for cleft of the soft palate is called staphylorrhaphy, and rmed in this way. When the child has been put fully under uence of an anæsthetic, and the mouth held wide open with a gag, geon seizes the tip of the uvula with his forceps, and by the aid of a lunt-pointed bistory rapidly pares off a thin strip from the tip of the o the angle of the cleft. Then, changing his forceps, he takes a paring from the opposite side, carrying the knife from the top of the the tip of the uvula. The fresh edges are then brought into appoy a series of fine wire sutures, which are twisted tight and cut off. ator and tensor palati muscles, together with the palato-pharyngeus, cut, in order to lessen the tension on the flap. This is accomplished ing a thin-bladed knife completely through the soft palate close to r side of each hamular process: the handle is then raised a little knife withdrawn with its cutting edge downward. The anterior need be only slightly longer than the width of the blade.

operation for closure of a cleft in the hard palate, called uranoplasty, more difficult, and, owing to the great success which has lately been by fitting artificial palates, is now passing into disrepute. For efts uranoplasty is almost hopeless, but narrow ones may be successsed by its aid. It consists in marking out two side flaps parallel to t on the roof of the mouth. These are dissected up with as much um as possible for a distance a little in excess of the length of the The median edges of these flaps are refreshed and brought together w of sutures, just as was done in operating upon cleft of the soft The final step of dividing the palatal muscles is the same in each. y surgeons the lines of suture are in all these cases protected by a plate made to fit neatly into the roof of the mouth.

larger the opening in the palate the more successful will be the treaty apparatus in comparison with that by the knife, for in the large s there is so little opportunity for refreshing the edges of the openbringing them together that the operation is very apt to be unsuc- In using the apparatus, on the contrary, the larger the opening the the ease with which the artificial palate can be adapted. The artialate has also a uvula edge to it, and has in many cases proved ly successful when applied by the hands of an expert.

me here emphasize the fact that after any operation upon the mouth infant the after-care, and especially the feeding, are of the utmost nce. The infant must be watched night and day to see that it does its fingers to its mouth and thus interfere with the stitches. Of course every time it cries the strain is greatly increased upon the stitches. We must, therefore, impress upon the nurse the importance of continually amusing the infant.

TONGUE-TIE.—In quite a number of cases the frænum of the tongue is abnormally short at birth. In extreme cases the tip of the tongue is so closely bound to the lower jaw that it cannot be protruded beyond the line of the gum or touched to the roof of the mouth. The mother usually notices that the infant does not nurse readily, and brings it to the physician to discover the cause. In most cases on passing the finger into its mouth the infant is found to suck fairly well; but there can be no doubt that this condition, which is called tongue-tie, interferes somewhat with the process of sucking.

The treatment is to cut the frænum. This operation should be followed by no hemorrhage and requires no dressing. Having the child's head held in a fairly good light by an assistant, and guarding the lower part of the tongue with the perforated flange of a director, a small cut is made in the tense frænum with a pair of blunt-pointed scissors. By making the cut close to the gum there is no danger of wounding the ranine artery. The cut is prolonged as far as is necessary by tearing with the finger-nail.

Children who have not learned to talk at the usual time in the second and third years are frequently brought to me with the statement that they are tongue-tied, and the parents wish me to treat this condition. Large numbers of children are brought to the physician under this supposition, but in very few instances are they tongue-tied. These children belong to a class which I shall describe when speaking to you of retarded speech (Lecture XXXVI., page 740). I shall merely say at present that the condition is a central one of the brain, and not a local one in the mouth, and that if children hear well and are bright and mentally well developed, even though they do not speak at the third, fourth, or even fifth year, as a rule they learn to speak later.

RANULA.—Beneath the tongue we sometimes find the mucous membrane bulging out as a bluish, translucent tumor which is soft, painless, and semi-fluctuating. This condition is called *ranula*, and is a retention cyst caused by the blocking of a mucous duct. When opened, a small amount of glairy fluid escapes, but the collapse of the walls of the cyst brings the edges of the cut together and they quickly adhere. The fluid will soon re-collect; therefore the only sure way of dealing with these cysts is to pinch up their anterior wall with fine forceps, and with the scissors remove so much of it as to leave no opportunity for the edges to adhere. A gentle application of nitrate of silver to the edges and interior of the sac after the cut has been made with the scissors materially helps to promote the cure. It is not common in new-born children, but it occurs often enough to deserve mention.

EARS.—A deformity which is quite frequent at birth, and which increases as the infant approaches childhood, is the protrusion of the ears. The ear, esides at times being placed in an irregular position on the head, has, in the ases to which I am now referring, a tendency to stand out from the head arther than is considered normal. This position of the ear usually annoys mother very much, and you will frequently be consulted as to the means y which the deformity may be rectified.

In a large number of cases the persistent application of pressure by neans of various devices, one of which is a fenestrated cap, will cause the ars to be flattened against the side of the head. In intractable cases an peration will have to be performed, but it is very simple and does not leave n unsightly scar. Dr. Warren's method for operating for this deformity s illustrated by one of my cases (Case 163), a boy eight years old, in which he operation resulted in a marked improvement in his appearance.

Dr. Warren dissects off a flap from the back of the ear that is shaped very much like the wing of a butterfly. A similar flap is taken from the side of the head just back of the ear. The two raw surfaces are then brought together, and the edges of the wound united with fine sutures. Dr. Warren tells me that the amount of tissue removed must be considerably larger than would seem at first sight to be necessary, because if the ear is not united to the head by a band of considerable thickness the subsequent stretching of the cicatrix allows of a return of the deformity.

OPHTHALMIA NEONATORUM.—This disease has been divided into two forms, the catarrhal and the purulent.

Catarrhal Ophthalmia.—The catarrhal form may be caused by any slight irritation of the eyes of the infant. It runs a very mild course, the inflammation attacking chiefly the palpebral conjunctiva. Often the only symptoms noticed are a slight photophobia and a collection of the secretion in the angles of the lids and upon their borders. Its whole course is mild, and often it is all over in a few days.

Purulent Ophthalmia.—Although a considerable number of causes for urulent ophthalmia in the new-born have been given, such as trauma, exosure to light and cold, and others, certainly ninety-five per cent. of all ases are caused by infectious material from the genito-urinary tract of the mother, and in most instances it is by gonorrhœal pus. If infection takes place during the birth of the child, the symptoms usually begin on the third day; but, as contaminated linen and fingers may carry the infectious material to the infant's eyes at a later period, the symptoms may be delayed indefinitely. The disease begins as a redness of the conjunctiva, with a slight discharge from the corner of the eye. This is succeeded with startling rapidity by intense inflammation of the lids. In twenty-four hours the upper lid may become so much swollen as to overhang the cheek and render opening the eye impossible. On separating the lids, a little greenish pus, which may even be tinged with blood, wells up between them. At first the cornea is unaffected, but if the pus accumulates under the œdematous lids it soon shows signs of ulceration. In the second twenty-four hours the ulceration may perforate the cornea and evacuate the aqueous humor, thus bringing the iris into contact with the posterior surface of the cornea. The inflammation may extend around the eye and well over the forehead and malar prominence, but it does not last in the latter region very long.

All the symptoms disappear slowly, and recovery takes place, except in those cases where from ulceration the cornea has been permanently injured.

In treating this disease we must be very prompt and energetic. It often may be averted by what is known as Credé's method. This consists in dropping one or two minims of a two per cent. solution of nitrate of silver into each eye of the new-born infant. Although this has been known to cause even a considerable amount of irritation, yet it undoubtedly exerts a powerful influence in warding off this dangerous disease.

After the disease has once begun, two indications must be kept in mind: (1) to reduce the inflammation, and (2) to prevent the pus from accumulating behind the tightly-closed lids. By far the best way of applying cold to the eye is by compresses of thin, soft pieces of linen cut into small squares. Not more than two thicknesses are to be used at once. These compresses are to be cooled by laying them on a piece of ice or floating them in ice-water. They must be constantly changed. To remove the pus, a gentle irrigation, such as can be easily obtained by using a medicine dropper, is sufficient.

Remember that this secretion is highly contagious, not only for the infant's other eye, but for yourself. Therefore you must avoid all spattering, and should cover the infant's well eye before you begin the irrigation.

You should first turn the child's head a little to the diseased side, and with the fingers of the left hand gently separate the lids as far as possible. Then, holding the dropper with the right hand, irrigate between the lids, directing the stream *from* the nose.

This should be done at least every half-hour, day and night, until the swelling has so far subsided as to preclude the danger of any secretion being retained.

For irrigation many solutions have been advocated. The most simple, and perhaps the best, is a saturated solution of boracic acid, or one of bichloride of mercury in the strength of 0.05 gramme (1 grain) to 480 c.c. (1 pint) of distilled water. In the later stages of the disease, where all the tissues are relaxed, a solution of nitrate of silver, 0.5 gramme (10 grains) to 30 c.c. (1 ounce) of distilled water, may cautiously be used once a day.

It is not within the scope of these lectures to describe in detail scarification of the cornea or other measures which may become necessary to save extensive sloughing from strangulation.

THE NECK. Hæmatoma of the Sterno-Cleido-Mastoid Muscle.—During the birth of the child, either from the violence of the expulsive efforts of the uterus, or, as more frequently happens, from the pressure of the forceps in head presentations, or from too vigorous traction upon the feet in breech presentations, or for no assignable reason, the sterno-mastoid muscle may be partially ruptured in its sheath and a hæmatoma form be-

een the torn ends. This tumor may be either in the sternal or in the cla-
ular portion of the muscle, or may be just above the junction of the two.
r a short time it is soft and tender, but gradually it loses its sensitive-
ss and becomes converted into fibrous tissue, which then tends to contract.
may appear as a small tumor, but in infants with fat necks it may not
noticeable at first. As turning the head towards the affected side lessens
tension upon the swelling, the infant will rigidly hold its head in that
sition. It is in this way that cases of infantile torticollis are thought by
st writers to arise.

TREATMENT.—After the painful stage has passed, the treatment is by
ntle massage and manipulations addressed to stretching the shortened
uscle. If these methods fail, the child must be placed in the hands of an
thopædic surgeon for more extended treatment, either by apparatus or by
vision of the tendinous attachments of the sterno-mastoid muscle.

BRANCHIAL FISTULÆ.—At an early period of development the neck
the fœtus has along its sides a series of four branchial clefts, which
mmunicate freely with the œsophagus and represent the gills of aquatic
imals. The upper one of these forms the tympanum and the Eustachian
ube, the rest are normally obliterated. Sometimes we find traces of these
ranchial clefts in the form of small fistulous tracts which admit a probe
short distance and end blindly. Their most frequent seat is just above
he sterno-clavicular articulation, but they may be found anywhere along
the anterior border of the sterno-mastoid muscle.

If they do not cause any inconvenience it is better to let them alone, as they often prove very intractable to treatment. If they are annoying from causing a slight mucous discharge, we can try to eradicate them with the galvano-cautery, or by passing a probe into the wound and dissecting from around it the lining of the sinus.

Sometimes the entrance of these fistulæ becomes stopped, so that they dilate and form large cysts containing mucus, blood, and atheromatous detritus. These form at times large and unsightly bunches, which require surgical treatment. Often the operation of obliterating them is not an easy one, for they are apt to have deep and complicated attachments.

LECTURE XIX.

THE TRUNK.

MASTITIS.—In certain infants during the early days of life we find swelling and hardness of one of the mammæ. This condition appears to b an inflammatory one, and is abnormal. In connection with the swolle condition of the mamma, a secretion is found to come from the nipple whic corresponds closely to milk, and which has been called "witches' milk."

A number of analyses have been made of this fluid, and here are som (Analyses 57 and 58) which represent the composition of it very well. O course only a few drops of the fluid can be expressed from the mamma one time.

ANALYSIS 57. (Schlossberger.)

Fat	0.82
Casein, sugar, and extractives	2.88
Ash	0.05
Total solids	3.70
Water	96.30
	100.00

ANALYSIS 58. (V. Gesner.)

Fat	1.45
Casein	0.55
Proteids	0.49
Sugar	0.95
Ash	0.82
Total solids	4.26
Water	95.74
	100.00

This condition occurs in boys as well as in girls, and, as far as I know has no especial significance. With ordinary antiseptic precautions th inflammation usually subsides in a few days, leaving the affected breast th same size as the other.

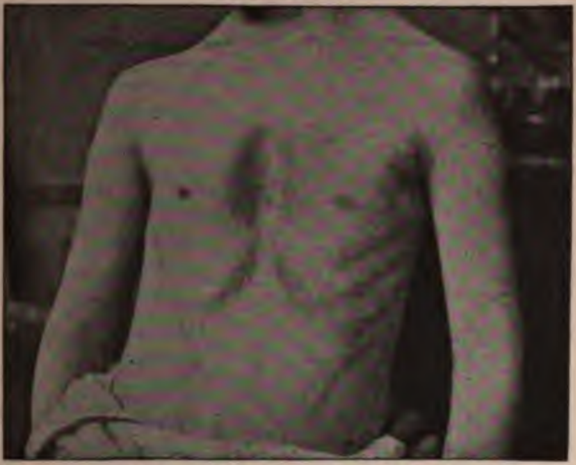
I have here a female infant (Case 164) who represents this condition o the mamma.

She is one week old, and the swelling of the mamma was noticed on the fourth day o her life. You see a little fluid looking like diluted milk can be expressed from the mamm The treatment of the case will simply be to keep it thoroughly clean by washing it wit sterilized water, carefully drying it, and applying a compress with a little simple ointme on it.

DEPRESSED STERNUM.—There are a great many congenital mal formations which may occur in different parts of the thorax. I have her show you a boy (Case 165) who was born with a depression of the lower t of the sternum.

He is now six years old, and has this rounded depression, about 4 cm. (1½ inches) in meter, beginning at the third costal cartilage and extending to the ensiform cartilage. is perfectly healthy. The cardiac dulness extends to 2.5 cm. (1 inch) to the left of the mary line, and its impulse is in the fourth left interspace. The spinal column is straight. epiphyses of the wrists are slightly enlarged, but there is no other evidence of rhachitis. en he was two months old he had a severe attack of pertussis, which lasted for over two ths. At five years of age he had a severe attack of bronchitis.

Case 165.



Congenital depression of sternum. Male, 6 years old.

You see, therefore, that he has been subjected to influences which would tend to increase alformation of this kind.

Although this depression of the sternum was present at birth, and has since increased epth and in circumference, it now seems to have ceased to enlarge. The circumference is head and that of his chest measure 50.5 cm. (20 inches).

The heart seems to be somewhat displaced upward and to the left, but is apparently ffected by its abnormal position, and the boy's circulatory system will probably not be red.

I have ordered for treatment light gymnastic exercises to broaden the chest and to ngthen the thoracic muscles.

Such a malformation as this sometimes results as one of the changes subsequent to 's disease. More often the sternum protrudes, but occasionally recession takes place, ely resembling the condition in this case.

PROMINENT STERNUM.—A prominence of the sternum, called *geon-breast*, occurs more often than the depression. It may happen thout an assignable cause, or it may be due to rhachitis, and may also sult from some spinal distortion, such as that of Pott's disease, or lateral rvature. In the latter case the sternum is often tilted to one side.

SPINA BIFIDA.—I shall next show you a series of that class of alformation called *spina bifida*. Spina bifida consists of a lack of closure f the laminæ of the vertebræ. This condition is normal at a certain period of intra-uterine life, but when persisting to a later period, and when occurring at birth, becomes abnormal from a developmental point of view and represents a distinct malformation. As the fusion of the laminæ at the base of the spinous process takes place in sequence from above downward, the most frequent seat for spina bifida is in the lumbar and lumbo-sacral regions. There it appears as a tumor situated exactly in the middle line, covered sometimes with healthy skin, but as frequently roofed over by nothing but a thin adherent transparent membrane. Rarely the tumor is solid, containing nothing but an empty sac that has been walled off from its connections with the spinal canal. It is then called *spina bifida occulta.* In true spina bifida the tumor is filled with cerebro-spinal fluid, which can be seen to increase in amount as the child cries, and can, by pressure upon the sac, be forced back, in this case often giving rise to cerebral symptoms. According to the contents of the tumor, spina bifida has been divided into several varieties.

1. SPINAL MENINGOCELE.—When there is a protrusion of the membranes filled with fluid the tumor is called a *spinal meningocele.*

2. MENINGO-MYELOCELE.—The most common form is where the spinal cord, as well as the membranes, is found in the tumor. It then becomes a *meningo-myelocele.*

The position of the cord in these tumors is a very variable one. It may run directly through the tumor, and even be suspended by a kind of mesentery, or, as is usually the case, it may be spread out like a fan over the surface; in any instance it is rudimentary in character.

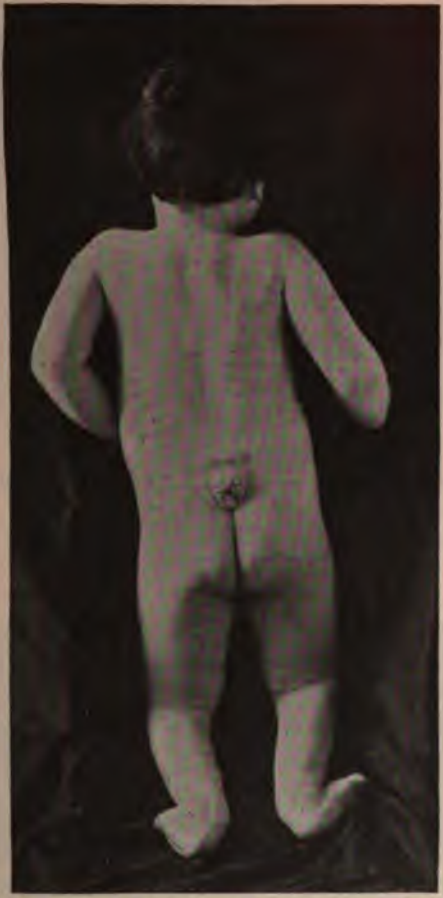
3. SYRINGO-MYELOCELE.—*Syringo-myelocele* is a rare form, in which the sac is formed of meninges and cord, the central canal of the cord being dilated to make the cavity of the tumor.

Spina bifida occurs usually in poorly-developed infants, and in a large majority of cases it is associated with other malformations, such as congenital hydrocephalus, harelip, club-foot, paralysis of the lower extremities, and in severe cases there may be incontinence of urine and of fæces. Sometimes the infant is well formed and healthy in every other respect.

If left to itself, the course of spina bifida is in one of two directions: (1) spontaneous closure and obliteration of the sac; (2) ulceration of the sac, followed by convulsions and death. In the first case, which is very rare, the sac shrivels up and thus effects a spontaneous cure. I happen to have here one of the first class of cases to show you (Case 166).

This boy, now four and one-half years old, shows an elevated cicatrix in the lumbar region, which suggests the former existence of a spina bifida. The case was of such interest that it was reported by Dr. Lovett in the *Boston Medical and Surgical Journal* as a form of spontaneous recovery from spina bifida. The case was seen by him when it was eighteen months old, and so far as could be learned there had been a large tumor present at birth similar to those which I shall presently show you. The sac burst in this case, and, contrary to the general result, the child did not die, but was left with paralysis of the legs, which makes it stand in this curious and abnormal position. He also suffers from incontinence of urine and of fæces. The child has never walked, and it seems probable that the present ty is caused by the fact that the nerves were spread on the walls of the sac, as is many cases, and that they were incorporated in the cicatrix.

Case 166.



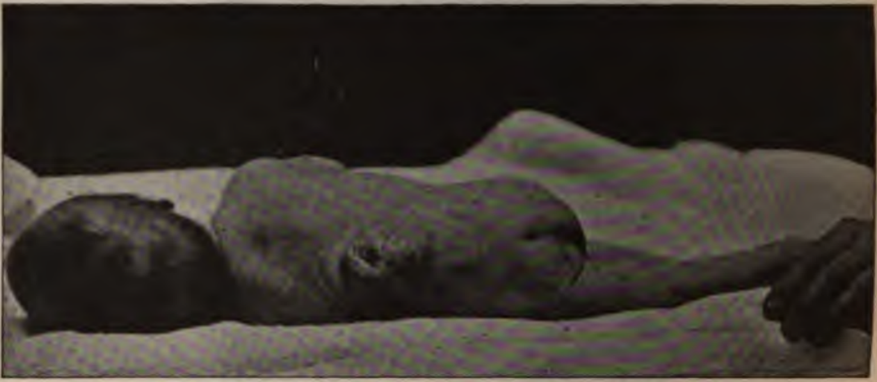
Spina bifida. Spontaneous cure. Male, 4½ years old.

A result like this is, however, very exceptional. The rule is, either that e is an ulceration of the sac, followed by a large loss of cerebro-spinal l, convulsions, and death, or that the opening in the spine being very ll the loss of fluid is constant, and the result is the same. In some nces there is an infection of pyogenic organisms through the walls of sac, which causes a septic meningitis in the cord and finally in the brain. a case has been reported and beautifully illustrated by Dr. Holt, of York, showing the presence of the bacteria and a resulting purulent rocephalus.

Here is a picture of another case (Case 167) of spina bifida which will trate the ordinary course of the affection.

It shows a large spina bifida in the dorso-lumbar region. The membrane covering the r was so translucent that the spinal cord could be plainly seen through it. At birth there was a small tumor. It filled with fluid at the end of twelve hours, and at the end of forty-eight hours it looked as it does in this picture. The top of the tumor suppurated, the fluid began to leak away, and the child died within ten days.

Case 167.

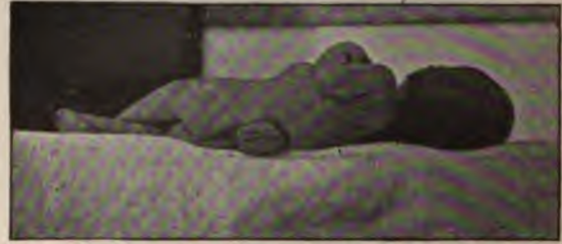


Spina bifida of dorso-lumbar region. Infant 48 hours old. Died when 10 days old.

This is the course pursued by the disease in the majority of cases which are not operated upon.

The next case (Case 168) is an illustration of a spina bifida in the lower dorsal region.

Case 168.



Spina bifida in lower dorsal region. Infant 5 days old. Died when 7 days old.

The sac was not so tense as in the case (Case 167) of which I have just spoken, and it was possible by feeling deeply with the fingers to find the opening in the spinal column. This opening was about 7.5 cm. (3 inches) long and 3.8 cm. (1½ inches) wide. The tumor was not covered with skin, as in the case previously mentioned, but with a thin, translucent membrane.

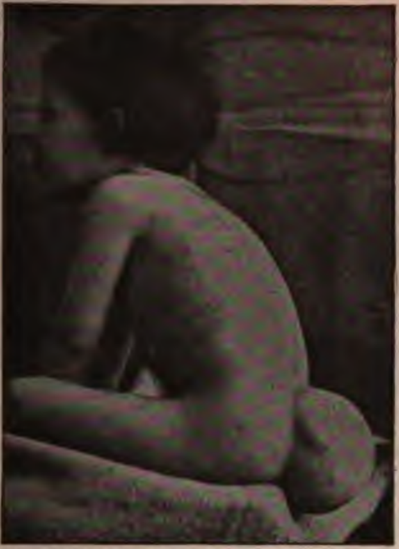
The infant was seen by Dr. Lovett in consultation twelve hours after birth, and an operation was deferred for a few days to see if any favorable change would occur. The operation was undertaken on the fifth day, as the sac showed signs of ulcerating and breaking. The sac was excised without apparently injuring the nerves, and the wound was closed by a plastic operation. The infant died in convulsions within forty-eight hours of the operation.

The next patient I have to show you (Case 169) is another case of spina bifida.

The boy is now five years old, and has had this large tumor since birth. It is situated over the lumbar region of the spinal cord, and is in the median line. The fluid has been withdrawn several times for purposes of examination, and when the sac is lax an opening

5 cm. (2 inches) long can be felt in the spinal canal. It is elliptical in shape. From the fact that the child suffers from incontinence of urine and has a certain degree of paralysis of the legs, it is fair to infer that the nerve-supply of the legs and pelvis is incorporated in the tumor.

Case 169.



Spina bifida of lumbar region. Male, 5 years old.

This case has been tapped and treated with an injection of Morton's fluid, but this treatment was entirely unsuccessful, and although the sac has been aspirated several times the fluid has always returned. There is little hope of the boy's being relieved by an operation ultimately, and he will probably continue to be a cripple for life.

Treatment.—Various methods for treating spina bifida have been proposed, and some of them warmly advocated. Repeated aspiration is one of the most simple, but its results have not been satisfactory. Ligature of the neck of the sac, if the sac is small, or the application of a clamp, has cured a few cases. Electricity has been recommended for this affection, as for about everything else in the field of medicine.

The two methods that are in the best repute are the injection of Morton's fluid and the plastic operation. Morton's fluid is a solution composed as is shown in this prescription (Prescription 46):

Prescription 46.

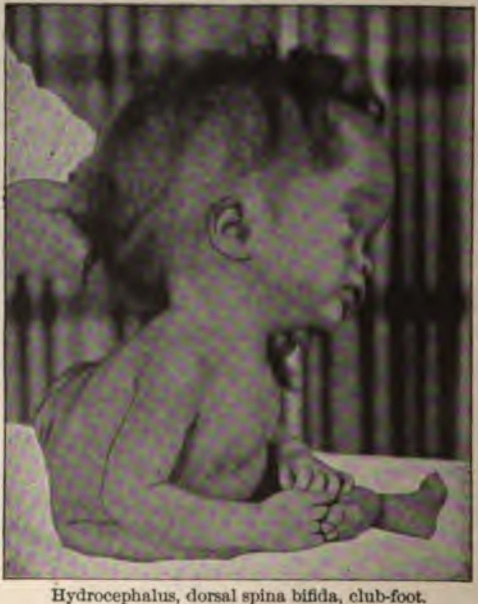
(*Morton's Fluid.*)

Metric.	Gramma.		*Apothecary.*	
℞ Iodi	0	60	℞ Iodi	gr. x;
Potassii iodidi	1	80	Potassii iodidi	gr. xxx;
Glycerini	30	00	Glycerini	℥i.
M.			M.	

From 1 to 4 c.c. (15 minims to 1 drachm) are used at each injection, which may be repeated several times at intervals of a fortnight. The reports of the Clinical Society of England show that more cures and fewer deaths have been reported following the use of this solution than from any other method.

Another method is the plastic operation. This is performed as follows. The tumor is opened, the nerves are dissected carefully from the walls of the sac and are returned to the spinal canal: the sac is then sewed up, and, if possible, used as a plug for the opening. It has been recommended that the laminæ of the vertebræ on both sides of the cleft should be broken and turned in. Finally, after the excision of all the thin covering, the fresh edges of the sound skin are united. To accomplish the closure of the wound, and yet to avoid dangerous tension on the stitches, it may, in the case of large tumors, be necessary to dissect up two lateral flaps of skin from the loins and slide them inward to join in the median line. The majority of cases which have been so operated upon have died within a week, but the few successes that have been attained lead us to hope that with a more perfect technique the results of the operative treatment of spina bifida may be such as to warrant our advising it in any case where the tumor threatens to rupture and where the child is otherwise fairly developed. You must clearly understand, however, that the operation will in most cases not help the paralysis or incontinence, and may very possibly increase instead of diminish a hydrocephalus, if this latter condition exists as a complication.

CASE 170.



Hydrocephalus, dorsal spina bifida, club-foot.

This photograph illustrates the condition of hydrocephalus accompanying spina bifida, which I have just described. The infant (Case 170), a case of Dr. Osler's, represents a combination of spina bifida, hydrocephalus, and paralytic deformity of the lower extremities.

ILEBITIS AND ARTERITIS UMBILICALIS.—The cause of f these conditions is a septic infection of the umbilical stump. It is ered by most pathologists to begin as an inflammation of the peri- ar cellular tissue, and only secondarily to invade the walls of the . The region around the umbilicus is red and hot, and we may be y gentle pressure to squeeze a few drops of pus from the stump of rd. It is a very dangerous affection, as septic emboli readily pass he infected vessels into the general circulation and set up metastatic mation in the thoracic as well as in the abdominal organs.

EATMENT.—The treatment is to sustain the infant's vitality by stimu- and thoroughly to disinfect the umbilicus with solutions of bichloride rcury or carbolic acid, followed by the application of boracic acid or rm powder. A flaxseed poultice is often of service, and some authors nend placing the infant upon its abdomen in order that gravity may draining away the pus.

NGENITAL UMBILICAL HERNIA INTO THE CORD.—Dr. rd Marsh, in the Report of St. Bartholomew's Hospital for 1874, ttention to the "familiar anatomical fact that from about the sixth to elfth week of intra-uterine life the cæcum and neighboring portions ileum are contained in the part of the umbilical cord which is next body of the embryo, and that they should subsequently withdraw e cavity of the abdomen. In some cases, however, this recession fails e place, and the intestine remains, even up to the time of birth, still in the beginning of the cord, which is dilated in the form of a ranous sac." Not only may portions of the intestine be thus left e of the abdominal wall, but, as in a case recently operated upon by arren, the liver may be found lying in a hernial sac made from the base of the umbilical cord.

infant (Case 171) was sent to Dr. Warren at the Massachusetts General Hospital ours after its birth. At the umbilicus was seen the cord, which was greatly dis- at its point of insertion into the abdomen, forming a tumor 6.5 cm. (2⅝ inches) in r. The coverings of the cord were inserted into a raised rim of skin, and were so that the contents of the hernia could not be determined.

hen the infant was one day old, Dr. Warren enlarged the umbilical ring somewhat, d the liver from the myxomatous tissue of the cord, which was in some places dherent to it, and returned the mass within the abdomen. The wound was tightly with strong silk sutures. There was considerable shock following the operation, re were no symptoms of peritonitis. In two weeks the wound had healed, and the recovered.

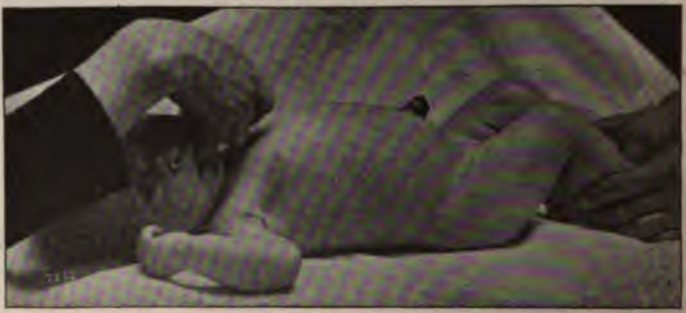
UNGUS OF THE UMBILICUS.—The umbilical cord, after being red at birth, falls off by the seventh or eighth day, leaving a clean, catrix. After the separation of the cord we sometimes-find a red pro- n, with a moist surface, that may even have a short central canal. This erally due to an imperfect disintegration of the cord. It may bleed readily if touched, and may give rise to a discharge so irritating that the skin for some distance around the umbilicus becomes eczematous. This condition is called *fungus* or *polypus of the umbilicus.*

The treatment is very simple. The larger ones are best removed by ligation; the smaller ones can be destroyed by the application of nitrate of silver or the actual cautery.

MECKEL'S DIVERTICULUM.—A condition which may at first simulate umbilical polypus, and of which umbilical polypus may be a symptom, is the persistence of a *Meckel's diverticulum.* This consists in the persistence of a piece of intestine, usually patent, connecting the small intestine with the umbilicus. It represents a vitelline duct that failed to atrophy when the placental circulation became established, and betrays its presence by an escape of fæces from the umbilicus. It is a rare malformation, but one which you should recognize at once.

I have here to show you the picture of a case (Case 172) that came to the Infants' Hospital last winter during the service of Dr. Lovett.

CASE 172.



Persistence of Meckel's diverticulum. Infant 3 days old.

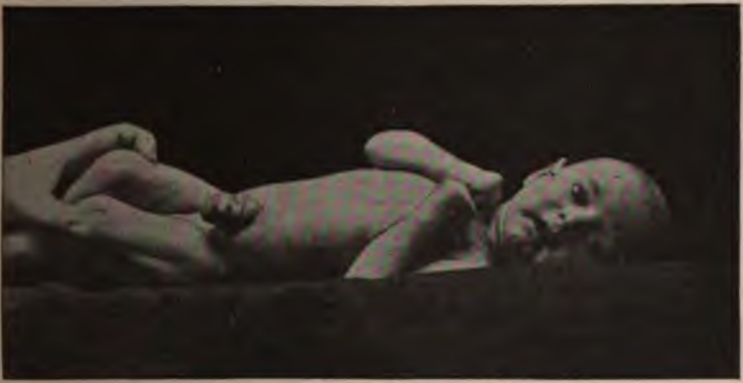
The infant at entrance was three days old and was very well nourished. You will notice the protrusion at the umbilicus, on the top of which is a bright red granulating surface, appearing black in the picture. There was a considerable fæcal discharge from the polypus, and the skin of the abdomen was much irritated in its vicinity. A medium-sized probe could with ease be passed 6.5 cm. (2½ inches). Laparotomy was performed by Dr. Lovett. The diverticulum, which was found to arise from the middle of the ileum, was resected and the intestinal wound sewed up. The polypus was not disturbed at the first operation, its blind stump being sewed off even with the inside surface of the abdominal wall. The line of incision, which was about 2.5 cm. (1 inch) to the left of the polypus, and 8.7 cm. (3½ inches) long, healed by first intention. A week afterwards the polypus was removed by two applications of the actual cautery. The infant was allowed to return home, but came back ten days later with a double pneumonia, from which it died.

UMBILICAL HERNIA.—The ordinary umbilical hernia, which is simply a protrusion of a knuckle of the intestines through the unclosed abdominal opening left by the separation of the cord, is of very common occurrence. The lighter grades tend to recover spontaneously, and it is not advisable to operate upon them, or in fact on any umbilical hernia, until it has proved to be absolutely intractable, for it is an operation accompanied considerable danger to the life of the infant. The lighter grades of bilical hernia are usually easily reduced, but there is often great trouble keeping them so. Various devices are employed for this purpose, but st of them are very unsatisfactory. At the Children's Hospital we are the habit of proceeding in the following manner.

Having gently reduced the hernia, the skin of the abdomen is so pushed between the fingers that it makes a vertical fold, at the bottom of which the umbilicus. The hole should be deep enough to lay one's finger in The tension is kept up by applying a wide strip of adhesive plaster nsversely across the abdomen. This makes a pad of flesh, which closes umbilical opening and retains the intestine in place. The cure is a slow , and the treatment must be continued for many months in severe cases, thout once allowing the hernia to come out. The milder cases are also ed by exercises which tend to develop the abdominal muscles. This can very simply effected by having the child lie on the floor, and, while the t are held down, making him rise to a sitting position with the back held aight. This is accomplished by the rectus muscles of the abdomen, and the opening is a transverse one it tends to close it.

This case which I have here to show you (Case 173) is an infant five months old. The nia, as you see, is very large, and has caused an eversion of the whole umbilical region. represents an extreme grade of the disease.

Case 173.



Umbilical hernia. Infant 5 months old.

Cases of incarcerated and even strangulated umbilical hernia have been ported, but are very rare. A few have been operated upon successfully. he danger from all such procedures is usually considered great, but there as been such an advance made in the modern methods of abdominal surgery at the operation is looked upon with increasing favor.

INGUINAL HERNIA.—The most common forms of *inguinal hernia* at occur in young children are (1) the congenital, (2) the funicular, and 3) the infantile or encysted. An ordinary acquired form such as is the rule n the adult may be met with, but it is not so common.

(1) Congenital Form.—The congenital form is that variety in which the knot of intestine has made its way along a still patent funicular process. If it reaches into the scrotum it will be found completely to envelop the testicle.

(2) Funicular Form.—In the funicular form, the tunica vaginalis having become shut off from the funicular process just above the testicle, the hernia comes down the patent process, but does not envelop the testicle as in the preceding variety.

(3) Infantile Form.—Compared with the two forms just mentioned, the infantile or encysted form of hernia is quite rare, nor can it be diagnosticated with certainty without an operation. In it the funicular process has closed above but not below, and the intestine encased in a pouch of peritoneum forces its way into the process and descends.

The diagnosis between direct and indirect hernia has little importance in childhood, as the inguinal canal is so short that the rings are practically at the same level. Most of the herniæ that you will meet in children are easily reducible, but you should remember that in attempting to get them back into the abdominal cavity you must use the greatest care, as nowhere can a little rough manipulation do more harm. If the hernia cannot be easily replaced, you must not think of leaving it where it is, simply because it gives rise to no alarming symptoms on the part of the child. No infant is safe with an irreducible hernia, and the sooner you put such a case in the hands of a surgeon the better. Strangulated and incarcerated herniæ occur at times as in adults, although they are rare. They demand the same treatment.

The condition with which you will most readily confound hernia is hydrocele. Both give rise to an elastic tumor in the inguinal region and in the scrotum, and in fact they resemble each other in many ways. Let me point out to you some of their differences.

Hydrocele is translucent by transmitted light; hernia is opaque. Hydrocele is always dull on percussion; hernia is usually resonant. If you can reduce them, hydrocele will go back slowly and noiselessly, hernia at the last quickly and with a gurgling sound. Hydrocele gives no impulse on coughing; hernia usually does. Lastly, in feeling for the inguinal ring in hernia you find it filled with the neck of the tumor; in hydrocele it is either empty or filled by a narrow stalk.

Treatment.—Although the treatment of inguinal hernia, whether by actual operation or by the application of the usual trusses, should be in surgical hands, yet one method of treating these herniæ is so simple and safe that every medical man should know about it; in fact, in our children's clinics here in Boston it is much used for all children under a year and a half. This method is the application of a worsted truss like this one.

The infant (Case 174) whom I am about to fit with this truss is eight months old. You see the bulging of the hernia here on the left side. Below it, feeling like another little sac, the testicle. We are, therefore, dealing with the funicular form. The mother tells that she noticed the hernia when the infant was two weeks old, and that it has grown adily larger.

On laying the infant on his back on the table, you see that after a minute of gentle is the hernia can be reduced, but it comes out again with a jerk when the child begins cry.

I shall now ask Dr. Dane, who has had much experience with these cases on the surgical e of the hospital, and who has given me much valuable advice on the surgical bearing all these cases which I have been describing to you, to reduce the hernia again and keep in place.

Dr. Dane, as you see, having reduced the hernia, and having the nurse prevent it m coming down by placing her finger over the inguinal ring, passes a skein of Germantown yarn under the infant's back and brings the left-hand end of it around its left e, with the strands separated so as to form a loop, till it rests over the nurse's finger. rough the loop he puts the right unseparated end of the skein, and carries it down the t groin, and up on to the back, where he finishes by tying it to the middle of the skein it crosses the hollow of the back just above the buttocks. As you see, he has, by thus reading the right closed end of the skein through the separated strands at the left end, ade a kind of soft slip-knot which lies directly over the inguinal ring, and, when the whole put on tightly, makes an excellent truss.

Having told the mother to buy some skeins like this one, and having shown her how adjust it herself, she can keep a clean truss, by washing them, on the child for a period f months, and if she is faithful in carrying out her part of the treatment the herniæ, hich do not depend upon an actual malformation of the ring, will probably be cured. f these herniæ are not cured within a year, the surgical treatment of the present time is by peration.

The next case (Case 175, facing page 430) which I have to show you came nder Dr. Lovett's care at the Infants' Hospital, and had to be operated upon:

This boy, who is now four years old, first came to the hospital two years ago. He en had a double inguinal hernia, both rings admitting the end of the index finger. He as fitted with worsted and pad trusses, but failed to return after the first few weeks. As u now see, the left ring has grown so much smaller that the hernia no longer descends. he right inguinal ring easily admits the middle finger, and when the hernia comes down is quite large. Below it you can feel the testicle. As the treatment with a truss has iled entirely, an operation will be advised.

In connection with this case I wish to speak of a complication that may xist with any hernia in male infants. If you will feel below the hernia of his child (Case 176), you will find no trace of the testicle, nor do you have ny better success after you have reduced the hernia. In this instance the esticle is not adherent to the bowel, and has not been pushed back with it, s is sometimes the case, but seems never to have left the abdominal cavity. I shall return to this subject again when speaking of the diseases of the esticle.

I must call your attention to a remarkable case that came into the hands of Dr. Monks, my colleague at the Boston City Hospital:

The child (Case 177) was two years old. Two months before he was seen by Dr. Monks the child's mother noticed a hard bunch in the right inguinal region. This became larger and more painful till, at this time, it extended the whole length of the inguinal canal and into the scrotum. The most prominent part was midway between the external ring and the testicle. It was very tender, about 2.5 cm. (1 inch) long, and quite hard.

There was no impulse on coughing. On aspiration there were found a few drops of pus, but on trying to find the cavity again with a director, nothing but inflammatory tissue was met. Under poultices the tenderness disappeared and the tumor was reduced somewhat in size.

On operation, two weeks later, the cause was found to be a hernia of the vermiform appendix, followed by an acute attack of appendicitis. The caput cæci and the base of the appendix were found inside the abdomen, and in a normal condition. An appendectomy was performed, and the child made a perfect recovery.

Femoral Hernia.—In femoral hernia the gut escapes from the pelvis under Poupart's ligament, and, making its way through the femoral canal, shows itself as a tumor directly under the saphenous opening. It can be diagnosticated at once from inguinal hernia by putting the finger on the spine of the pubes and noticing whether the origin of the tumor is to the outer or the inner side of that point. If outside, you are sure the hernia came through the femoral canal, no matter how far it may have extended up on to the abdomen. Femoral hernia is, however, extremely rare in young children, even in girls. In infancy the spine of the pubes, Poupart's ligament, and the anterior superior spine of the ilium are all much nearer together than in the adult. As a consequence, the femoral opening is so small and so well protected that it is usually impossible for the hernia to force its way through. Dr. Cushing, my colleague at the Children's Hospital, has reported a case (Case 178) of irreducible femoral hernia in which the sac contained a mass of omentum so matted together as to give a feeling that without special care might have been mistaken for that of lipoma. Such a condition must certainly be very rare. Dr. Cushing has described in his account of his operation upon this case a new incision that must prove very useful.

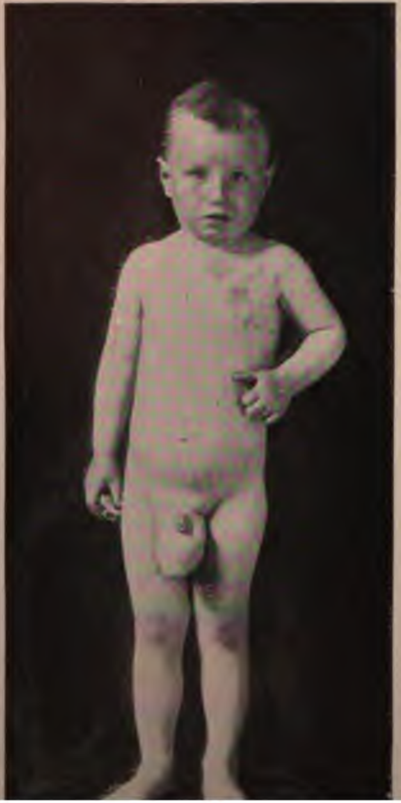
HYDROCELE.—I have already spoken of the general appearance of hydrocele in giving you rules for differentiating it from inguinal hernia, with which it is often associated.

Several anatomical varieties are met with in hydrocele, as in hernia. Thus, if the collection of fluid occupies a freely open funicular process, we have the *congenital variety*, and the fluid can easily be returned to the abdominal cavity by placing the child on its back and elevating the scrotum. This is true also of *funicular hydrocele*, where the fluid occupies an open funicular process, but is bounded below at the point where the tunica vaginalis has become walled off, leaving the testicle in a separate compartment underneath. Where the funicular process has become walled off from the abdomen, but is still in communication with the tunica vaginalis, there may be a collection of fluid, which is then known as an *infantile hydrocele;* in this form the fluid is irreducible. True hydrocele of the tunica vaginalis may be met with in children as well as in adults, but it is rare.

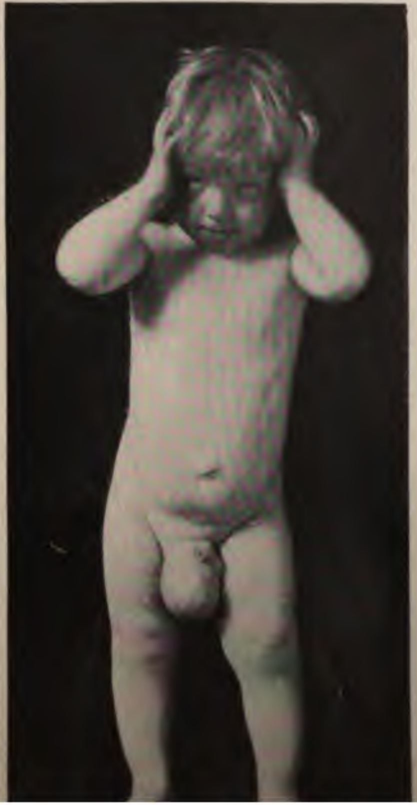
Encysted Hydrocele of the Cord.—There is another form of hydrocele which often escapes recognition, but perhaps still oftener is diagnosticated as hernia and treated with a truss. This is the encysted hydrocele of the cord.

If in the course of the spermatic cord a hard, rounded swelling appear,

CASE 175.



CASE 180.



of Dr. Lovett. A little below the inguinal ring on the right side was a small
he mother said that she had noticed the swelling for about a week, and the day
carried the infant for advice to a local physician. He had attempted to reduce
pposed was a hernia by gentle taxis. Failing in this, he gave the infant ether,
vas unsuccessful. The next morning, in company with an associate, he etherized
nd tried unsuccessfully for an hour to effect reduction.
fant was then brought to the hospital for operation. The hydrocele was aspirated,
he removal of a little over 2 c.c. (30 minims) of clear fluid all trace of the
rnia disappeared.
fant was brought back a week later, as the hydrocele had again accumulated. A
ration effected a cure.

ation this case in order to impress upon you how careful the
who is practising among children should be not to meddle with
ch should at once be placed under the care of a surgeon.
STED HYDROCELE OF THE CANAL OF NUCK.—Analogous to
of the cord in boys is an accumulation of fluid in the canal of
girls. The appearance of the swelling is the same in both cases,
eatment should be the same.
TMENT.—The treatment of all forms of irreducible hydrocele is
septic evacuation of the fluid with a fine canula and trocar, or by
ting needle. If this, after repeated trials, fails to effect a cure,
n of the sac is the only sure method, although the injection of a
tion of iodine is highly recommended by many authors. It is,
dangerous in children, as the occasional connection of the hydrocele
he abdomen is not to be forgotten.
ible forms of hydrocele are generally to be treated by a truss, in
manner as herniæ, to try to effect a closure of the neck of the
f this is successful they can then be treated in the ordinary
e outlook, however, is poor, and such treatment is generally un-
y.

I would here mention that cases of hernia, whether umbilical or inguinal, are especially difficult to manage if the infant has some such disease as pertussis. There seems to be some evidence that hernia is hereditary. Félizet reports eighty-five cases of hernia occurring in his practice, where, omitting all cases in which the father pursued some laborious trade, such as that of a blacksmith, he found that in 24.7 per cent. the parents had had similar herniæ. Malgaigne reports a percentage of 29 due to heredity in a series of three hundred and sixteen cases of hernia.

Infants are at times brought to our hospitals with a history of colic who, on examination, are found to have more or less incarceration of these herniæ. This should impress upon you the importance of making a systematic physical examination in every case of abdominal hernia, and of not taking it for granted that the symptoms are caused by indigestion.

TESTICLE.—The testicle should descend into the scrotum at about the eighth month of intra-uterine life. In certain cases it does not descend, and if the descent does not take place within the first few years of life its function is lost from its becoming atrophied. It is, therefore, important in those cases where the testicle descends and returns to the abdominal cavity to retain it in the scrotum by means of apparatus. Operation for this condition is not often successful. At times an undescended testicle is found in combination with an inguinal hernia. A case of this kind came under my care about two years ago.

A little boy (Case 181), four years old, was found to have an inguinal hernia. The testicle was also found at times to be absent on the side of the hernia. Sometimes the hernia would descend and the testicle remain in the abdominal cavity, and again the testicle would come down with the hernia. It was exceedingly difficult to maintain the testicle in the scrotum, even when it was found to be there, as it would slip back with the greatest facility.

I placed the case under Dr. Lovett's care, and he finally succeeded in seeing the boy at a time when both the testicle and the hernia were down, and in reducing the hernia while the testicle was kept in the scrotum. A carefully adapted truss now prevents the testicle from returning to the abdominal cavity and the hernia from entering the scrotum.

TUMORS OF THE TESTIS.—We may at birth find an enlargement of the testis due to sarcoma or carcinoma. The former is much the more common. As an illustration of this type of disease I will show you this infant (Case 182), who was operated upon by Dr. Lovett three months ago.

After a normal labor, it was noticed that the infant had a swelling as large as an egg on the right side of the scrotum. This was at first considered to be a hydrocele, but, as it steadily increased in size, more active measures were employed. On bandaging, the skin over the tumor, which was at first normal, became so much inflamed that lead water had to be used as a wash. The treatment had no effect on the size of the scrotum or on the discomfort which it seemed to cause the little patient. The infant was now two weeks old.

As the tumor had a semi-fluctuating feeling, aspiration was tried, and 2 c.c. (½ drachm) of blood-stained serum were obtained. A second tapping gave only a little clear blood.

The infant was then brought to Dr. Lovett for consultation. The tumor was found to be quite large, being 20 cm. (8 inches) in circumference, and it had nearly hidden the penis its mass. After a preliminary tapping, which gave the same result as the previous one, an peration was performed. A testicle 5 cm. (2 inches) in diameter was removed. The cord, hich was found enlarged to a diameter of 1.2 cm. (½ inch), was removed as far up as the xternal ring, but laparotomy, in order to extirpate the cord as fully as possible, was not erformed. The infant made an excellent recovery, and no return of the growth can be tected in either the scrotum or the pelvis.

On section, the tumor was found to contain scattered throughout its mass about a zen cysts of different sizes. Microscopic examination showed it to be a mixed-cell sarma with fibrous and myxomatous tissue in different parts of it. Here and there were attered small areas of cartilage and a few striped muscular fibres. As you know, muscle bres are found in the tumors of only two organs, the kidney and the testicle, and even in hese they are very rare.

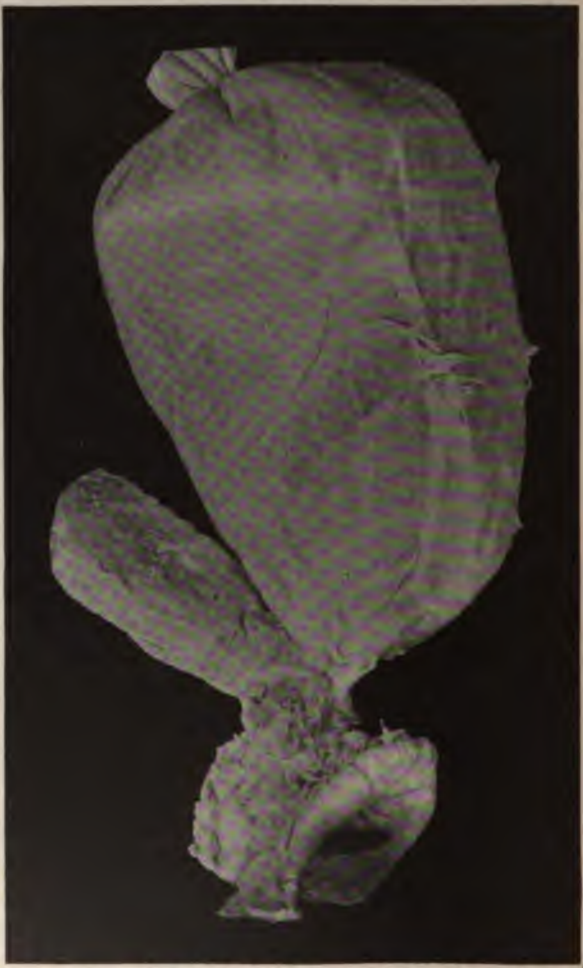
MALFORMATIONS ABOUT THE RECTUM.—In speaking of arelip I told you in a general way how at an early stage of development of he embryo the intestinal canal ended blindly and afterwards by an inagination of the outside wall a communication was brought about and the tomodæum formed. An analogous process of development goes on at the ther end of the intestinal tube, and results in the formation of the rectum nd anus. The hind-gut at first ends blindly, then as it descends it is met by an ascending dimple, and usually these two fuse and the *protodæum* is formed.

As in the mouth a series of malformations may arise from a failure in the completion of this process, so in the anal region we may meet with a similar series. The rectum may have come into its normal relations and the anal depression have failed to form, or it may have gone the whole of the distance between the end of the intestine and the skin and yet the final step, the fusion of the membranes, have failed to take place. To both of these, and to any intermediate condition, the name of *imperforate rectum* is given. On the other hand, with the rectum and the anus fused we may, nevertheless, find a thin parchment-like membrane spread over the external orifice just where the skin and the mucous membrane join. This is called *imperforate anus*.

When an infant is born the physician should carefully examine it, in order to determine whether it has any malformation. The most important malformations which it is necessary to recognize are those at the anus. Unless an infant has a passage of meconium soon after its birth, an examination should be made in the rectum with the finger, and if the anal opening is found to be closed, either just at the outlet or higher up, we must consider what is to be done to relieve this condition. If nothing but a web obstructs the anus, we can easily break it through with a director and then dilate the orifice with the finger. If there is more than the thinnest bulging membrane, a cutting operation will have to be done, and perhaps a severe one. The general principles are to begin with a staff in the bladder, and, using this as a guide, to make a careful and systematic dissection in search of the missing gut. If we fail in this, we should perform the operation known as Littré's, which consists in opening the sigmoid flexure in the inguinal region and making an artificial anus there. There is a still more sever form of operation, in which an attempt is made to cut through the sacrum and make the gut open there, but it has many practical objections in very young infants.

Here is a specimen (Fig. 89) taken from an infant, a patient of Dr. John Ware's. It died on the fourteenth day of its life. From the time of its birth it had constant vomiting and much distress. The autopsy showed the lower part of the small intestine and the whole of the large intestine to be acutely inflamed, and that there was ulceration of the latter. You see that the upper and lower portions of the intestine terminate in a cul-de-sac a short distance from the anus and are separated for about 0.85 cm. ($\frac{1}{3}$ inch).

FIG. 89.



Imperforate rectum. Male, 14 days old. Warren Museum, Harvard University.

The large bulging mass above represents the rectum distended with meconium and separated from the anal opening by an isthmus of soli

ve tissue. The smaller mass in front (to the left in the picture) is
der.

CLUSION OF THE VAGINA.—Sometimes we find a thin gray
extending across the mouth of the vagina from just below the
opening to the posterior commissure and blocking up the vagina.
be complete or partial. This condition should be dealt with while
nt is still young, as if left until puberty it will cause a retention of
ses, and, moreover, by that time will have become much thicker and
quite vascular. It is easily broken through in the young child,
piece of carbolized cotton be put between the torn edges to prevent
hering, the malformation can be cured permanently. Atresia from
ation of the labia is said to occur in rare instances.

POSPADIAS.—The malformation known as *hypospadias* is the
an arrest of development in the formation of the urethra and of
us spongiosum. The urethral groove should normally be converted
nal by the growth and joining together of its sides. This process
t the base and extends to the end of the penis. By an interruption
process the urethra may be brought to an end and open at any point
the peno-scrotal angle and the base of the glans. In the most
forms of hypospadias the glans alone is imperforate.

ATMENT.—The treatment is wholly by plastic operation, and it
the most delicate surgery to obtain a good result in the face of the
rious obstacles that this malformation presents.

SPADIAS.—The malformation of *epispadias*, in which the urethral
ens upon the dorsum of the penis, is still more difficult to deal with
hypospadias. It is commonly associated with extroversion of the
, and is very rare.

artial plastic operation and the wearing of some form of urinal con-
bout all that can be done for these cases.

LECTURE XX.

THE EXTREMITIES.—GENERAL DISEASES.

FINGERS.—Various malformations of the extremities are met with in new-born infants, and I happen to have one of these to show you to-day.

This infant (Case 183) has six fingers on each hand instead of five.

CASE 183.

Infant's hand with six fingers.

This is only one of a type of malformations which you are liable to encounter. Another malformation of this kind, called webbed fingers, is quite common.

TOES.—Infants are at times born with *extra toes* and *webbed toes*, and it becomes a surgical question to determine whether they shall be operated upon. This, of course, is a question of orthopædics, and is one which we need not deal with except so far as to appreciate the importance of preparing the foot properly for future use.

The greater freedom of movement required for the fingers, and the fact that the hand is always in sight, render surgical interference much more necessary in malformations of the hand than in those of the foot.

Congenital hypertrophy of the feet and hands, and *congenital deficiency* of one or more extremities, may be spoken of in this connection, but are too rare to be more than referred to.

These malformations have been thoroughly described by Thomas Annandale, and I shall refer you to his work on this subject for information regarding them.

CLUB-HAND AND CLUB-FOOT.—Club-hand and club-foot are congenital malformations which may be due to an undeveloped condition of either the bones, the ligaments, or the muscles. In the more simple forms the extremity is pulled into the malposition by the action of contracted muscles and tendons, while in the severe forms the bony framework may be so misshapen that the separate segments are almost unrecognizable. Club-hand is often accompanied by absence of the radius.

TREATMENT.—The treatment of this class of deformities is, of course, purely in the province of the orthopædic surgeon. All that I wish to do in referring to them is to suggest to you how much may be accomplished by simple manipulations with the hand. The mother should be instructed to rub the foot and leg twice daily, and to make firm pressure against the shortened muscles by trying to bring the hand and foot into the normal

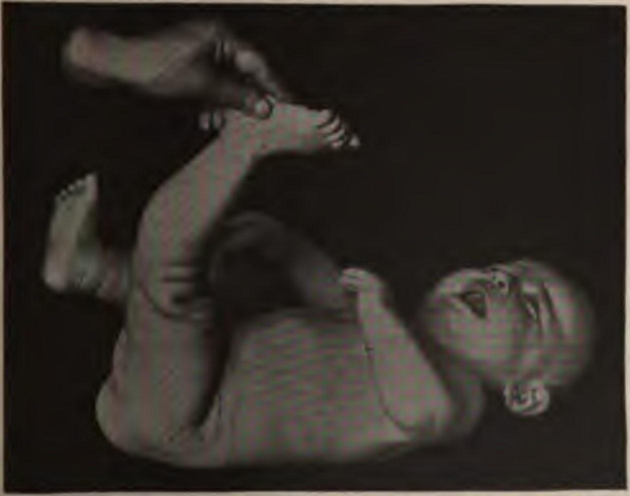
n. I have seen light cases cured by this simple means, and even ately severe ones so much benefited that subsequent treatment with edic apparatus became much easier.

NGENITAL DISLOCATION OF THE HIP.—Congenital dislo- s of all the joints are sometimes found, the most frequent and most tant being dislocation of the hip. This is now thought to be caused ulty development of the acetabulum and the head of the femur. The oms are of a kind that readily escape notice during infancy, and are een when the child should begin to walk. It is then noticed, if he old himself on his feet at all, that the abdomen is very prominent, ck arched, and the buttocks seemingly enlarged: at least this is the the deformity is bilateral, which is the form usually met with. On ning the joint we find that the trochanter is above Nélaton's line, but by traction on the leg be drawn down to its proper place without g any discomfort to the child. If the deformity is unilateral, one leg ppear shorter than the other, and the child will walk with a rolling This condition should be carefully looked for when an infant at the fourteen or fifteen months has made no especial attempt to walk, or on attempting to do so it does not succeed.

s operative treatment has not proved very successful in these cases and to be employed until the child is over three years old, the best method eatment is by massage. If the disease is unilateral it should in addi- to the massage be treated with a high shoe.

ONGENITAL DISLOCATION OF THE KNEE.—Next in order requency to congenital dislocation of the hip, but rare in comparison, is

CASE 184.



Congenital partial dislocation of the knee. Female, 5 months old.

islocation, or rather a partial dislocation, of the knee. In this condition tibia is found riding forward upon the femoral condyles, so that the knee-joint can readily be put into hyperextension and the toes made to point towards the forehead. Here is a case (Case 184) which illustrates this condition.

The infant is five months old, and was delivered with instruments after a long labor. It was a head presentation. As you see, there is a remarkable range of motion at the knee. Not only can I put it into hyperextension, but I can move it considerably from side to side. This abnormal mobility is due to a very lax condition of all the tissues about the knee, and especially of the lateral ligaments. It has been treated, its mother tells me, by a plaster bandage for about a month, and no improvement has taken place. We should not be at all surprised at this result, when we consider that keeping the knee immovable simply tends to increase the already existing atrophy.

A far better form of treatment is the application of a light steel support which will check all lateral motion and by means of a "stop joint" at the knee will allow flexion but will prevent hyperextension. This apparatus, together with systematic massage, will probably effect a cure.

BIRTH PARALYSIS.—Birth paralysis will be considered in connection with diseases of the nervous system. It may be present either in the muscles of the face or in those of the extremities, and is due to pressure upon the nerves made by the forceps or by too great traction.

CONGENITAL OBLITERATION OF THE BILE-DUCTS.—One of the rarer forms of congenital malformations in new-born infants is represented by the *obliteration of the bile-ducts.* The most extended work which has appeared in the literature of this subject is that of Dr. John Thompson, of Edinburgh, whose valuable thesis I have used in my description of the disease.

SYMPTOMS.—The infants who are born with this disease are either icteric at first or become so within the first few weeks of life. They often appear otherwise healthy and well nourished. In some cases there is a discharge of normal meconium followed by colorless dejections. In other cases the fæcal movements are clay-colored from the very first and remain so. The urine is deeply stained with bile. The jaundice is of a dark-greenish tinge, lasting until death. Spontaneous hemorrhage from the umbilical cord commonly occurs within the first two weeks, and in other localities in those infants who survive this early period. The liver and spleen are increased in size. If the infants survive for some months they become more or less emaciated. Convulsions and vomiting are apt to occur, and death usually takes place from exhaustion or from some trifling intercurrent disease.

PATHOLOGY.—There are a number of different morbid processes which have been supposed to produce this pathological lesion of the ducts. Each of these processes has in certain cases, in all probability, had much to do with causing the disease, but it is usually the combination of one or more of them which must be considered in determining its etiology. Thus, the results of intra-uterine peritonitis, by compressing the ducts, or by being a source of inflammation which has spread to the walls of the ducts, may finally cause their obliteration. A primary inflammation or lesion of the or of defective development as being the chief cause. This malfor- a probably affects to a considerable extent the walls of the ducts, and, ompson has stated, it consists in the narrowing of their lumen. The erence which is thus caused to the outflow of bile gives rise to a hal condition which finally blocks and obliterates the ducts, owing to flammatory process spreading to the walls of the ducts and the gall- er. This progressive inflammation goes on slowly spreading, the local ion gradually becoming worse during many months if the patients live. bliterated ducts or gall-bladder, or portions of them, may entirely ear, not even leaving a distinct band of fibrous tissue to indicate their al position. The obliteration generally becomes complete at a variable arly period of intra-uterine life: occasionally it does not occur until birth. The occurrence of peritonitis is probably in most cases second- the blocking of the ducts.

'hen the lumen of the duct has become so narrowed that the bile does ss freely into the intestine, a cirrhotic condition begins in the tissues liver, and as it goes on interferes with the functions of that organ.

t the post-mortem examinations of these cases the liver usually is to be much enlarged and its tissues to be increased in consistency: it a dark-brown color, owing to the presence of numerous masses of sated bile in the smaller bile-ducts. In a large number of cases is found a complete obliteration of some part or parts of the hepatic, on, or cystic ducts, or of the gall-bladder, while, with very few ex- ns, implication of the blood-vessels is conspicuously absent.

n speaking of the explanation which may be given for the occurrence e symptoms which I have just mentioned, Thompson remarks that the earance of the disease in several members of the same family can be ined only by the theory that a congenital defect of development is in cases the cause of the malformation. The fact that the onset of the ways, one of which may arise if mercury has been administered. The tendency to spontaneous hemorrhages may be due to the occurrence of a condition of chronic blood-poisoning, since the arrest of the outflow of bile damages the liver to such an extent that its functions are interfered with and organic fluids of a poisonous nature may thus pass into the circulation. The enlargement of the spleen, the convulsions, and the vomiting are probably more or less connected with this same condition of blood-poisoning. The fact that the children live as long as they do, and usually do not become emaciated in the early days of life, is to be explained on the ground that the presence of bile in the intestine is not absolutely necessary for digestion. When the nutrition and general health begin to suffer, it is probably due to the interference which the secondary changes in the tissues of the liver are causing with the more important functions of that organ.

TREATMENT.—The treatment must necessarily be symptomatic, there being no known means by which we can counteract the results of this malformation.

CONGENITAL OBLITERATION OF THE INTESTINE.—I shall merely refer to a malformation which is represented by an obliteration of the intestine. Malformations of this kind may arise from constrictions of the parts affected by fibrous bands, probably the remains of peritoneal adhesions.

CONGENITAL MALFORMATIONS OF THE ŒSOPHAGUS AND STOMACH.—Congenital malformations of the œsophagus and stomach are rare, and can best be described in connection with diseases of these parts.

MALFORMATIONS OF THE HEART AND THE BLOOD-VESSELS.—I shall defer what I have to say concerning the various anomalies of the heart and blood-vessels until later (Division XVII., p. 1020).

ASPHYXIA.—The earliest pathological condition which is brought to our notice at birth, and one which requires immediate treatment, is asphyxia. This condition, which is a failure of the circulatory mechanism to assume its extra-uterine function of oxygenating the blood, endangers the life of the infant from carbonic acid poisoning. It may arise either from mechanical pressure, as from winding of the cord around the neck, from an incomplete expansion of the pulmonary alveoli, *atelectasis*, or from other causes connected with the imperfect oxygenation of the blood, of which we have very little knowledge. In any case the cause, if known, must be quickly removed. This class of cases belongs so directly to the province of obstetrics that it need hardly be more than mentioned in a course of lectures on pediatrics. Prompt measures for performing artificial respiration, as by Credé's method, and the stimulation of the pneumogastric nerve by the application of heat, cold, and electricity, should be borne in mind: they are well described in Dr. Edward Reynolds's work on practical midwifery.

ACUTE FATTY DEGENERATION OF THE NEW-BORN (Buhl's Disease).—An affection which has been called acute fatty degeneration of the new-born was described by Buhl in 1861. It is not a disease of common occurrence, and its etiology and pathology have not yet been satisfactorily

orrhage in the new-born as omphalorrhagia and melæna. If the
us causes of hemorrhage from the cord had been more carefully
ed anatomically, the disease would probably not have remained so
known.

MPTOMS.—The infants who are affected by this disease are usually
a condition of extreme asphyxia without any apparent cause for it.
pts at resuscitation are, as a rule, only partially successful, and at times
all so, many of the cases dying at once. Diarrhœa is commonly
, and is often accompanied by blood from the rectum. There is
mes vomiting of blood. Often, after the cord has separated, there
e a parenchymatous hemorrhage, which, although small in amount, is
es sufficient to cause death. There is usually a bluish color of the
which changes gradually to yellow or a mixture of yellow and blue.
rrhages occur frequently in the skin, the conjunctivæ, the mucous
ranes of the mouth and nose, and sometimes the outer ear. Icterus
e present in these cases, and at times may become intense. Sometimes
a occurs, and without any noticeable rise of temperature there may be
d collapse, followed by death, commonly within the first fourteen days
. These symptoms are not always so well marked as I have just
bed them. The external hemorrhages may not occur, and the cyanosis,
at first, may rapidly increase and be followed by sudden death. This
mes happens so quickly that we are reminded of the conditions which
t with in cases of death by violence.

AGNOSIS.—A definite diagnosis cannot be made without a careful
copic examination. The disease must not be confounded with phos-
s or arsenic poisoning, where the organs undergo similar pathological
s. The history of the case and a chemical examination of the organs
able you to eliminate these other causes of fatty degeneration. The
ntial diagnosis between this disease and cases of sepsis in which

by death from suffocation that its presence becomes a question of great importance from a medico-legal stand-point. The cyanosis, the condition of the lungs, and the ecchymoses, also the absence macroscopically of other organic changes, can easily suggest suffocation. For this reason in all cases of death among new-born children where there is a suspicion of asphyxia, a careful microscopic examination should be made of all the organs.

PROGNOSIS.—The prognosis in this disease is very unfavorable: all the cases in which the symptoms are pronounced die. It is possible that the milder forms of the disease can recover, but as yet we do not know enough about this class of cases to state what proportion of them lives.

ETIOLOGY.—The etiology of acute fatty degeneration of the new-born is very obscure. The disease occurs in animals as well as in human beings, but the investigations made by different observers both on animals and on infants are so varied in their results that we cannot at present consider that we know much about the cause of the disease. It is significant, however, that Buhl in his classic description of the disease states emphatically that the vessels of the cord are not affected, so that if it is due to sepsis the sepsis must have occurred in intra-uterine life through the mouth, the intestinal canal, or the umbilicus, but without producing any change in the umbilical vessels. This can scarcely be considered probable. We know nothing concerning the etiology of this disease, not even whether it is of intra- or extra-uterine origin.

PATHOLOGY.—The pathological conditions which represent the disease consist of a parenchymatous inflammation, followed by a fatty degeneration of the tissues of the heart, liver, and kidneys, and hemorrhages in the various organs. The post-mortem examination of infants dying of this disease, as a rule, shows the following changes. The cadaver is livid and usually icteric. Hemorrhages and œdema are often found in the skin. The umbilicus and the tissues surrounding it are at times stained with blood, but, as a rule, are otherwise normal. The umbilical vessels are in most cases normal. These hemorrhages are especially found in the dura and pia mater, in the pleura and pericardium, and in the connective tissue of the mediastinum: they also occur in the thymus gland, in the peritoneum, in the muscles, and in most of the mucous membranes.

The *brain* is found to be soft, usually full of blood, and, if icterus is present, is stained yellow.

The *lungs* often show hemorrhagic infarction, and in the bronchi bloody mucus or pure blood. The alveolar epithelium is in a condition of fatty degeneration.

The muscles of the *heart* are friable. In the early stages they are rigid and dark red, while in the later stages they become softer and paler. In almost all of them the process of fatty degeneration is found.

In recent cases the tissues of the *liver* are blood-red, while in the later stages they are pale and icteric. The liver-cells contain fat-drops and granules of biliary coloring matter.

The *spleen* is usually found to be enlarged, and its parenchyma is soft nd almost fluid.

Hemorrhages may be found in the walls of the *stomach* and *intestine*, nd their cavities are often found to be filled with blood.

Multiple hemorrhages are found in the parenchyma of the *kidney*. The ortex is swollen in the early stages, is filled with blood, and is pale and ellowish. The epithelium of the convoluted tubules shows marked fatty degeneration, and the canals are often filled with fatty degenerated material.

The process of fatty degeneration does not in all cases affect all the rgans. In some the changes may be absent or a parenchymatous condition may be present.

TREATMENT.—From what I have said concerning this disease you will eadily understand that the treatment is usually unsuccessful. Stimulants hould be used and the food carefully regulated.

LITERATURE.—You may perhaps like to know the sources (Table 90) from which Runge has obtained his facts in describing the acute fatty degeneration of the new-born.

TABLE 90.

1. HECKER, v., u. BUHL, Klinik d. Geburtskunde, 1861, Bd. i. S. 296.
2. HECKER, v., Monatsschrift f. Geburtskunde, Bd. xxix. S. 321; Bd. xxxi. S. 197; Bd. xxxii. S. 197.
3. HECKER, v., Arch. f. Gynäk., 1876, Bd. x. S. 537.
4. MÜLLER, P., Die acute Fettentartung der Neugebornen, Handb. der Kinderkrankheiten, von Gerhardt, 1877, Bd. ii. S. 186.
5. COHNHEIM, Vorlesungen über allgem. Pathologie, 2. Aufl., Bd. i. S. 651.
6. HERZ, Oesterr. Jahrb. f. Pädiatrik, 8. Jahrg., 1877, S. 139.
7. RUNGE, MAX, Charité-Annalen, 7. Jahrg., 1882, S. 720 u. 727.
8. FÜRSTENBERG, Virchow's Arch , 1864, Bd. xxix. S. 152.
9. ROLOFF, Virchow's Arch., 1865, Bd. xxxiii. S. 553.
10. ROLOFF, Virchow's Arch., 1868, Bd. xliii. S. 367.
11. BOLLINGER, Virchow's Arch., 1873, Bd. lviii S. 329.
12. BIRCH-HIRSCHFELD, Handb. der Kinderkrankheiten, von Gerhardt, Bd. iv., 2, S. 707.
13. FRIEDBERGER, FRANZ, u. FRÖHNER, EUGEN, Lehrbuch d. spec. Pathologie u. Therapie d. Hausthiere, III. Auflage, 1892, Bd. ii. S. 16 ff.

INFECTIOUS HÆMOGLOBINÆMIA OF THE NEW-BORN Winckel's Disease).—Infectious hæmoglobinæmia is an affection which is met with in new-born infants usually in the early days of life, and, as a rule, arises as an endemic disease in hospitals. The specific micro-organism which produces it has not yet been discovered, yet the fact of its endemic character and the changes which are produced in the blood warrant us in supposing that it is an infectious disease. Although it had been described at an earlier date, yet the most systematic description of it which had appeared up to the year 1879 was that by Winckel, who in that year reported twenty-three cases of an endemic affection observed by him at the Dresden Lying-in Hospital. The disease was characterized by extreme cyanosis, icterus, hæmoglobinuria, somnolence, rapid collapse, and the absence of fever.

Although in many respects it resembled closely the acute fatty degeneration which I have just described to you, yet it had such characteristic symptoms and conditions of its own that it cannot, until further light shall have been thrown on the subject, be separated from that disease.

I am indebted to Runge for a description of this disease. An analysis of Winckel's cases shows that it usually begins on the fourth day of life, and that it may attack strong, well-developed infants. The course of the affection is very rapid, its average duration being about thirty-two hours. Twenty-five and a half per cent. of all the children born at the time when this epidemic occurred had the disease, and of these nineteen per cent. died.

SYMPTOMS.—The first symptoms were generally restlessness and cyanosis, not only of the face but also of the body and extremities, and especially the back. The color increased progressively until it became a deep blue. To this was added an icteric color, which when death did not occur within twenty-four hours became very marked. The respiration was rapid; the pulse was not especially increased in rate. The rectal temperature never rose higher than 38.1° C. (100.6° F.). The skin generally felt cool. Vomiting and diarrhœa occurred in some cases. The most striking symptom was the appearance of the urine. It had a pale-brownish color, and was passed frequently, and often with considerable straining. An examination showed that the color was due not to bile, but to hæmoglobin. In the sediment were found numerous epithelial cells from the walls of the kidney, granular casts with blood-corpuscles adherent to them, micrococci, masses of detritus, and urate of ammonia. A small quantity of albumin was present. Later in the disease convulsions occurred, followed rapidly by death. It was noticed that if the skin where the cyanosis was most marked was scratched and then pressed hard, a tenacious, almost black-brown fluid exuded. An examination of the blood showed a marked increase of leucocytes and numerous granules.

In other cases besides those of Winckel's where the blood was examined the condition was found to be one of hæmoglobinæmia. The percentage of hæmoglobin was high, and free hæmoglobin was found in the blood-serum, while the erythrocytes were greatly reduced in number, at times amounting to only 1,700,000 or even less.

PATHOLOGY.—A careful post-mortem examination of Winckel's cases showed that there was cyanosis of the external and internal organs. Except in one instance, no pathological condition of the vessels of the cord was described.

The cortex of the *kidney* was found to be wider than normal, to be of a brownish color, and to present numerous minute hemorrhages. In places the pyramids were entirely black-red in color, and in other places numerous black streaks were found which converged to the papillæ. This color was aused by the filling of the straight tubules with granules of hæmoglobin. ntact erythrocytes were never found.

The *bladder* was found to contain greenish-brown urine.

The *spleen* was strikingly enlarged and hard. Its length was about 7.5 m. (3 inches), and its weight 25 grammes ($\frac{5}{6}$ ounce). It was black-red in olor, and on section the surface was smooth. Microscopic examination howed a considerable accumulation of brownish coloring matter, partly ree and partly in the pulp-cells.

In addition to these appearances in special organs, minute hemorrhages ere found in nearly all the organs, but especially in the pleura, pericardium, ndocardium, mucous membranes of the stomach and small intestine, and idney: they were also found in the dura and pia mater and under the apsule of the liver. The lymph-follicles were swollen, especially Peyer's atches and the mesenteric lymph-glands.

A microscopic examination showed fatty degeneration of many impor-ant organs, especially the liver, and at times of the muscles of the heart.

The bacteriological examinations were, as a rule, negative, especially as egards the tissues of the intestine. Clumps of bacteria were found only nce in the liver and once in the kidney.

ETIOLOGY.—The etiology of this disease is obscure. Winckel had careful examinations made of the organs chemically for poisons, such as phosphorus, arsenic, and chlorate of potash, but with negative results. Examinations in regard to carbolic acid poisoning have also been made in these cases, with negative results.

The resemblance of this disease to acute fatty degeneration of the new-born is very striking. Most of the symptoms are common to both diseases. Larger hemorrhages are also not uncommon in this disease, but are not so marked as in acute fatty degeneration. The striking points of difference are the presence of hæmoglobinuria, and that large numbers of cases are affected at the same time in infectious hæmoglobinæmia, while these conditions have not been found to occur in acute fatty degeneration. In studying the literature of this disease we find a number of observations by different authors. Dr. W. S. Bigelow describes an epidemic at the Boston Lying-in Hospital in which the chief symptoms were a dark color of the skin resembling somewhat that produced by the administration of nitrate of silver, æmoglobinuria, diphtheritic deposits on certain of the mucous membranes, nd dark brown fæcal dejections. In this epidemic ten infants were attacked nd eight died, the average duration of the disease being five days. In one f these cases phlebitis umbilicalis occurred. Similar cases have been ported by Parrot and Herz in which the urine was brown and strongly inged with blood and the kidneys and liver showed the condition of fatty egeneration.

Epstein, of Prague, mentions similar cases where prominent features were the thickening of the blood, which made it impossible to get a drop to examine, and the dark brown-red color of the urine. Epstein thinks that this disease is a septic process which probably starts in the gastro-enteric tract. He believes that he can controvert the apparent absence of fever by the fact that in the diseases of new-born infants great and sudden variations of temperature occur, and in consequence the temperature, for its record to be of value, should be taken very often.

Whether this is so or not, the disease has certain peculiarities, pointing in some cases to an apparent relation with sepsis, and in others to acute fatty degeneration.

The obscurity as to the etiology of the disease has been rendered still greater by the incomplete examinations which have been made of this class of cases, with the exception of those by Winckel and Birch-Hirschfeld.

TREATMENT.—The treatment should be the administration of oxygen and stimulants, and forced feeding by means of a dropper where the infant is too weak to suck.

LITERATURE.—I have placed in this table (Table 91) the literature which Runge has made use of in his description of this disease.

TABLE 91.

1. WINCKEL, Deutsche Med. Wochenschrift, 1879, Nr. 24, 25, 33, 34, 35.
2. BIRCH-HIRSCHFELD, Deutsche Med. Wochenschrift, 1879, Nr. 36.
3. BIRCH-HIRSCHFELD, Handbuch der Kinderkrankheiten, von Gerhardt, 1880, Bd. iv., 2, S. 702.
4. EPSTEIN, Prager Med. Wochenschr., 1879, S. 343
5. SANDNER, Münch. Med. Wochenschr., 1886, Nr. 24.
6. STRELITZ, Archiv f. Kinderheilkunde, 1890, Bd. xi. S. 11, and BAGINSKY, Berl. Klin. Wochenschr., 1889, Nr. 8, same case.
7. BAGINSKY, Lehrbuch der Kinderkrankheiten, IV. Auflage, 1892, S. 59.

HEMORRHAGE IN EARLY LIFE.—Spontaneous hemorrhage occurring at some period during the early years of life is not uncommon. These hemorrhages may occur either in the skin or from some trifling traumatic lesion, or they may take place in various internal organs, and especially from the mucous membrane of the mouth and the gastro-enteric tract. A definite division of this class of cases has never been thoroughly made, so that the subject has always been somewhat involved in obscurity. The probability is that these spontaneous hemorrhages are simply symptomatic of different specific diseases, and that as our knowledge of these diseases increases we shall find it necessary to make a clear distinction between cases which now are spoken of under one head. The propriety of separating cases of spontaneous hemorrhage which occur in the early days and weeks of life from those which arise later has been shown by Dr. Townsend. He has by a series of observations corroborated the now generally accepted opinion that the hemorrhages which occur in the new-born should be separated from those met with in connection with the hæmophilia of a later period of childhood and of adults. He has called this disease the *hemorrhagic disease of the new-born.* The hemorrhages which occur in new-born infants are so general in their distribution, and yet so uniform in their general symptoms, that they can well be classed under this one heading. These hemorrhages occurring in the early weeks of life run a definite course, and end in death or in complete recovery. The self-limited nature of this affection corresponds to what is seen in the acute infectious diseases, and suggests a relationship to them. The hemorrhage may arise from the gastro-enteric tract, from the mouth, the nose, or the umbilicus, also from the skin, and in the latter case may show itself in the form of ecchymoses. Again, it may occur in the form of hemorrhages in the abdominal cavity, the meninges of the brain, the pleura, the lung, and the thymus gland.

Dr. Townsend has collected fifty cases of this disease, and has tabulated the sources of the hemorrhage, as follows (Table 92):

TABLE 92. (Townsend.)

Locality.	Cases.
Intestines	20
Stomach	14
Mouth	14
Nose	12
Umbilicus	18
Ecchymosis in skin	21
Scratch of skin	1
Cephalhæmatoma	3
Meninges	4
Abdominal cavity	2
Pleural cavity	1
Lung	1
Thymus gland	1
From the gastro-enteric tract, nose, and umbilicus, accompanied by ecchymosis in the skin	3
From the gastro-enteric tract alone	19
From the umbilicus alone	3
From ecchymosis in the skin alone	6

The mortality in these cases was 62 per cent. The bleeding first showed itself in all but three within the first seven days of life, the exceptions being on the eighth, ninth, and fourteenth days. The hemorrhage in the majority of these cases began on the second or third day, thirteen starting on the second and sixteen on the third day, while only eight began on the fourth and two on the first day. One-half of the fatal cases lasted one day or less, and all the others died within a week, except one case, in which death took place from the effects of the hemorrhage on the eighth day and several days after the bleeding had ceased. The cases that lived recovered within nine days, and two-thirds of them within five days.

The cases of pseudo-menstruation which occur not uncommonly in the early days of life should not be included in the cases which are classed under the heading of hemorrhagic disease. The *hemorrhagic disease* is apparently a general and not a local one, and is found more frequently in hospitals than in private practice. This fact is well exemplified by comparing the percentage of hemorrhagic cases which occurred among 7225 infants observed in the Boston Lying-in Hospital and its out-patient department. The percentage of the disease in the hospital itself was represented by .57, while .10 represents the proportion outside of the hospital. In Townsend's fifty cases the proportion of females to males was as 20 to 30. In four of Townsend's cases the hemorrhage took place in several places as well as at the base of the cord, but the patients recovered and the cord separated, in one case in two days and in the other three in four days after the cessation of the disease, without a fresh hemorrhage occurring.

In fourteen of these fifty cases the temperature was carefully observed, and in all but two was found to be elevated at first from 38.3° C. (101° F.) to 39.5° C. (103.1° F.), and in one case to 40.1° C. (106° F.). After the cessation of the hemorrhages the temperature was normal, and often subnormal.

To recapitulate: it would seem that we are warranted in considering the disease as one of a general nature, and infectious, for the following reasons. (1) It occurs usually in hospitals. (2) It is self-limited in its course, and, although a dangerous disease, may be recovered from in one or two weeks completely and never return. (3) The temperature is raised during the continuation of the chief symptoms, and becomes normal or subnormal when the hemorrhage has ceased.

Ritter at the Prague Foundling Hospital has also noticed a great preponderance of cases occurring in hospital deliveries over those which were met with outside of the hospitals.

In connection with the hemorrhage which occurs in the gastro-enteric tract, the tar-color of the intestinal dejections, arising from the hemorrhage taking place high up in the intestine, is noticeable. The resemblance of the color of the dejections to that of meconium may cause the disease to be overlooked. A slightly pink tinge on the napkin around the dejection is often, however, seen, and where there is a doubt as to whether the stain is from blood or not, it can usually be determined by means of the microscope. Where the corpuscles have become disintegrated, as at times occurs, the hæmin crystals may be recognized by means of a simple test which I shall speak of later. The post-mortem examination which was made in nine of these cases throws no additional light upon the nature of the affection. The source of the hemorrhage was found, but in no case were there any gross lesions of the mucous membrane or the blood-vessels. In all these cases the infants looked very anæmic. In one case cultures were made by Professor Councilman from the blood, with negative results. We do not know what the cause of this disease is, but it is probable that in the great majority of cases it has an infectious origin.

I will now show you a case (Case 185) which is especially interesting, as it shows an unusual result of the blood examination.

The infant is three days old, and presents a blanched appearance of the skin, with stains on the napkin around the intestinal discharges. These stains have been examined in the following way. A drop of the semi-liquid dejection was mixed with a little glacial acetic acid and a few crystals of common salt on a glass slide and heated to boiling. On drying e preparation and examining it under the microscope, the dark rhombic crystals of min were easily recognized, showing us that we are dealing with a case of hemorrhage king place probably high up in the intestine. Dr. Wentworth's blood examination ves the following results:

BLOOD EXAMINATION 37. (Wentworth.)

Erythrocytes	6,245,000
Hæmoglobin	125 per cent.

The blood spread out very thickly and stained poorly, but the polynuclear leucocytes peared greatly in excess of the other forms.

The cases which are commonly designated as *melæna neonatorum* should classed under this heading of the hemorrhagic disease of the new-born, d are represented by this case (Case 185), in which the blood examination as made by Dr. Wentworth. The child died in a few days.

An interesting case (Case 186) of this disease was seen by Dr. Townsend d myself in consultation with Dr. Bush.

A male infant apparently healthy at birth developed on the third day of its life ecchyoses on its head, groins, and one foot. There was also hemorrhage from the upper part of e intestine on the fifth and sixth day, the dejections being tar-colored from altered blood hich simulated meconium. On the fifth day the child developed a marked paralysis of e left side of the face, and to a less degree of the left arm and leg, presumably from a eningeal hemorrhage. On the seventh day of the disease the hemorrhage had apparently ased, as the paralysis was beginning to disappear. On the twelfth day the paralysis of he left arm and leg had improved: there was, however, still some paralysis on the left side f the face, but this did not continue to any great extent, and in the third and fourth weeks ecided improvement took place in the child's condition, and there were no longer any evidences of hemorrhage nor any paralysis.

This infant improved rapidly during its first year, and is now living, healthy and strong. It learned to walk and talk rather later than usual, but now at four years of age is in a normal condition both mentally and physically.

I have met with a number of cases in which these hemorrhages occurred and in which they varied greatly as to extent and persistence. The cases in which umbilical hemorrhage was present showed this same tendency to self-limitation, and could be distinguished from those which are classed under hæmophilia. In fact, it is probable that most cases of umbilical hemorrhage are caused by infection and are not especially connected with hæmophilia.

I have here to report to you a case (Case 187) of umbilical hemorrhage in a male which illustrates what I have just said concerning the desirability of separating the hemorrhages taking place in the early days and weeks of infancy from those which occur later and in childhood.

The parents of the infant were well and strong, and were Russian Poles. They had another child, eighteen months old, which was healthy. The mother had never had any miscarriages, and stated that her parents were healthy, as were also those of the father.

The labor was a normal one, the child presenting in the first position, and nothing abnormal was noticed, except that the placental end of the cord continued to bleed quite freely notwithstanding the application of two ligatures. On the day following the delivery the mother and infant were both doing well; the latter showed slight signs of icterus, but nursed well, and the former had plenty of good breast-milk. The infant continued t thrive, except that there was a slight hemorrhage around the insertion of the cord, which fell off on the eighth day. After the separation of the cord a slight hemorrhage from th umbilicus continued. On the thirteenth day the hemorrhage increased and became s extensive that I was sent for to see the infant. It was then found to be decidedly jaundiced, though not deeply so. It was nursing well, but looked thin and puny. Nothin abnormal was found on making a physical examination. Pale watery-looking blood wa oozing from the umbilicus, and quite a large cloth had been soaked with the blood fro the umbilicus, giving evidence of a considerable hemorrhage. The umbilicus was plugge with small pieces of lint soaked in perchloride of iron, firmly compressed by a bandage, an alternate drop doses of fluid extract of ergot and tincture of chloride of iron were order to be given three times a day.

On the fifteenth day the hemorrhage had somewhat abated, but it was not thoug advisable to remove the bandage; the ergot was omitted, on account of nausea an vomiting.

On the sixteenth day the infant was reported to have vomited and cried a great dea and the plugs of lint had been forced out of the umbilicus, leaving a bleeding surface; th umbilicus was then tamponed with Monsel's solution of subsulphate of iron; the tinctu of chloride of iron was omitted, as it caused vomiting. The hemorrhage then lessened an at times ceased.

On the seventeenth day the older child pricked the infant's lip with a pin, and on th eighteenth day the lip was found to be still bleeding. The point of hemorrhage wa cauterized with a stick of nitrate of silver. This controlled the hemorrhage for about tw hours, when it returned and continued. Compression of the lip finally stopped the hemorrhage on the twenty-fourth day.

On the nineteenth day the hemorrhage had ceased at the umbilicus, and the child continued to nurse well.

On the twenty-ninth day the bandage and lint were removed from the umbilicus, an the abdomen was washed. There was no hemorrhage; the child looked better, and the was not so much icterus.

On the thirtieth day the infant was reported to be perfectly well. It continued t thrive from this time, with no recurrence of the hemorrhage.

On the sixtieth day, although I advised that the operation should not be performed the infant, in accordance with the Jewish custom, was circumcised. I was present at th circumcision, to see if the hemorrhage would be easily arrested. The circumcision was performed without accident, and the hemorrhage was immediately arrested by a weak solutio of iron. From this time there was no hemorrhage, and the child continued to be strong and well.

This case is an instance of the self-limitation of the hemorrhagic disease of the newborn, since, although it was a pronounced case of umbilical and general hemorrhage at the beginning of the infant's life, this tendency had ceased by the end of the second month, as was evidenced by the ready control of the hemorrhage after the circumcision.

In another case (Case 188) which came under my notice the hemorrhage took place from the umbilicus in the early days of life at the time of the separation of the cord, and was completely uncontrolled even by ligatures passed around needles introduced through the skin of the abdomen on either side of the umbilicus This case eventually recovered.

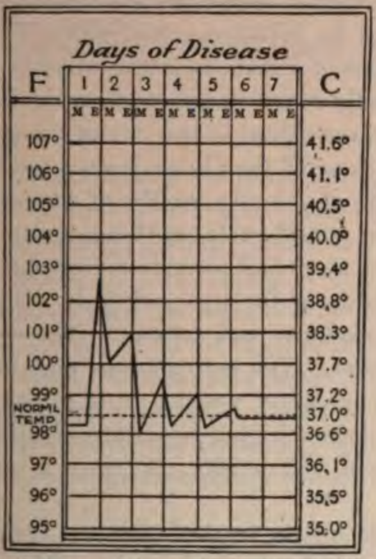
In none of these cases has a tendency to bleeding developed in later life.

At times we meet with what are apparently very mild cases of thi disease. I have here the record of a case which occurred in the practice o Dr. George Haven, with whom I saw it in consultation.

The infant (Case 189), a girl, well developed, and weighing 3358 grammes (about pounds 6 ounces), was born at 12.45 A.M. Nothing abnormal was found on examining and it was perfectly quiet until fourteen hours after its birth, when it began to be very restless. This restlessness continued, and the temperature, which at birth was 38.6° C. (101.5° F.) in the rectum, began to rise, until at the end of twenty-four hours it had reached 39.4° C. (103° F.). When it was thirty-six hours old, minute hemorrhagic maculæ were noticed, first on the back of its right hand and arm and then on the right side of its back. A few hours later a number of these maculæ also appeared on the right side of the chest, near the arm. It nursed vigorously, and did not show any signs of weakness, but its respirations were at times quite irregular. From this time no new lesions of the skin appeared, and no hemorrhages from any other locality, the maculæ gradually fading away in ten days. After the first day the temperature fell gradually, and on the fifth day was again normal. Whether there was any loss of weight during the first ten days of life was not known, as it was considered unwise in the infant's precarious condition to weigh it.

Here is the temperature chart (Chart 9), which illustrates what I have already told you in describing the disease,—namely, the rise of temperature, and in favorable cases the return to the normal degree in a few days.

CHART 9.



Hemorrhagic disease of the new-born. Female, 24 hours old.

The cord separated on the tenth day without hemorrhage, and subsequently no abnormal symptoms arose, and the infant continued to thrive during the whole period of its lactation.

HÆMOPHILIA.—In contradistinction to the hemorrhages of infectious origin which occur in the early weeks of life is that class of hemorrhages which, as I have already said, can be classed under the term hæmophilia.

Hæmophilia simply means a morbid condition characterized by a tendency to bleed spontaneously or from any insignificant wound. Individuals who are liable to bleed in this way are designated as having a hemorrhagic diathesis. The disease is not especially common in the early weeks of life, and usually occurs at a later period of development. It begins to be more frequent towards the end of the first year, and is apparently well established in the second year and later in childhood. It does not have a self-limited course, as is the case with the other form of hemorrhage. It is not infectious, and is not accompanied by fever. It may be for many years masked, and then may arise from some trivial cause, such as the extraction of a tooth. It is a dangerous disease, and death is very liable to occur from inability to control the hemorrhage.

There is no treatment which has been found successful in these cases beyond the active local employment of styptics and compression.

TETANUS NEONATORUM.—Although the group of symptoms representing the disease usually known as *tetanus neonatorum*, or *trismus nascentium*, is essentially of a nervous character, yet, as it occurs invariably in the early weeks of life, I have thought it best to speak of it in this connection.

The whole course of the disease, its self-limitation, and the high temperature at the time of its invasion, would naturally lead us to classify it among the other diseases of infectious origin which I have just described to you. The disease usually occurs in infants from the third to the twelfth day of life, and is almost always fatal in two or three weeks.

ETIOLOGY.—The cause of the disease is supposed to be the same as that of tetanus in the adult; that is, the *bacillus* of *tetanus*.

SYMPTOMS.—After considerable restlessness and muscular twitching lasting for some hours, the infant assumes a very characteristic appearance. There is extreme rigidity of the legs and body. This rigidity sometimes takes the form of opisthotonos and trismus. The eyes are almost closed, but the infant is sleepless. The entire trunk and limbs are so stiff that the infant remains in whatever position it is placed in. It is unable to nurse, and is found to have a high temperature, occasionally reaching 40° C. (104° F.), and a pulse of 150 or 160. At times it will have slight convulsive attacks.

This disease is epidemic in tropical climates, but as we see it is usually of a sporadic nature. It is extremely fatal. When recovery takes place the improvement is very gradual, the temperature and pulse decreasing and the rigidity of the muscles passing away very slowly, with at times a recurrence of the symptoms.

TREATMENT.—The treatment of this disease has thus far been very unsatisfactory, although a great number of drugs have been employed, such as atropia injected subcutaneously, and such sedatives as bromide of potash and hydrate of chloral.

The form of treatment which appears to me most rational is to place the child during the continuation of the tonic spasm in a warm bath and to give it .06 gramme (1 grain) of hydrate of chloral every hour until the effects of the drug are shown by the lessening of the muscular rigidity and by a disposition to sleep. In addition to this treatment, small quantities of milk, 15 c.c. (about ½ ounce), should be given to the infant by means of a dropper every hour, and to each feeding three drops of brandy or some stimulant ould be added. Under this treatment a certain number of cases have been nown to live.

I have here a case (Case 190) which was first brought to the hospital wo days ago with the following history :

A male, said to have been healthy at birth and to have nursed without difficulty during he first week of its life. It then refused to nurse, apparently from inability to open its jaws. It sometimes cried, but feebly. There were no convulsions, no vomiting, and no rigidity in ny other part of the body. The temperature was not taken. On physical examination it was found that, although the infant could swallow, the jaw could not be opened wider than 1.2 cm. (½ inch). On forcing the finger between the jaws, nothing abnormal was discovered in the mouth or pharynx. The respiration was regular, but rather shallow, and there was no evidence of injury. Nothing else abnormal was discovered about the infant.

The infant was given .06 gramme (1 grain) of hydrate of chloral three or four times in the twenty-four hours, and to-day shows marked improvement, and, with the exception of still being unable to open the jaws widely, nothing else abnormal has been discovered. The rectal temperature is to-day normal.

The infant has probably passed through the active part of the disease in safety, and it eems likely that it will recover.

This, of course, is not a typical case of tetanus neonatorum, but is one of the milder forms of trismus.

SCLEREMA NEONATORUM.—*Sclerema neonatorum* is a disease which occurs in the early days of life, and usually among those who are born in the midst of exceedingly poor hygienic surroundings and in cold weather. It is characterized by a hardening of the skin and the subcutaneous cellular tissue and by a great reduction in the temperature. The tissues continue to grow cooler and harder until death, which occurs usually about the ninth day. It is a rare and exceedingly fatal disease.

It should not be looked upon as a local disease of the skin, but as some obscure constitutional affection of the respiratory and circulatory systems, as shown by the shallow respirations and the diminished activity of the circulation.

SYMPTOMS.—Soon after birth, spots of circumscribed hardness appear on the skin. These spots soon become diffuse, and the disease, starting, as it usually does, in the feet or the calves of the legs, passes up the thighs to the trunk. It may, however, first appear upon the face and upper extremities, though not commonly. The skin has a waxy and glistening look, and is hard and cold ; the limbs become thick, stiff, and misshapen. The infant soon grows weak and somnolent, and refuses to take its food ; the breathing becomes rapid and superficial, the voice is weak and whimpering, and the pulse small and retarded. Towards the end of life a discharge of bloody serum from the mouth and nose often occurs, and death takes place seemingly from inanition.

TREATMENT.—There is no treatment which has been especially successful in this disease, but the affection should be recognized at once, and energetically treated with inunctions of hot oil and by massage and stimulants.

A number of cases of this disease have been reported in Europe, and several in this country, notably by Osler. Some investigators think t they have found characteristic changes in the skin. The observations Northrup, however, who published the first report of a typical case of t kind in America, seem to show that there is no definite lesion of the s Northrup made a careful study of his case, and has plainly shown by s tions of the skin compared with normal control specimens that the histol of the disease does not reveal any change which can be regarded as cha teristic. Dr. Northrup's case embodied every feature of the typical scler of the new-born. The infant was a foundling, born in a wretched, da habitation, and was the weaker of twins. On the fifth day of its life feet were found to be swollen, and soon began to give on palpation a feel of hardness like that of a board. This condition soon spread upward the legs, thighs, hips, shoulders, arms, face, and scalp. The whole b felt as though it were half frozen. The temperature in the rectum w under 35° C. (95° F.). The infant died on the ninth day.

NÆVUS.—There are two forms of pathological disturbance in conn tion with the blood-vessels of the skin which, appearing at birth, constit a disease called *nævus*. Both these forms can appear on the skin of a part of the infant, but its occurrence is especially unfortunate when it located on the face.

The first form is very superficial in its distribution, and is the one wh is usually called "port-wine mark." This form can in a number of ca be destroyed by the use of electricity. The second form, which is deep its distribution, as a rule needs to be treated by the knife or the Paque cautery. Cases of the superficial form of nævus are quite common a vary greatly in degree. A frequent locality is between the eyes at bridge of the nose, and another is on one of the eyelids. Often in th cases the disturbance disappears of itself after a few weeks or months a does not return. In other cases the lesion remains, often increases, a continues, unless treated, through life. In the second form much can done by operative interference. This form also varies greatly in size a in the degree of the telangiectasis. In operating on these cases it sho be remembered that at times the hemorrhage is great, and that the infa are liable to die from exhaustion. The following case (Case 191) was s by me with Dr. Lovett:

An infant four months old was born with a superficial nævus on the forehead. T nævus increased in size, and at four months showed a deep discolored protrusion the siz a half-dollar on the left side of the forehead. Dr. Lovett removed the growth by incis extending into the sound tissues. There was much hemorrhage at the time of the op tion, and after the operation great prostration, apparently from loss of blood. The in was treated with stimulants and the application of heat; it was fed on a carefully arra substitute food for a number of days, and finally recovered. At the end of a year all remained of the original lesion was a very slight scar on the forehead.

DIVISION IX.

DISEASES OF THE SKIN.

LECTURE XXI.

In a previous lecture (Lecture XIII., page 320) I referred to the importance of inspection as a means to be employed in making a diagnosis of diseases in children. The rule that the child should be inspected in every part is especially applicable to the class of cases which I am now about to describe to you.

The lesions of the skin in children differ somewhat from those which occur in adults, and these variations, both in degree and in kind, often make a differential diagnosis more difficult than in adults. Every practitioner has doubtless been struck by the similarity which at times is seen in the cutaneous lesions of the various forms of erythema to those of such diseases as syphilis, scarlet fever, and erysipelas. I have seen in consultation the delicate pink of an abdominal erysipelas in a young infant mistaken so completely for scarlet fever that the precaution of removing the carpet in the room had already been taken. In like manner I have known a slight grade of the efflorescence of scarlet fever to be mistaken for that of erythema neonatorum. I have also seen a harmless papular erythema closely simulating and mistaken for one of the papular efflorescences of syphilis.

Another rule, and one of equal importance, is that no single dermal lesion, whether it be a macule, a papule, a vesicle, or a pustule, makes it justifiable for us to decide that an especial disease is present. We must remember that the same cutaneous lesion may appear in almost any disease, and that it is the combination of dermal lesions and general symptoms which makes up the entire picture of the disease and justifies us in making a diagnosis.

I shall not attempt to speak at length concerning the local diseases of the skin. These diseases come rather within the province of the dermatologist. I wish, however, to show you a few illustrative cases of the more common cutaneous affections which you will meet with in your practice and will be obliged at least to differentiate from the constitutional diseases with dermal lesions which you will have to treat.

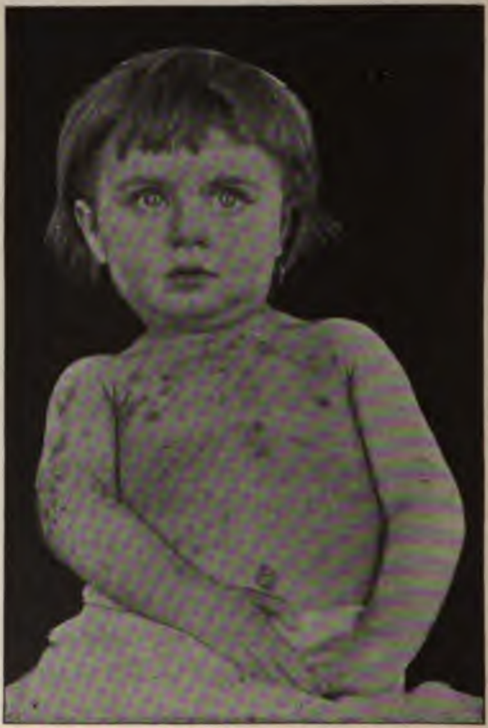
The first case (Case 192) that I have to show you is one which represents the purest type of a primary disease of the skin. It is caused by an especial parasite of the skin, the *Acarus scabiei.*

SCABIES.—This child, two and a half years old, is healthy and well developed.

For the last two weeks it has been very irritable, and its mother has brought it to the hospital to inquire about an efflorescence which has appeared on its skin.

On investigating the lesions we find a number of small papules and a few pustules scattered irregularly over the arms and chest, and one or two small pustules on the soles of the feet. The fingers are not especially affected, but in one or two places at the base of the fingers the efflorescence may be plainly seen. In addition to the papules and pustules there are numerous lesions of the skin caused by scratching. Here on the delicate skin of the abdomen is a minute black line with a vesicle at one end of it. On removing carefully with a needle a little of the fluid in this vesicle and placing it under the microscope, you will see the parasite, which evidently had its habitat in the vesicle. This organism, which I shall not describe more fully, as it is best illustrated in your course on diseases of the skin, is called the *Acarus scabiei*, and is the cause of this special dermal lesion. The black line represents the burrow by which it enters and through which it travels as far as the vesicle, where it lodges and produces irritation, causing first a minute papule, and then a minute vesicle. Finally the vesicle may become pustular.

CASE 192.



Female, 2½ years old, with lesions of the skin caused by the Acarus scabiei.

In contradistinction to the effects of the *Acarus scabiei* on the skin of adults we find in oung children that the parasite may attack the soft skin of the soles of the feet, adult we do not find the lesions on the soles, as in walking the skin has become that locality. In adults efflorescences on the soles of the feet and the palms are, as you know, rather unusual unless they are connected with syphilis or ma.

and young children are usually infected by the *Acarus scabiei* from sleeping in some adult who has scabies. In this case you see that the child's mother shows scabies between her fingers.

MENT.—In the treatment of this disease it is of course very to treat it in the mother as well as in the child. The clothes , of the mother, and of the infant should first be thoroughly order to kill the parasite, and it should be impressed upon the t the treatment must be carried out very carefully, and that all which have come in contact with the skin must be thoroughly

atment of scabies in the child should differ somewhat from that mployed when the disease occurs in the adult, because the skin ner is much more sensitive than that of the latter. The severe hich can properly be used in treating the adult should not be n the treatment of infants and young children.

case I shall adopt the method which I have been in the habit of and which was recommended to me by Dr. Bowen as successfully m in his practice.

eatment consists in an application to the skin of this ointment on 47):

PRESCRIPTION 47.

Metric.	Gramma.	Apothecary.
Peruviani,		℞ Balsami Peruviani,
. . . .	āā 60	Petrolati . . . . āā ℥ii.
		M.

infant as old as this, and for older children, an ointment contain-ulphur could be employed without much danger of irritating the ription 48):

PRESCRIPTION 48.

Metric.	Gramma.		*Apothecary.*
sublimati	7	5	℞ Sulphuris sublimati . . . . . ʒii;
Peruviani,			Balsami Peruviani,
. . . .	āā 30	0	Petrolati . . . . āā ℥i.
			M.

use of either of these ointments the following technique should d. The child is to be first thoroughly washed with warm water The skin is then dried, and the ointment is applied over the , avoiding the head, which is seldom attacked by the parasite.

The face especially might be irritated by the ointment. The ointment is allowed to remain on the child during the night, and in the morning is washed off with warm water and soap. The skin is then thoroughly powdered with the zinc and starch powder which I have already mentioned (Prescription 2, page 130). This treatment is continued for three or four days, and then, if the disease is not entirely cured, it can be repeated for a few days more.

A certain amount of eczema usually follows the treatment, owing to the irritation produced by scratching, which is very difficult to prevent. This eczema should be treated by soothing applications.

PEDICULOSIS.—A parasite whose nidus is on the head appears quite frequently in children as well as in adults. It is especially met with among the poor and ill cared-for. This parasite, the *pediculus capitis*, causes extreme irritation of the skin, which often results in eczema. Although the pediculus itself is in the hair, yet by its irritating action on the scalp of the child it frequently gives rise by reflex influence to patches of eczema grouped about the nose and ears.

TREATMENT.—In treating these cases the hair and scalp should first be saturated with petroleum. This application is allowed to remain on the head for several hours, and later is thoroughly washed off with soap and water. The nits should then be carefully removed by means of a fine comb wet with vinegar. It is usually necessary to repeat the treatment for two or three days.

IMPETIGO CONTAGIOSA.—I have here two children (Cases 193, 194) who have a parasitic disease of the skin called *impetigo contagiosa*. It is a disease which usually occurs in children, but it may be found in adults. It sometimes appears as an epidemic, and in these cases, in all probability, is caused by the same micro-organism as in the isolated cases. It is usually met with among the poorly cared-for, but it may attack the healthy as well as the sick and weak.

The form of the efflorescence is variable. Beginning as small vesicles, the lesions soon spread over a larger area, coalesce, usually form pustules, and later become rapidly covered with a thick yellowish crust. The lesion may occur on any part of the body, but is especially common on the face and hands. The itching is very slight in these cases, and there is no constitutional disturbance caused directly by the parasite. In accordance with the idea that it is of parasitic origin, the prognosis is favorable, and the disease can usually be cured in a week or ten days.

These boys live in a damp dwelling. They both have lesions on their skin which cannot be explained as those of any of the diseases of which I have previously spoken to you which I am about to show you.

The first case (Case 193) is nine years old. He has lesions on the arms and on the base of the nose. They are characterized by some yellowish crusts.

The other boy (Case 194) is eleven years old, and was apparently infected by the former. He presents lesions of the same character as in the first case on the end of his nose and on the corner of his mouth.

TREATMENT.—The treatment of impetigo contagiosa is very simple, and consists in cleanliness, exposure to sunlight, and the application of an ointment such as this one (Prescription 49):

PRESCRIPTION 49.

Metric.	Gramma.		*Apothecary.*	
℞ Acidi borici	3	75	℞ Acidi borici	ʒi;
Adipis	30	00	Adipis	℥i.
M.			M.	

FURUNCULOSIS.—Closely connected with impetigo contagiosa is *furunculosis*, which is supposed to be caused by the same micro-organisms that give rise to impetigo contagiosa, but which affects a different part of the skin, such as the deeper portions of the hair-follicle, in contradistinction to the upper layers of the skin, the part affected by impetigo contagiosa. These micro-organisms are those which are called the "pus organisms," and are usually represented by the staphylococcus pyogenes aureus.

TREATMENT.—The treatment should be with an anti-parasitical ointment or solution preferably containing boracic acid. In many cases in addition to this local treatment some form of constitutional treatment should be employed, as the children are usually in an abnormal condition. The lesions should be bathed every day with this solution (Prescription 50):

PRESCRIPTION 50.

Metric.	Gramma.	*Apothecary.*	
℞ Acidi borici	15	℞ Acidi borici	℥ss;
Aq. destil.	240	Aq. destil.	℥viii.
M.		M.	

After the parts have been thoroughly bathed with this solution an ointment should be spread on linen compresses and applied to the lesions. This ointment should be made in the following way (Prescription 51):

PRESCRIPTION 51.

Metric.	Gramma.		*Apothecary.*	
℞ Acidi borici	3	75	℞ Acidi borici	ʒi;
Petrolati	30	00	Petrolati	℥i.
M.			M.	

MOLLUSCUM CONTAGIOSUM.—Another probably parasitic disease which is rare, but which is more frequent in children than in adults, is molluscum contagiosum. It occurs most commonly on the face, though it may be found on other parts of the body. The lesions consist of small, firm nodules of a whitish color, with a central depression from which matter of a sebaceous consistency may be pressed. The diagnosis is not difficult for one who has once seen the efflorescence, the only condition with which it might possibly be confused being verruca, which, however, does not occur commonly on the face, has no central depression, and does not contain any substance which may be squeezed out.

Treatment.—The treatment of these lesions is to puncture them, squeeze out their contents, and dress them with the following anti-parasitic ointment (Prescription 52):

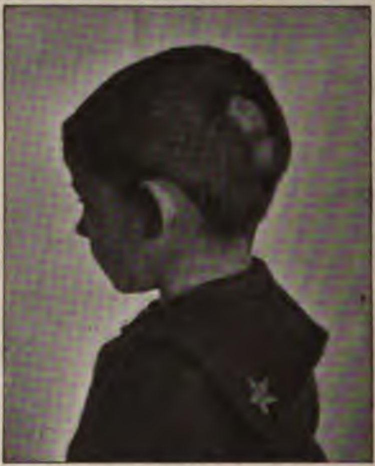
Prescription 52.

	Metric.	Gramma.			Apothecary.	
℞	Acidi borici	3	75	℞	Acidi borici	ʒi;
	Adipis	30	00		Adipis	℥i.
	M.				M.	

TINEA TRICOPHYTINA (Ringworm).—The disease called *tinea tricophytina* occurs clinically in two forms. The first form affects the scalp, and is called *tinea tonsurans.* The other form attacks the non-hairy portions of the body, and is called *tinea circinata.*

This little boy (Case 195) has, as you see, two bald spots on the back of his head. The hair over the rest of his head is thick, and there are no appearances of loss of hair anywhere else on his scalp. The areas of scalp attacked by this disease vary in size. In this special case, however, the spots are about 2.5 cm. (1 inch) in diameter. As a rule, they have a fairly regular circumference. On examining the spots you will see that there are little short hairs on their surface, which evidently have broken off from lack of nutrition. On the edges of the spots this is especially noticeable. If you place one of the hairs under the microscope, you will find a specific organism which has been determined to be the cause of this disease. It is of vegetable origin, and consists of masses of spores composed of threads of mycelium, some long and some short, which are divided into numerous segments.

Case 195.



Tinea tonsurans. Male, 8 years old.

The *disease* itself is called *tinea tricophytina,* and the parasite which causes it is called the *Tricophyton tonsurans.*

Tinea tricophytina has the peculiarity of not appearing on the scalp except in children, but is the same disease that occurs in adults in various localities, as on the face in men, destroying parts of the beard. It may also occur on any part of the body both in children and in adults. Its cause can usually be traced to the same parasitic affection in some other person or some animal.

TREATMENT.—The treatment of this disease should be active, and it is usually necessary to continue it for a long time, especially in cases where the parasite has attacked the head. This ointment (Prescription 53) is a good one to begin the treatment with:

PRESCRIPTION 53.

Metric.	Gramma.		*Apothecary.*	
℞ Acidi salicylici,			℞ Acidi salicylici,	
Sulphuris	āā 3	75	Sulphuris	āā ʒi;
Lanolini	30		Lanolini	℥i.
M.			M.	

It should be applied twice daily, and should be thoroughly rubbed into the bald spots, the skin first having been washed with soap and water.

Where the case proves to be somewhat intractable, still stronger applications can be used, and, if necessary, a certain amount of carbolic acid can be mixed with the ointment, from one-half to one drachm to the ounce of ointment.

The second form of tinea tricophytina, tinea circinata, may at times appear as numerous multiple lesions in different parts of the body, and is easily affected by anti-parasitic applications.

TINEA FAVOSA.—The next case (Case 196) represents a parasitic disease called *favus*. Its favorite seat is the scalp, though it may attack any part of the body. It appears in the form of small, bright yellow, cup-shaped crusts, which upon their removal leave a permanent but superficial cicatrix. These yellow crusts penetrate the hair-follicle and destroy the growth of the hair. When placed under the microscope they are found to consist almost entirely of mycelium and spores of the form called *Achorion schoenleinii*. The crusts often become confluent, forming a large thick covering over an extensive area.

TREATMENT.—The treatment is the application of an ointment to soften and remove the crusts, epilation, and anti-parasitic ointments such as I have already mentioned (Prescription 53).

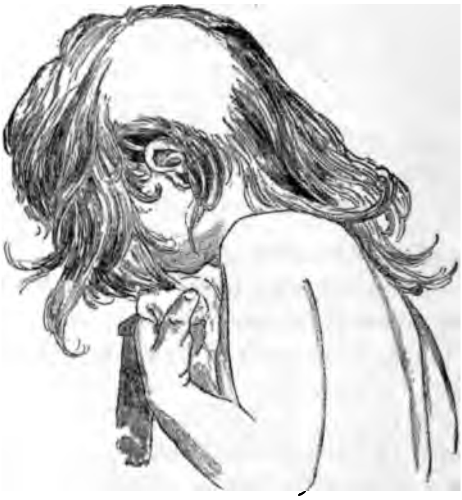
ALOPECIA AREATA.—On comparing the bald spots on this little boy's head with these on the head of this little girl (Case 197) you will notice certain differences.

You see on drawing aside her long hair that an irregular surface of the scalp is entirely free from hair up to where the long hair begins to grow on its edges. The appearance of the skin over this spot is normal.

The nature of the disease has not yet been determined. It must be differentiated from this case (Case 195) of tinea tricophytina which I have just shown you, and, as you see, it has an entirely different appearance, the skin looking sound and healthy, while in the case of tinea there are numerous short hairs, which, as I have already explained to you, are broken off through the action of the parasite.

Alopecia areata is somewhat intractable to treatment and runs a rather long course, but, as a rule, in children can be cured.

CASE 197.



Alopecia areata. Female, 5 years old.

The diagnosis is made by finding a bald spot on the head having the appearance which you see here. The remaining part of the scalp is found to be in a healthy condition and well covered with hair.

TREATMENT.—The treatment is the continual application of stimulating remedies, such as ointments of sulphur and tar (Prescriptions 54, 55).

PRESCRIPTION 54.

Metric.	Gramma.	Apothecary.	
℞ Sulphuris	3.75	℞ Sulphuris	ʒ i;
Petrolati	30.00	Petrolati	℥ i.
M.		M.	

PRESCRIPTION 55.

Metric.	Gramma.	Apothecary.	
℞ Olei cadini	3.75	℞ Olei cadini	ʒ i
Petrolati	30.00	Petrolati	℥ i
M.		M.	

These remedies should be used so as to produce a slight rubefaction, b not inflammation.

PEMPHIGUS NEONATORUM.—In addition to the true pemphig of adults, the epidemic pemphigus infantilis, and the pemphigus which ondary to diseases of a debilitating nature, we at times meet with a form pemphigus which seems to be caused by a parasite of the skin. Blom- rg has reported cases of this kind; one in a girl six days old who had an lorescence of pemphigus beginning on the lower legs and quickly spread- g to the thighs, the abdomen, and the front of the thorax. Later the earm and head were attacked, but only a few bullæ appeared on the ck. The lesions developed quickly on a previously normal skin, and sappeared after a few days, leaving a moist, reddened corium. One of the llæ on the head was 1.2 cm. (½ inch) in diameter. On the right foot one lla covered all the toes and the sole of the foot. The sole of the left foot s covered by three bullæ. Entire recovery took place. No evidence of epidemic was found to account for this case. Three servant-girls in the nily who took care of the child and who washed its clothes were affected from three to six days with the same efflorescence on their hands and ms. Another child and the mother had a few bullæ develop on them. omberg inoculated himself on the forearm with fluid from the bullæ, and the following day he was affected with a similar efflorescence, which sappeared in three days.

This class of cases has not yet been fully accepted by dermatologists, d we must remember that on the delicate skin of infants and young ildren impetigo contagiosa may cause the lesion of pemphigus through the tivity of the parasite and the great vulnerability of the skin.

PEMPHIGUS.—Pemphigus is a disease of a constitutional character, nd is represented by large blebs and bullæ. It occurs at times in infants and children as it does in adults. It is very rare, and I shall not describe it in detail. There is a form of pemphigus, however, which I have met with in infants and children in which bullæ of various sizes appear upon he limbs and trunk, and which is not connected with syphilis. It usually ccurs in poorly-nourished children, and can come not only as a disease of self, but also as one of the sequelæ of debilitating diseases, such as pneu- onia, rheumatism, and others. Where it is secondary to other diseases it presents a condition of malnutrition, and in all probability is not con- cted with the real disease pemphigus. In my experience this class of ses is not especially serious, but merely represents a greater or less degree lack of vitality of the skin.

TREATMENT.—There is no especial local treatment which appears to efit this condition of the skin, but it soon disappears when the general trition of the child has again become normal.

This form of pemphigus, in which the efflorescence is secondary to other cases, is not usually seen upon the soles of the feet or the palms of the ds, and this is of considerable aid in distinguishing the disease from the llous form of syphilis.

Where pemphigus occurs as an epidemic among infants in foundling pitals it is of a more serious nature, and is accompanied by constitu- nal symptoms, represented by fever, sometimes lasting from three to six weeks. In these cases it is usually acute, but it may become chronic, and last, with intervals of recurrence, for many weeks or months. These cases are more apt to be fatal than the other forms. The true epidemic form of *purulent pemphigus*, as it has been called, is almost always fatal, and in cases where it is not secondary to any other disease has a grave prognosis. Many of the reported cases of this epidemic form, as well as of the other forms of pemphigus, may really be only manifestations of the staphylococcus invasion.

DERMATITIS EXFOLIATIVA NEONATORUM (Ritter's Disease).—In the year 1878 Ritter gave the first complete description of the disease *dermatitis exfoliativa neonatorum*. Previous to this date cases of this affection had been reported, but many of them were regarded as some rare or unusual manifestation of pemphigus. Ritter studied and reported the cases which he saw at the Foundling Asylum in Prague from 1868 to 1878. A careful review of Ritter's original observations of these cases has been made by Elliot, to whom I am indebted for what I have to tell you concerning this rare disease. The majority of cases were in male infants, and the mortality was found to be 48.82 per cent.

The disease occurred rarely before the end of the first week, and usually appeared between the second and the fifth week, of life. It was found to vary greatly in the intensity of its symptoms. In some cases a dry scaly condition of the skin preceded the subsequent lesions, which had apparently lasted after the physiological desquamation of the epidermis had taken place.

SYMPTOMS.—The first symptom noticeable in these cases was a diffuse redness, usually over the lower half of the face about the mouth, sometimes, however, beginning in some other portion of the body, and at times being universal from the beginning. This hyperæmia of the skin spread rapidly, and in a few days became universal, the extremities, as a rule, being the last parts affected. The mucous membrane of the mouth and nose was at times affected, and the conjunctivæ usually participated in the hyperæmia. The color of the efflorescence varied from a light to a dark purple-red. As the hyperæmia extended to new surfaces, those which were first affected began to desquamate. This desquamation at times gave no evidence of exudation, the epidermis being simply thickened, and the loosened epithelium separating easily. At times other lesions appeared, such as milia, and sometimes the horny layer of the skin was raised above an intensely reddened base, and large, irregularly-shaped bullæ filled with fluid were formed. After the desquamation had taken place the skin recovered its normal condition, sometimes very rapidly, but it remained for some time rough and irritable. In the cases where there was no exudation a longer time was necessary for the separation and regeneration of the epithelium.

Usually the disease was found to run its course in from seven to ten days. Relapses were sometimes observed ten or twelve days after the first attack, but were always mild.

pical cases the process was unaccompanied by any fever or systemic ces unless some complication existed. The functions were normal, weight of the infant remained stationary or was even at times . The fatal cases resulted either from the intensity of the attack some intercurrent affection or sequela, such as furunculosis. The usually recognized as a local septic infection of the skin, and it em that it should be distinguished from the pemphigus which the early weeks of life.

e myself seen but one case in which it seemed that this diagnosis titis exfoliativa could reasonably be made.

ase (Case 198), a male infant, at the fourth or fifth day of its life presented condition of erythema neonatorum. After a few days this erythema began to slightly, but somewhat later a pronounced dermatitis appeared and ran its course During the course of the disease there were lesions of various kinds represented stules and bullæ, but mostly by an intense erythema. The lesions gradually tense, a profuse desquamation took place, and the skin then presented a normal . During the course of the disease the infant did not show any constitutional and gained somewhat in weight. The parents were healthy, strong people, with nic surroundings.

ll now speak of some of the more simple forms of dermal lesions quently occur in infants.

THEMA.—Erythema plays an important part in the diseases of nd young children. Although it is one of the most common and iagnosticated diseases of the skin which occur in early life, yet at s quite difficult to differentiate it from other diseases, owing to the f its forms. It may be divided into two broad classes: (1) the e form, or *erythema simplex*, which is caused by traumatism and by lrugs, and is also symptomatic of the acute exanthemata; (2) the tory form, *erythema multiforme*, which may affect any part of the either small or large surfaces. It has, however, a predilection for of the hands and of the feet. Its lesions may be represented by or in the process of its evolution these maculæ may develop into apules, vesico-papules, papules, vesicles, and even bullæ. The ary in size. The color varies from bright red to purplish red, metimes very vivid. The delicate texture of the skin of young s more likely to show variations in the color and the form of its an is the fully developed and stronger skin of the adult.

TOMS.—The symptoms of the congestive form are varied, and they company each manifestation of the disease with any especial regu- The slightest local irritation, whether from parasites or trauma of , changes in temperature, reflex irritation from the close connection he digestive organs and the skin, and many other reflex manifesta- y produce the disease.

ythema multiforme there may be pains in the joints simulating sm, malaise, slight fever, nausea, coated tongue, loss of appetite, and a swollen, tender skin. These more marked symptoms are, however, often absent, and the lesions of an erythema multiforme commonly appear on the skin of young subjects without any especial general symptoms accompanying them. It is better in your nursery practice not to endeavor to classify this protean disease under special names which have been handed down from time immemorial in the text-books, and which have no particular significance. These names have been used indefinitely by physicians, and the same form of lesion is sometimes called by one name and sometimes by another.

TREATMENT.—The treatment of all forms of erythema is practically the same. It consists chiefly in the application of a simple powder (Prescription 56) of oxide of zinc and starch, and of a lotion consisting of either lime water or rose water in which calamine and oxide of zinc are suspended (Prescription 57).

PRESCRIPTION 56.

Metric.	Gramma.		*Apothecary.*	
℞ Zinci oxidi	7	5	℞ Zinci oxidi	ʒii;
Amyli tritici	60	0	Amyli tritici	℥ii.
M.			M.	

S.—For external application.

PRESCRIPTION 57.

Metric.	Gramma.		*Apothecary.*	
℞ Zinci oxidi,			℞ Zinci oxidi,	
Calaminæ præparatæ āā	7	5	Calaminæ præparatæ āā	ʒii;
Aquæ calcis	240	0	Aquæ calcis	℥viii.
M.			M.	

S.—For external application.

ERYTHEMA INTERTRIGO.—The form of erythema which is called intertrigo I have already referred to in my lecture on nursery hygiene (Lecture V., page 112, Plate III., A), and I showed you a case (Case 42, page 132) of this disease at the time that I was explaining the proper way to preserve the infant's skin from irritation. I shall, therefore, not speak any more in detail concerning this condition, but shall merely state that it should be classified as belonging to the congestive form of erythema.

In the more severe forms of this disease, where the erythematous condition has become eczematous, and where the skin in the folds of the groins, of the neck, or of the axillæ shows fissures and the moist condition represented by eczema madidans, I have found an application of boracic acid powder quite efficacious.

ERYTHEMA NODOSUM.—Another form of erythema, called *erythema nodosum*, is a disease which is closely allied to erythema multiforme. The general characteristics and symptoms of erythema nodosum can be well learned by studying the case of this child who has been brought to my clinic for examination.

She is a little girl (Case 199), five years old, and until two days ago was perfectly well. At that time she began to have loss of appetite, fever, and malaise, followed by pain in both her legs. Following these general symptoms this efflorescence appeared in various places on her legs. You will notice it above and below the knee, but mostly over the tibia and extending down as far as the ankle. These lesions vary from 1.2 to 2.5 cm. (½ to 1 inch) in diameter, and are of a somewhat irregular elliptical outline. They are of an erythematous type and have a delicate pink color. The skin over these lesions is hot in comparison with the unaffected portions of the skin around them. The lesions are tender on pressure, and their tissues are somewhat indurated, so that the feeling is that of a hard, raised swelling.

The disease is self-limited, but is irregular in its course. It usually disappears in about two weeks. Its cause is not known. The treatment is simply palliative.

URTICARIA (Nettle-Rash, Hives).—The term *urticaria* has been applied to an efflorescence characterized, as a rule, by wheals, which appear suddenly and disappear quickly. It is accompanied by intense itching and burning, and may show itself on any part of the skin, in lesions either small or large in number.

It is commonly caused by irritation of the gastro-enteric tract. The disease may end in two or three days, but usually lasts for some weeks, and may become chronic; it is essentially, however, an acute affection.

If the lesion has been severe there may be slight desquamation, but this is rare. Sometimes there may be only one attack; again there may be relapses, and in some forms and in certain skins it may occur from year to year.

When seeking for the cause of an outbreak of urticaria you must investigate carefully as to whether there has been an error in diet. In children some simple article of food may cause an urticaria to appear, just as in some adults the disease occurs from an idiosyncrasy which prohibits them from eating oysters, lobsters, strawberries, or certain other articles of diet. Again, in some individuals certain drugs, such as chloral, bromide of potash, chlorate of potash, and belladonna, may cause the dermal lesions of urticaria. The wheals of urticaria frequently occur as a symptom in the course of various diseases, such as scabies, or may be caused by the bites of insects.

TREATMENT.—The treatment should be directed first to the removal of the cause of the dermal irritation. When this cause has been removed the dermal lesions will, as a rule, disappear, unless still further irritation has been produced by scratching the lesion or by its being too severely treated by the physician.

The diet should be milk for a time, and experiments should be made with different articles of food to see which one may cause this especial form of irritation. The bowels should be carefully regulated. The local applications consist in remedies to relieve the itching and burning, in the wearing of unirritating clothing and soft linen next the skin, and in a powder of starch and zinc, made as I have already described to you (Prescription 57), frequently applied to the lesions at intervals during the day. Where the itching is extreme, anti-pruritic lotions and ointments should be applied, such as the following (Prescriptions 58, 59, page 468):

PRESCRIPTION 58.

Metric.	Gramma.		*Apothecary.*	
℞ Pulv. calaminæ	7	5	℞ Pulv. calaminæ	ʒii;
Aq. calcis	240	0	Aq. calcis	℥viii;
Acidi carbolici	1	87	Acidi carbolici	ʒss.
M.			M.	

When this lotion is not sufficient to allay the irritation and where the burning is extreme, this ointment (Prescription 59) can be used:

PRESCRIPTION 59.

Metric.	Gramma.		*Apothecary.*	
℞ Menthol	0	6	℞ Menthol	gr. x;
Adipis	30	0	Adipis	℥i.
M.			M.	

I have here a little boy (Case 200), sixteen months old, who has been brought to the hospital for advice concerning these lesions on his skin. The mother gives the following account of the case.

A woman who had been taking care of him, and beside whom he had slept at night, was attacked with facial erysipelas of a rather severe type. The mother was exceedingly worried at this occurrence, and consulted her physician as to the probability of her infant's having contracted erysipelas. She was assured by the physician that it would be unlikely for infection to take place under these circumstances.

This was two days ago, and to-day she says that early this morning the infant was found to have considerable fever, to be vomiting, to feel dull, and to seem quite ill. While holding the infant in her lap she noticed that there was a red appearance of the skin covering its right knee, and another member of the household, who considered that she had a great knowledge of diseases in children, announced to the mother that the infant had erysipelas: the mother at once supposed that it had contracted it from the woman who had facial erysipelas.

On examining the skin you will see that the knee and the upper part of the lower leg are swollen and of a vivid red color. On touching it we find that it is not painful, but that the skin is hot, and that there is considerable swelling of the tissues. The infant's temperature is 40° C. (104° F.), its pulse 150, and it looks as though it were suffering from some grave constitutional disease. The color of the efflorescence is identical with that which we at times see in cases of erysipelas, and this fact, in connection with the constitutional disturbance, would make the mother's supposition that her infant had an attack of erysipelas a reasonable one.

I have already impressed upon you the rule that we should examine the entire skin before making a diagnosis of any special disease connected with it. I shall, therefore, although it is highly probable that this is a case of erysipelas, investigate the case still further.

Now that its clothes are removed you see that there is no other dermal lesion on the infant's front, but on looking at its back you will see a number of lesions, some papular, others papulo-vesicular, and here, just below the right scapula, you see a wheal. The infant also shows evidence of irritation from the way in which it endeavors to scratch. These lesions on the back are evidently not those of infantile erysipelas, and on looking again at the original source of disturbance you will notice that instead of the diffuse redness so closely simulating erysipelas, which you saw a few minutes ago, there is now an efflorescence gradually fading away and becoming lighter in color.

This change in the appearance of the efflorescence, in connection with the very evident lesions of urticaria on the infant's back, leads me to defer making a diagnosis until I have questioned the mother still further concerning the infant.

She now tells me that yesterday the infant had been taken care of by a friend, who allowed it to eat some unusual articles of food. We can, therefore, account for the vomiting, loss of appetite, malaise, and fever by a disturbance of the digestive organs.

I can now readily make the correct diagnosis, which is very evident, and which would have been impossible if we had only seen the efflorescence as it occurred on the knee and at the time when we first saw it. It is a typical case of one of the more severe forms of urticaria.

The next case (Case 201) is a little girl, six years old. There is no history of constitutional disease in either of her parents. The mother states that she has had no miscarriages. The child is said to have been a healthy infant, to have had no diseases, and to have been well until six months ago. She then began to complain of frontal headache and to be slightly feverish at night. Her appetite grew poor, and she lost in strength and weight. She has had no cough. She is slightly anæmic and decidedly nervous. She sleeps well, but the bowels are constipated. Nothing unusual has been noticed about the urine. She complains of palpitation and dyspnœa on exertion. There have been no articular or muscular pains. A few weeks ago an efflorescence attended with much itching appeared first on her legs and then on her back and face. The soles of her feet and the palms of her hands were not affected. The efflorescence consisted of macules, at times wheals, evanescent from day to day, and, as you see, irregular in their distribution. At times the lesions have been maculo-papules in certain areas, and also papules.

An examination of the chest shows the lungs to be normal. The cardiac area of dulness is normal. There are no continuous cardiac murmurs. There is slight irregularity of the cardiac rhythm, and a sharp ring to the cardiac sounds, especially the second pulmonic sound. At times also there is a soft evanescent murmur heard over the base of the heart.

On closer examination of the efflorescence we find on drawing the finger gently over it that the pink color disappears, showing that the macules are caused by a congestion of the blood-vessels supplying these areas of the skin. We therefore are not dealing with a constitutional condition such as purpura, which would have resulted in a rupture of these vessels, and which also would have been free from itching and darker in color.

The mother naturally asks, what is this disease which so disfigures her child's appearance. What shall we tell her? In the first place, I have inquired about the child's diet, and have found that it has not been a nutritious one. What, however, especially struck me was that the child has had for the past year a diet consisting largely of tea. We can at once, then, account for her general condition on the supposition that she is an inveterate little tea-drinker. Her failure in general health, her headache, nervousness, and occasional cardiac murmurs, all correspond to the history of tea-poisoning.

We can now with these facts obtained from the general history of the case diagnosticate the efflorescence which plays so great a *rôle* in the child's case, for it is the rash for which the mother has brought her for treatment. The general appearance and description of the case permit us to eliminate in our diagnosis the various efflorescences occurring in the course of scarlet fever, measles, and varicella. The absence of hemorrhage, as I have already stated, precludes purpura. The absence of heat, of pain, of swelling, and of induration of the subcutaneous tissues allows us to eliminate erythema nodosum. The evanescence of the macules and the great irritation enable us to state that we have not a syphilitic erythema to deal with, for, as a rule, the macules of syphilis do not itch. The appearance and description of the lesions are not those of eczema. In a word, you have before you a case of urticaria.

As to the cause of the urticaria, we must remember that certain drugs, which I have already referred to, may produce appearances of this kind on the skin, and the close reflex connection between the delicate terminal filaments of the nerves of the stomach and the skin may cause a great many disturbances, among which are irritation of the gastric membrane and a resulting indigestion.

In addition to tea and improper food, I find that the child has had quite large doses of tincture of chloride of iron given to her. This preparation of iron is a valuable one, and was given for the child's anæmia, but in young children it often is of itself a cause of gastric irritation such as is represented in this child. It was, therefore, especially as the child was constipated, not indicated in her case.

In this class of cases I think that it is best not to give iron at firs
stomach to recover itself by regulating the diet. I shall treat the child
slightly alkaline with lime water, and with thin soups and bread one
exclude from her diet tea, pastry, and fried foods of all kinds. Later
to have a more laxative and less irritating form of iron, such as this (Pre
391).

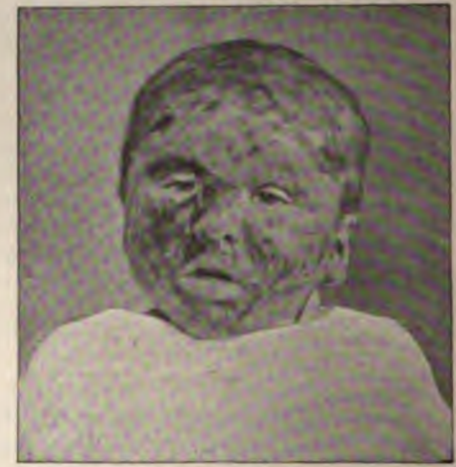
Under this treatment, combined with freedom from excitement, bat
fresh air, I can, from my previous experience with such cases, safely pr
that the child will improve, become strong and rosy, and soon be relieved
able symptoms.

ECZEMA.—Eczema is a disease of the skin which plays
rôle in infancy and early childhood than in any other period
one of the lesions of the skin which should be placed in
dermatologist. It is so difficult to cure that it must alw
upon as a grave disease. You should, therefore, even in
insignificant forms of eczema, be careful not to give a favor
until you have treated the disease for some weeks, for at an
extend to new areas of the skin. I shall not attempt to giv
very brief description of it.

It is essentially a form of dermatitis, and we may find th
appearing in cases which have been exposed to certain veg
such as the rhus toxicodendron, to various artificial irritants,
of temperature.

Before speaking of the general treatment of these cases
in the hospital, I shall show you a few of the cases that have
care.

Case 202.



Eczema capitis.

Here is an infant (Case 202) in whom the lesions on the skin are co
and face.

These lesions consist of papules, pustules, crusts, some excoriated p
scratching, and a thick rather œdematous condition of the skin, especiall
nose, and eyes. The hair has been cut off, and you see various lesions on

parts of the scalp you will notice a reddened moist condition, which represents what is d eczema rubrum. This is the same case as the one (Case 48) that I showed you previous lecture (Lecture V., page 143) as representing bow-legs. He has returned to ospital with a recurrent eczema of the face and head.

The treatment of this case is as follows. The crusts and the thickened tissue of the face scalp will first be softened by means of a poultice. After the larger crusts have been ved, the mask, which I have already shown you (page 143), will be applied to the face scalp. The inner surface of this mask is thickly spread with this ointment (Prescrip-60):

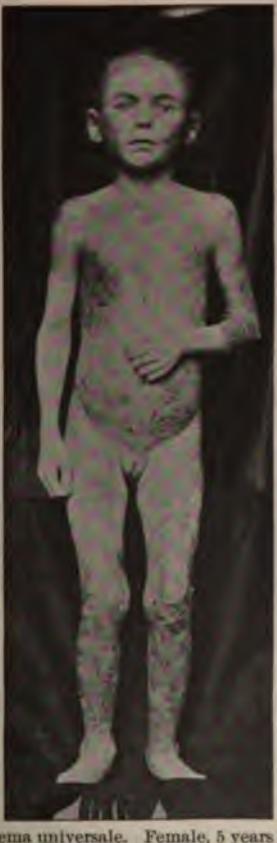
PRESCRIPTION 60.

Metric.	Gramma.		*Apothecary.*	
nguenti zinci oxidi,		℞	Unguenti zinci oxidi,	
anolini . . . . āā	30 | 00		Lanolini	āā ℥i.
I.			M.	

ECZEMA UNIVERSALE.—The next case (Case 203) which I have to

CASE 203.

I.



Eczema universale. Female, 5 years old.
I. Before treatment.

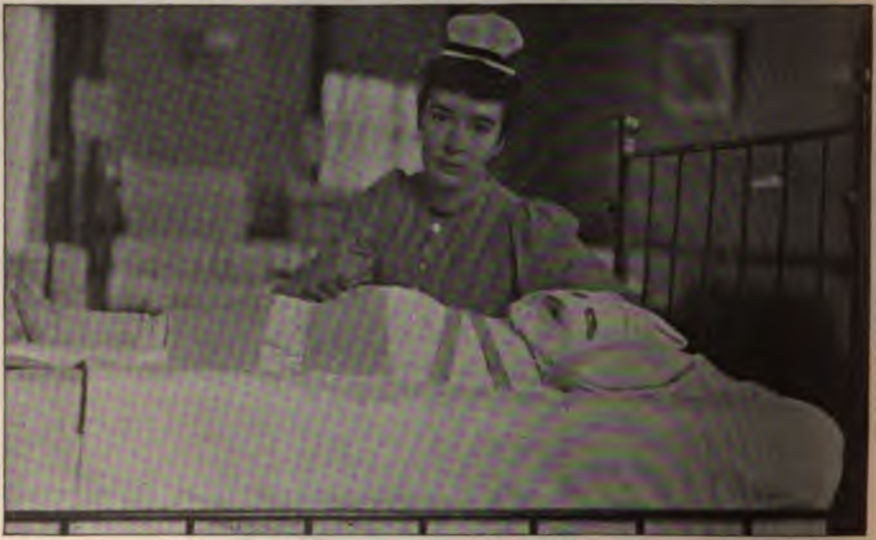
show you is one that is being treated for an eczema which has attacked the face, head, trunk, and extremities of a little girl five years old.

You see on examining her naked that almost every form of eczema is represented in some part of her skin. Here on the right lower arm and the upper right leg we have a form of eczema rubrum; that is, an intensely reddened and moist surface. On the left upper leg are numerous papules, representing the papular form of eczema. On the backs of the hands and upper surfaces of the feet are some spots, which represent the macular form of eczema. On the trunk, lower legs, upper arms, face, and scalp is a collection of thick crusts, representing the form which is called eczema impetiginosum. There is such intense itching connected with this form of eczema that the child is continually scratching its skin and making the disease worse. You must remember this fact, because scratching for a few minutes may retard the recovery of an eczema for many weeks, and therefore it is of the utmost importance for the success of the treatment of a case of this kind, as well as of any of the milder and more local forms of eczema, absolutely to prevent the child from scratching. This can in such cases as are here represented be accomplished only by the complete control of the child's movements.

We should first endeavor to allay the itching by means of applications to the whole skin, and secondly to bandage the child in such a manner as to make any attempt to scratch impossible. I will have this child while you are here in the ward treated by the method by which we are accustomed to control and almost invariably cure this disease.

CASE 203.

II.



Eczema universale. II. Treated by complete rest.

The child is covered from head to foot with soft cotton cloth compresses thickly sprea with the ointment which I have just mentioned (Prescription 60).

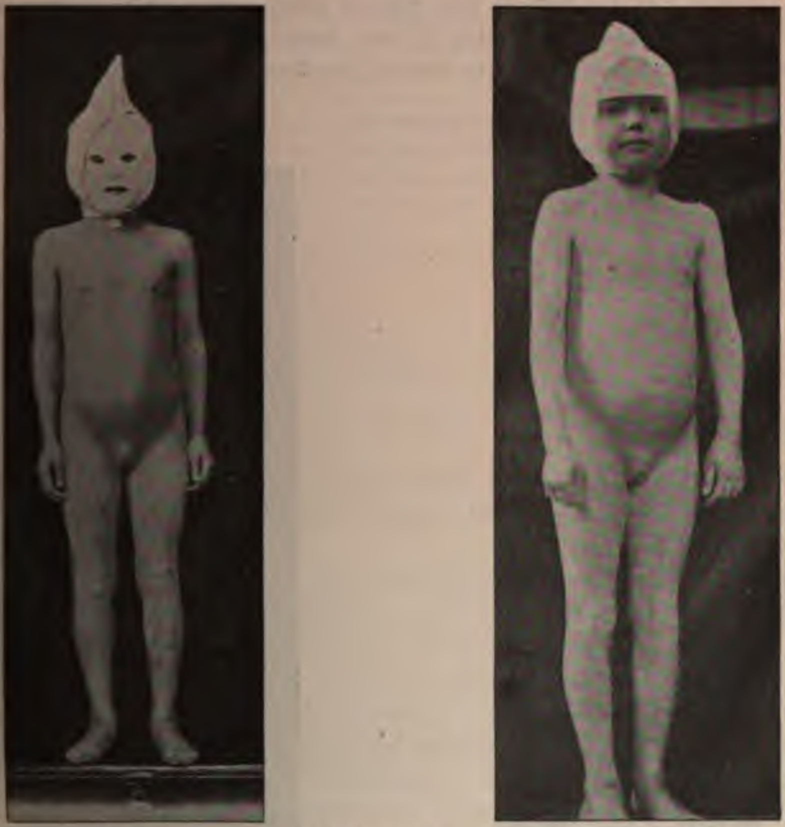
It is then placed in this position on its back in bed, and broad straps are drawn acros its legs, abdomen, chest, and shoulders, thus completely binding its arms to its sides an keeping the legs in extension. On either side of the head are placed soft, heavily padde sand-bags, which prevent it from moving its head from side to side and thus by rubbing ir tating the eczema of the face.

It is necessary to have a nurse in continual attendance, in order to soothe the child, an by amusing it in various ways induce it to forget what at first may be a rather uncomfortab position. This feeling of discomfort usually soon passes away.

This is not a cruel form of treatment. The irritation is soon relieved when the child kept quiet and prevented from scratching. If necessary, in the early hours of the treatme some drugs of a soothing nature may be given to prevent an undue nervous condition the patient. The nurse should be instructed to be very gentle with it, and continually to divert its mind from its skin. Under this treatment in a few days the eczematous condition of the skin will improve and the itching will diminish.

(Subsequent history of the case.) You remember the case (Case 203) of universal eczema which I showed you at a previous lecture being treated in bed. It had improved so much within two weeks that it was allowed to be out of bed and dressed, and to have the ointment applied merely on its face and head (III.).

Case 203.


III. IV.

Eczema universale. III. Three weeks after beginning of treatment. IV. Four weeks after beginning of treatment.

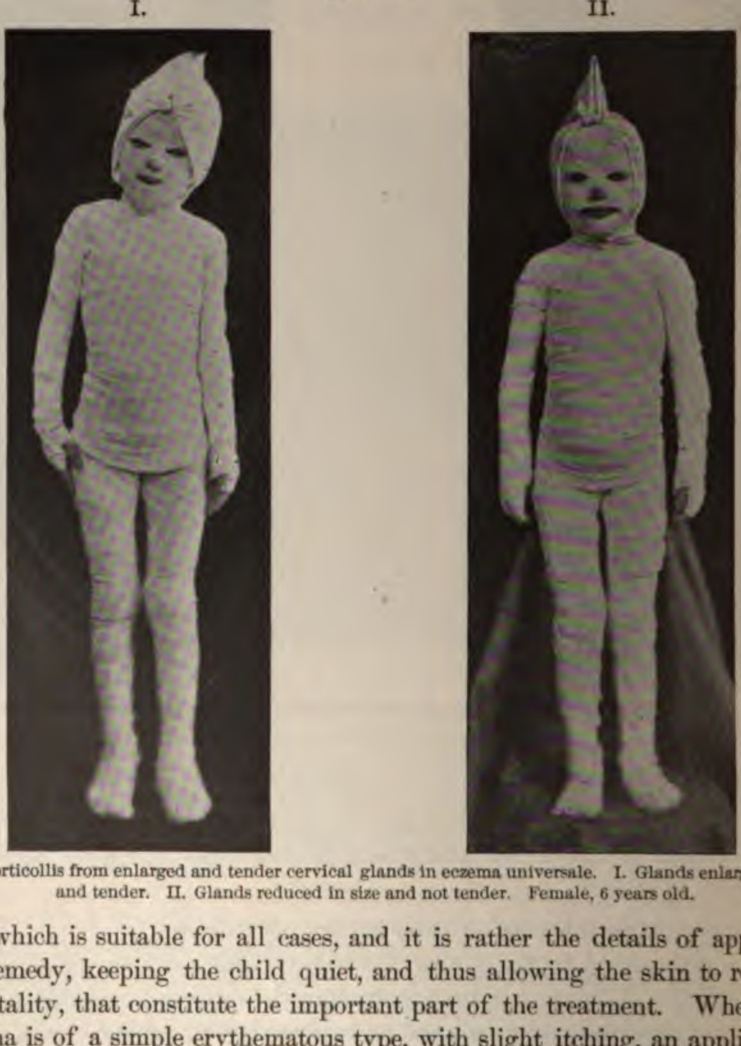
You see that the skin of the trunk and extremities is almost entirely well. The face also is in a much improved condition, and during most of the day she is allowed to have the face uncovered and the ointment and bandage applied to her head only (IV.). I hope in a few weeks to be able to omit entirely the application of the mask to the face and of the bandages to the head.

The eczema in this class of cases is very apt to recur.

It is often asked by the mother and nurse whether the eczema of infants is contagious. I have seen instances where the nurse who was taking care of a case of eczema in an infant had an eczema develop on her hands. This was apparently caused by the nurse having washed the infant's napkins. The hands of the nurse were cured by local treatment, and by using rubber gloves in washing the napkins she did not again contract the eczema. Cases of this kind give rise to the idea that eczema is contagious, but the probability is that they are simply cases of artificial dermatitis caused by irritating substances of various kinds, and that there is no especial germ which causes eczema. We can, therefore, say that the disease is not contagious, and that simple cleanliness and protection of the hands by means of rubber gloves are all that is necessary to prevent the disease being contracted.

I might mention that in this class of cases of universal eczema other applications besides that which I have mentioned may often be useful, though in my experience and in that of Dr. Bowen there is no one application which is suitable for all cases, and it is rather the details of applyin
the remedy, keeping the child quiet, and thus allowing the skin to recove
its vitality, that constitute the important part of the treatment. Where th
eczema is of a simple erythematous type, with slight itching, an applicatio

Case 204.



Torticollis from enlarged and tender cervical glands in eczema universale. I. Glands enlarged and tender. II. Glands reduced in size and not tender. Female, 6 years old.

of some powder such as this one (Prescription 57, page 466) is often useful,—the child being placed between two sheets and thoroughly dusted with the powder, while a nurse is in constant attendance to prevent scratching.

There is one interesting form or rather complication of universal eczema a case of which I happen to have here in the wards to-day to show you.

This little girl (Case 204) came to the hospital to be treated for torticollis. The head was drawn to the left side as she now shows you (I.), and she could not straighten it. This condition had lasted for many months.

On examining the child I found that she had the usual universal eczema of a chronic type affecting the head, face, and extremities. On examining the neck I found a number of enlarged tender glands. These enlarged glands were evidently caused by reflex irritation from the eczema, and were the cause of the torticollis.

She was treated with the ointment which I have just described to you (Prescription 60, page 471), and the usual bandage and mask, and to-day, although the eczema is not yet cured, the irritation in connection with it has been so much lessened that the glands of the neck have gradually subsided and have now disappeared, and the child, as you see, is able to hold her head straight (II.).

I expect the child to receive still further benefit from the treatment, and that she will be discharged from the hospital cured.

In addition to the forms of eczema of which I have already spoken, you will meet with many instances of a local eczema which has been produced by some irritation either at or near the place affected, or perhaps in some entirely different part of the body. This is usually called *reflex eczema*. An example of this form of eczema is where the irritation is on the scalp, such as occurs from pediculi, and develops a local reflex eczema on the back of the neck.

PSORIASIS.—This little girl whom I shall now show you has certain lesions on her back which it will interest you to examine. These lesions are characteristic of the disease called *psoriasis*. Nothing is known of the real cause of psoriasis. So far as we can ascertain, it is not dependent on any micro-organism. When the disease is well developed the diagnosis is very simple, and its lesions correspond, as a rule, to those which are commonly met with in the adult. It begins with small papules, which almost immediately become covered with scales. These scales have a pearly white color, and on removing them we find a bleeding surface, showing that they are more closely connected with the corium than is the case in other diseases where desquamation takes place, such as dermatitis or scarlet fever.

The efflorescence of psoriasis is general, and is, as a rule, marked on the elbows and knees, for in these places the lesions coalesce and the scales are especially thick.

I have noticed in the psoriasis of children that the type of the disease is often so mild that we can scarcely believe we are dealing with the same affection that we are accustomed to see in the adult. In some cases a few lesions scattered here and there, especially on the back over the scapulæ, will be all that represent the disease, and will be easily cured, even disappearing of themselves in a few months. Besides affecting the trunk and extremities, the efflorescence may occur on the scalp, especially along the edge of the hair on the forehead, but the disease is not very common on the face. Psoriasis is apt to recur even at intervals of years, so that we cannot say that it can be absolutely cured, though at times it may disappear under treatment and never return.

This child (Case 205) has had the disease once previously, and returns to the hospital for additional treatment.

The lesions, as you see, vary in size, and in this case are rather small, being only about 5 cm. (¼ inch) in diameter. Many of them are still smaller. You see what an almost regular outline they have, and how in the centres of many of them are the characteristic small, pearly white scales which at once enable us to diagnosticate the disease. Where these scales are not present, as sometimes occurs in the early stages of the disease, it is much more difficult to make the diagnosis.

The disease is not accompanied by any constitutional symptoms, and, as a rule, there are no local symptoms, such as pain and heat. On palpation you find the spots to be accompanied by more or less induration of the surrounding tissue.

TREATMENT.—The treatment of psoriasis in children should be milder in its form than that which you would employ in treating the adult. In this case I shall have this ointment (Prescription 61) of chrysarobin applied to the lesions in the evening and washed off with soap and water in the morning, there being no treatment during the day.

PRESCRIPTION 61.

Metric.	Gramma.	*Apothecary.*	
℞ Chrysarobini	0 | 60	℞ Chrysarobini	gr. x;
Petrolati	30 | 00	Petrolati	℥i.
M.		M.	

This ointment stains the skin, but not permanently. It should never be applied to the face or the scalp, and should be used with great care, as it causes on some skins considerable irritation, and at times a severe dermatitis. With ordinary caution, however, this need not occur.

In intractable cases where this milder form of ointment is not efficacious, the strength may be increased to 1 or 1.5 grammes (15 or 20 grains) to the ounce.

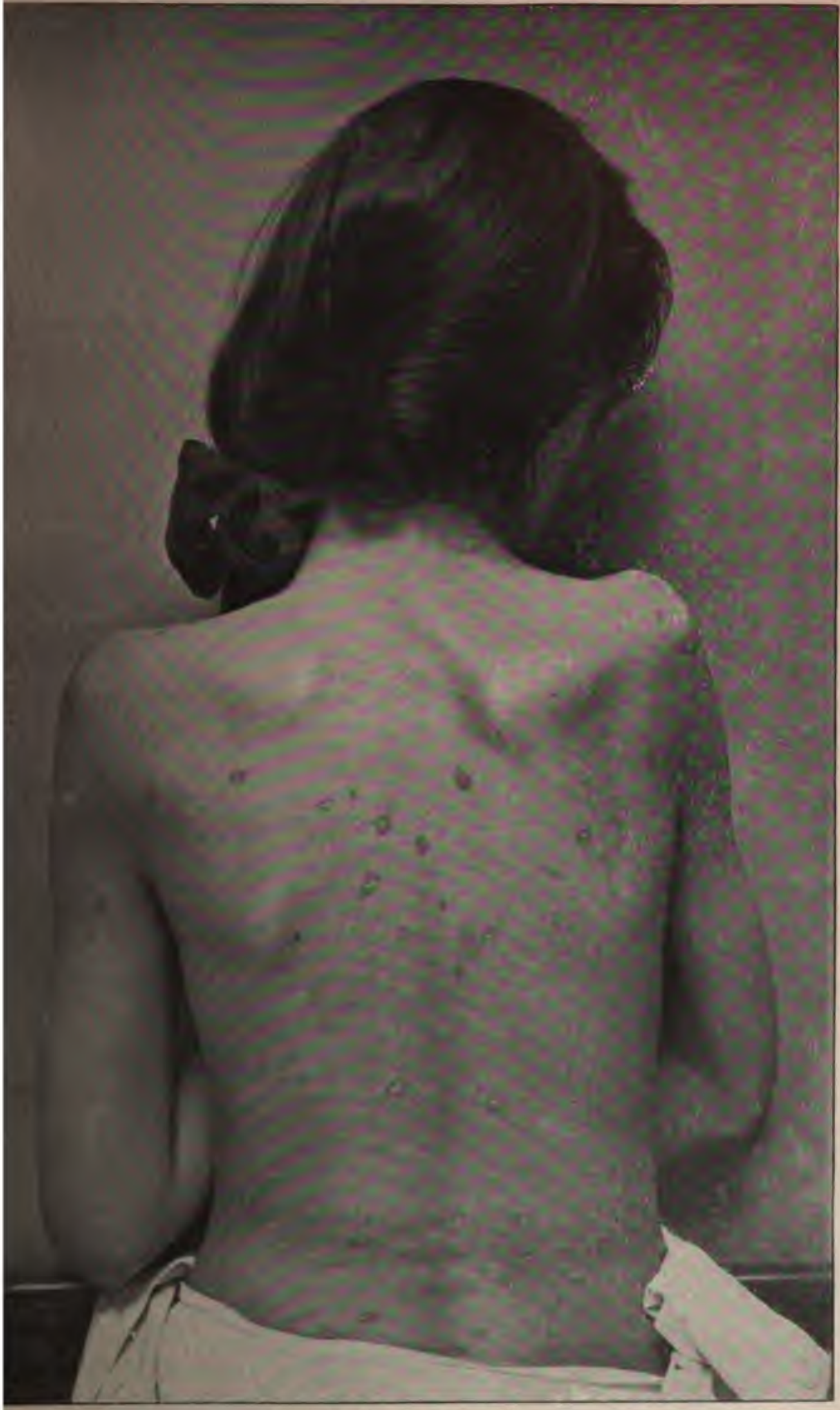
You should remember that chrysarobin stains the clothes black indelibly, so that old sheets and night apparel should be used while the treatment is being carried out.

In place of this ointment you can use on especially irritable skins, or on the face and scalp, this preparation (Prescription 62) of sulphur and tar:

PRESCRIPTION 62.

Metric.	Gramma.	*Apothecary.*	
℞ Sulphuris	3 | 75	℞ Sulphuris	ʒi;
Olei cadini	1 | 87	Olei cadini	ʒss;
Adipis	30 | 00	Adipis	℥i.
M.		M.	

Case 205.

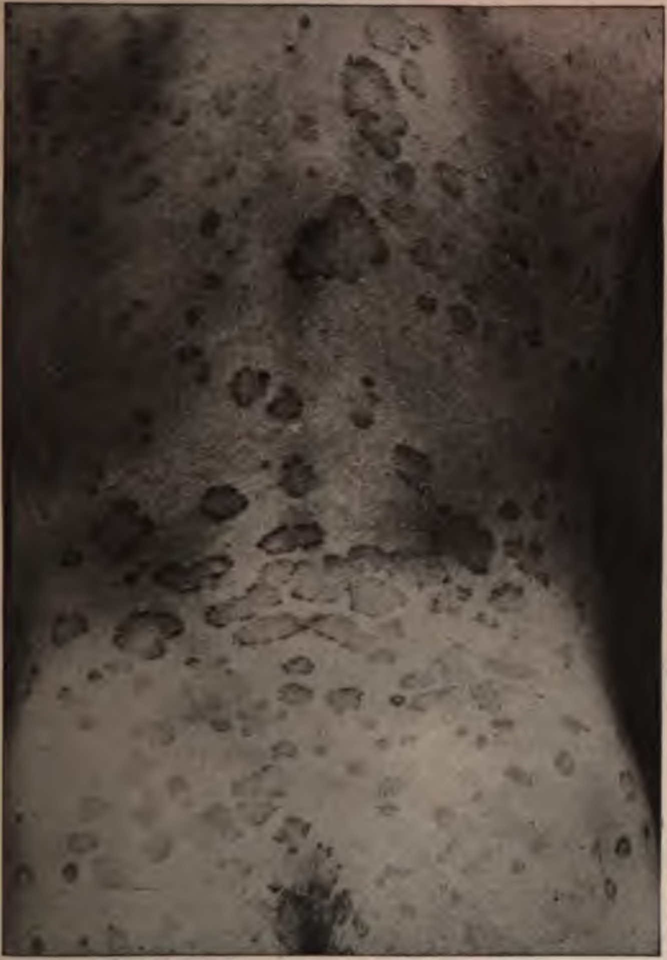


Psoriasis Female, 9 years old.

ave here another case (Case 206) of a boy now seventeen years old, who first came reatment for psoriasis when he was seven years old.

der the usual treatment the efflorescence disappeared and the child was apparently

CASE 206.



Recurrent psoriasis. Male, 17 years old.

The lesions, however, have reappeared from time to time, and come and go without e to treatment. The lesions which you see on his back are much larger than those ou saw on the girl (Case 205). They vary from 2.5 cm. to 5 cm. (1 to 2 inches) in r, and have a thick, irregular outline.

URIGO.—Prurigo occurs in two forms in infants and children,—*rigo mitis infantilis* and (2) *prurigo ferox.*

PRURIGO MITIS INFANTILIS.—Prurigo mitis infantilis occurs in two or three months old, and may last for some years.

MPTOMS.—It begins with little nodular infiltrations, especially marked anterior surface of the extremities, and is accompanied by great itching. It may appear on the face. It does not lead to an infiltration of the skin or to the formation of pus.

TREATMENT.—The treatment consists in remedies to relieve the itching and allay the eczema with which it is usually complicated.

It is closely allied to papular erythema, but is more chronic and has a greater tendency to recur. It is very rare in America.

(2) PRURIGO FEROX.—Instead of this mild form a more severe type of prurigo occurs at times. This latter form is far more serious in its symptoms and in its prognosis, and may continue through life. The disease, which is characterized by the same dermal lesion as that just described, is progressive from the beginning; it usually starts on the legs, and the skin becomes thicker as it descends. The efflorescence is accompanied by enlarged glands, especially in the inguinal region.

The disease is rare in America, but is common in Germany.

Its etiology is very obscure, and it is a most intractable chronic affection.

TREATMENT.—The treatment is palliative.

For the extreme itching caused by the papules an application of this ointment (Prescription 63) may be used:

PRESCRIPTION 63.

Metric.	Gramma.		*Apothecary.*	
℞ Unguenti diachyli,			℞ Unguenti diachyli,	
Petrolei . . . . āā	30	00	Petrolei	āā ℥i.
M.			M.	

S.—To be applied on flannel three times a day for ten minutes, and to be followed by the application of this ointment (Prescription 64):

PRESCRIPTION 64.

Metric.	Gramma.		*Apothecary.*	
℞ Unguenti diachyli,			℞ Unguenti diachyli,	
Petrolati . . . . āā	30	00	Petrolati	āā ℥i.
M.			M.	

If there is much infiltration, sapo viridis should be applied at night and washed off the next morning. It must, however, be used with caution, as it is very irritating.

HERPES ZOSTER.—I have here two cases (Cases 207, 208) to show you,—a boy and a girl. They represent the disease called *herpes zoster*. This disease is one which affects both children and adults. I shall, therefore, not dwell especially upon it, but shall merely give you the main symptoms and the characteristic appearances of the skin, which will enable you to diagnosticate it.

SYMPTOMS.—The general symptoms of herpes zoster are fever, loss of appetite, and pain in some part of the head, trunk, or extremities. The pain is always located in the course of certain nerves. In this little girl (Case 207) it is in the nerves which supply the skin of the upper part of he back, the upper part of the axilla, and the upper part of the chest in ront.

One of the characteristics of the efflorescence is that, as a rule, it is nilateral. It is extremely rare for the affection to e bilateral and to extend around the body. Cases f this kind, however, have occurred, and do not seem o be any more severe, except that larger surfaces re affected than where the affection is unilateral. The haracter of the efflorescence is essentially vesicular, nd it is to be differentiated from varicella, which might e accompanied by the same general symptoms and is lso essentially a vesicular disease. As I shall presently xplain to you, the efflorescence of varicella is general, s not limited to any special distribution of the nerves, or is it painful, while the efflorescence which we are xamining here is, as you see, limited to the distribution of a special set of nerves,—in this case the brachial plexus. The vesicles become somewhat pustular, and soon crusts are formed. In this case, which has lasted three days, there are still a few vesicles to be seen, but a large part of the efflorescence is represented by crusts.

CASE 207.

Herpes zoster of right upper chest. Female, 4 years old.

The disease runs a definite course of about fourteen days, and from the beginning is accompanied by considerable pain, though according to my observations the pain is not so severe in children as in adults, nor is the itching so annoying.

Herpes zoster, so far as we can determine, is not caused by a micro-organism, but is a constitutional disease closely connected with the nerves.

The next case (Case 208, page 480), a boy, has the same disease, but it affects a different set of nerves.

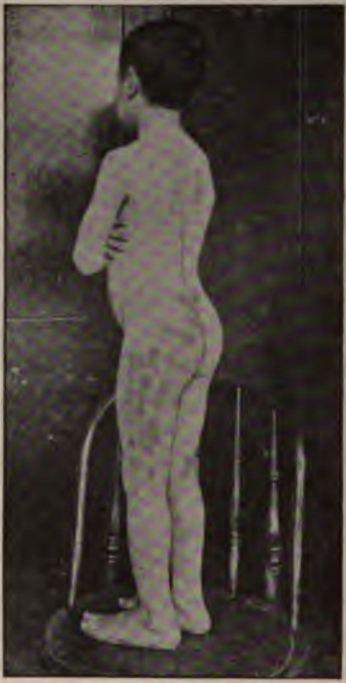
In this case the efflorescence starts at the sacrum, while in the case of the little girl it started over the cervical region. Beginning at the sacrum, it follows the course of the nerves, over the left buttock and down the left leg as far as the knee. The various lesions are the same as I have just described in the previous case.

You see, then, the perfect similarity in the character of the lesions and in the distribution so far as following a particular set of nerves is concerned.

DIAGNOSIS.—The diagnosis of this disease is very easily made from the general symptoms of pain, fever, and malaise, in combination with the characteristic efflorescence, and we at once know with what disease we are dealing, for no other affection of the skin has so definite a distribution.

TREATMENT.—The treatment is simply palliative. What I am accustomed to do is to regulate carefully the child's diet, as I would in any disease with general constitutional symptoms, and to endeavor by the application of lotions to allay the pain. The treatment which I shall adopt in this case is to powder the lesions thickly with some simple powder (Prescription 2, page 130).

CASE 208.



Herpes zoster of left leg. Male, 6 years old.

PITYRIASIS.—*Pityriasis* is a term that is now, like the word lichen, seldom used without an accompanying adjective. There are two recognized forms of the affection.

(1) PITYRIASIS RUBRA is a rare disease in children, characterized by hyperæmia and fine scales affecting, as a rule, the whole cutaneous surface. It may be attended with great constitutional disturbance and lead to death. Its duration is always uncertain.

(2) PITYRIASIS MACULATA ET CIRCINATA, or PITYRIASIS ROSEA, affects children as well as adults. It appears in the form of small patches of scales scattered over the trunk, legs, and arms. These patches either spread peripherally or unite to form larger patches while the centre undergoes involution: we thus see a reddish scaling border and a characteristic yellowish centre. There may or may not be great pruritus accompanying it. In Vienna this affection is still regarded as a form of ringworm, a position that cannot, however, be maintained. Its etiology is obscure. It gets well spontaneously in from two to ten weeks, and is best treated by mild, soothing, and antiparasitic applications.

VERRUCÆ (Warts).—Warts are circumscribed outgrowths of the e of the skin with an accompanying increase in the thickness of the mic layers. They are common in children, especially on the hands, ie old view that they are contagious and auto-inoculable has gained adherents of late. They are of various aspects and shapes, and may ıted, as a rule, locally with success, although some are quite obstinate. ıost efficacious method of treatment is painting each with a solution cylic acid in flexible collodion (Prescription 65).

PRESCRIPTION 65.

Metric.	Gramma.		*Apothecary.*	
i salicylici	3	75	℞ Acidi salicylici	ʒi;
›dii	30	00	Collodii	℥i.
			M.	

is is applied with a camel's-hair brush twice a day for three days. t is soaked off by prolonged bathing in warm water, with the addition aice soap if there is no inflammation. This will usually remove a ı of the wart, and the process should be repeated as long as any of ›wth is left.

e treatment with salicylic acid is not always successful, and recourse ıen be had to glacial acetic acid, or to some other caustic, carefully l; or the growth may be excised.

ENTIGO (Freckles).—Freckles are small aggregations of pigment ted in the skin, and are commonly seen in children of ten years and d, especially in those of light complexion. They are usually situated face and hands, but may occur on the covered portions of the body, a ıat led Hebra to regard them as not due to the action of the sun. can be no doubt, however, that the sun is the chief agent in their ction. Their removal is often difficult and requires the use of strong ts, such as corrosive sublimate. It is rarely advisable to attempt emoval in young children.

ELANODERMA LENTICULARIS PROGRESSIVA (Kaposi's Disease) is a are disorder, and is seldom met with in this country. In this affection of pigment like freckles appear on the uncovered parts of the body inally extending more or less over the whole cutaneous surface. The nt-spots are the first lesions seen, but later an atrophy of the skin and rmation of small angiomata dotted over the surface take place, giving ild an extraordinary appearance. The disease is usually found in more ne child in the same family, and its etiology is very obscure. Malig- umors with a fatal ending usually result from this affection.

CHEN.—Many of the affections that were formerly included under ad of lichen are now considered by most authorities to belong in other , notably in that of eczema. A diagnosis of lichen is never made by can dermatologists, but *lichen planus* is a well-marked skin disorder retains a place of its own. It rarely occurs in children, but when present it follows about the same course as in adults. It is characterized by firm papules of an irregular shape and glistening appearance, of a peculiar reddish-blue or violet color, with usually a slight depression in the centre. The individual papules may coalesce, so as to form patches of greater or less extent, covered with fine scales. It is often accompanied by great itching and discomfort. It attacks all parts of the body, showing a predilection, however, for the flexor surfaces of the arms and legs. It may last for many months, and in the most favorable cases does not disappear for several weeks. The general health is not usually affected, except by the exhaustion that may be caused by intense itching. It may be confounded with a papular syphilide, which it often closely simulates, and sometimes it may be mistaken for an eczema. Arsenic is of value in chronic cases, and antiparasitic lotions and ointments, especially those containing tar in some form, give relief as external applications.

ICHTHYOSIS.—The disease *ichthyosis* as it occurs in infants and young children does not differ in its general pathology from that which is seen in adults. It may occur in intra-uterine life, and is then designated *fœtal ichthyosis.*

The most thorough work which has been done on the ichthyosis of infancy and childhood is that of Ballantyne of Edinburgh, who designates that form which has occurred in utero and is fully developed at birth as (1) *fœtal ichthyosis*, while the form which begins in the early weeks of infancy he speaks of as (2) *ichthyosis neonatorum.*

(1) FŒTAL ICHTHYOSIS.—The severity of fœtal ichthyosis varies greatly. The grave form, according to Ballantyne, is developed probably about the fourth month of intra-uterine life, and is characterized at the time of birth by the existence all over the body of horny epidermic plates separated from one another by fissures and furrows, associated with deformities of the mouth, nose, eyes, lips, and limbs, and leading within a few days or even hours to the death of the infant. As in most cases infants with this disease are born alive, fœtal ichthyosis cannot be considered to be a cause of intra-uterine death. The disease does not seem to affect especially the size and weight of the infant. As a rule, the viscera at the post-mortem show nothing abnormal except an unusual degree of congestion. The microscopic examination shows no extension of the keratinizing process on any of the mucous membranes.

The minute anatomy of the disease has been carefully studied by Kyber and Carbone, and the most striking feature of the diseased condition is found to be an enormous thickening of the epidermal layer. This increase in the epidermis is due almost entirely to hypertrophy of the stratum corneum. The results of still further investigation seem to show that the proliferating activity of the cells, instead of being increased, is actually diminished. In a case examined by Southworth the rete Malpighii, the corium, the sweat glands, the sebaceous glands, and the hair-follicles were found to be normal.

SYMPTOMS.—In the early hours of life infants with this disease usually
loudly and continuously, but sometimes the cry is feeble and often very
liar. The respiration is usually impeded by the blocking of the nostrils
epidermic masses. Suction is rendered difficult or altogether impossi-
by the presence of ichthyotic plates around the mouth. They, how-
, are usually able to swallow readily. As a rule, nothing abnormal is
d in connection with the urine or the fæces. Insomnia is a marked
ptom.

These infants have a very repulsive appearance, and there is a cadaveric
r arising from the abnormal condition of the skin. This ichthyotic
dition of the skin is usually universal, but is most evident upon the face.
e mouth is ordinarily kept open by the contraction of the surrounding
ts, and from its angles radiate fissures which simulate the rhagades of
hilis. The lips are thick and everted, so as to form an irregular entrance
he gaping buccal cavity. The chin is receding. The nose can scarcely
seen, it is covered so thickly with the epidermic plates around the nostrils.
ere is usually ectropium of both eyelids, but sometimes only of the upper
, the orbits seeming to be occupied by fleshy tumors. If, however, we
arate the swollen eyelids, the normal eyeball is found to lie beneath.
e external ear seems to have disappeared almost entirely.

In contradistinction to the opinion formerly held that fœtal ichthyosis
a general seborrhœa, it is now generally supposed to be connected with
disease as it occurs in the adult.

PROGNOSIS.—The prognosis of the disease is almost always unfavor-
.

TREATMENT.—The treatment should be active and directed towards
ening the epidermic scales by means of warm oil inunctions.

Besides the grave form of fœtal ichthyosis, there is a much milder form
he disease. It develops during intra-uterine life, and shows a contin-
s layer of a substance resembling collodion extending over the whole
y and falling off in small flakes resembling pieces of tissue-paper. These
eral appearances are sometimes accompanied by ectropium and eclabium.
disease is not, as a rule, fatal, and often terminates in complete or partial
. There have not been any instances, so far as I know, of an infant's
ng born dead with this form of ichthyosis.

TREATMENT.—The treatment of this second form should be by con-
al stimulation of the child's general strength and by great care of the
n.

(2) ICHTHYOSIS NEONATORUM.—Ichthyosis in the new-born infant,
re at birth there was no sign of the disease, may occur. It presents the
e appearances as the milder form of fœtal ichthyosis and the ichthyosis
he older child and the adult.

This is the common form of ichthyosis, which occurs at all ages. It
lly begins in the early months of life, is essentially chronic, and is very
ctable to treatment.

TREATMENT.—It should be treated by the administration of a warm bath once daily, followed by an inunction with glycerite of starch.

SCLERODERMA.—*Scleroderma* is a disease which at times occurs in children as it does in adults. It consists of an induration of the skin either in bands or in patches, or is diffuse, having a board-like hardness, so that the skin cannot be raised by the fingers and feels as though it were tacked down. Scleroderma affects the motions of the joints, and when it occurs about the chest and throat may interfere with respiration. It appears to be a condensation of the fibrous layers of the skin, so that the bundles of muscular fibre are packed closely together and are increased in number. It is chronic, is not very dangerous, and is best treated by massage and lubricating applications.

SCLEREMA NEONATORUM.—*Sclerema neonatorum* is evidently a constitutional disease, and I have therefore already described it in my lecture on "Diseases of the New-Born" (Division VIII., Lecture XX., page 453).

ŒDEMA NEONATORUM.—*Œdema neonatorum* is a rare disease, which some authorities describe as distinct from sclerema neonatorum, the chief difference being that the skin pits on pressure and is not so hard as in the latter disease. The general symptoms of the two diseases resemble one another very closely.

ACUTE CIRCUMSCRIBED ŒDEMA.—A lesion of the skin which has been termed *acute circumscribed œdema* is represented by the sudden appearance of circumscribed swellings of certain parts of the body, varying in intensity and size in different localities. It is closely allied to urticaria, and was formerly described under the name of *giant urticaria*. We do not know much about either its cause or its pathology. I have sometimes met with it in children where it was evidently of reflex origin, depending, probably, upon irritation in various parts of the body, such as the mouth, the genitals, and the gastro-enteric tract.

It is not dangerous, may occur at any age, and its treatment is simply symptomatic.

A case illustrating this disease came under my notice not long since.

A little boy (Case 209), two and one-half years old, had had diarrhœa during the summer, and had been left in rather a weak, debilitated condition. He had for some weeks been pale, fretful, and constipated. His appetite had been capricious, and he had not cared to take any food but milk. When he was nineteen months old an egg had been given to him, which he vomited, and later a slight swelling of both eyes had occurred, lasting for a day or two.

When I saw the child the history that was given me was that in the morning he had eaten an egg. Soon after he became rather dull and cross, but did not vomit. A slight swelling of both eyes was then noticed, and later, when I saw him, the right eye was very much swollen, so that the conjunctiva was corrugated, and the tissues of the eyelids and of the cheek under the eye were so swollen that the eye itself could be examined only with the greatest difficulty. Each time that the child had eaten an egg this swelling occurred in about fifteen minutes. In the course of a few hours the swelling passed off, and did not return. An examination of the urine gave the following result:

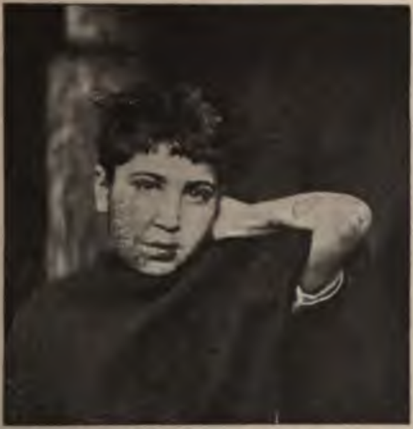
ANALYSIS 59.

or	Normal.
ction	Acid.
phæin	Diminished.
oxyl	Increased.
a	Increased.
umin	Absent.
ar	Absent.
pigments	Absent.
cific gravity	1024.
orides	Normal.
thy phosphates	Normal.
aline phosphates	Slightly increased.
iment	Slight increase of mucus and of epithelial cells.

ier instance (Case 210) of this kind occurred in a little boy, three years old, in peripheral irritation was evidently dependent upon a tight and irritating prepuce. e sudden œdematous swellings of the fingers and backs of the hands would occur r times, lasting for a few hours, and would then entirely disappear. These mani- continued until the child was circumcised, since which time the symptoms have ed. In this case, also, the urine was found to be normal.

ERCULOSIS OF THE SKIN (Lupus, Scrofuloderma).—The that I have to show you is one of a class the cause of which for ars was unknown. It was designated by various terms, according ifferent forms which it assumed on the skin. Thus, in one form it d lupus, in another scrofula. We now know that all these forms d by the same micro-organism, the *bacillus of tuberculosis*, and that llus may find its nidus in the skin as it does in various other organs dy. That is, we may have a local tuberculosis of the skin.

CASE 211.



Tuberculosis of the skin. Female, 7 years old.

ittle girl (Case 211), seven years old, shows the lesions produced by the tubercle-

ee these lesions on the arm where they have assumed a circular form, and in the middle part of the forearm is one with the active part of the disease on the periphery. Where the disease has destroyed the skin in the middle of the lesion you will notice the whitish color of the atrophied skin and the resulting scar. On the right side of the face, under the right eye, and around the upper and lower lips are hard masses of indurated tissue covered with small nodules, papules, pustules, and crusts. You will also notice that, following the general rule of tuberculosis of the skin, the forehead and scalp are not affected.

The child was treated at the Children's Hospital for the disease with the actual cautery. Later the tubercular process appeared in the form of nodules in the scar. This disease, under all circumstances, is very intractable to treatment, and often causes great deformity.

This affection does not differ in the child, in its appearances, its course, and its general symptoms, from the same disease as met with in the adult. I shall, therefore, not speak of it more fully. As a rule, it causes, next to syphilis, the greatest destruction of tissue of any known disease of the skin. The time of its appearance varies, but it is more common in adults than in young children.

TREATMENT.—The treatment is the same as is employed when the disease occurs in adults. The fundamental object to be attained is the destruction of the diseased tissue. Where there is a small isolated area which can be easily removed by the knife, this method of treatment should be employed. We must remember, however, that by this method it is almost impossible to avoid removing the sound tissue with the diseased, and that such good results as the avoiding of unsightly scars are not obtained so well by this method as by others. Therefore where the tissues are extensively diseased and areas are involved where it is desirable to avoid scarring, such as the face, a locality which is very frequently attacked by tuberculosis, the actual cautery or electro-cautery may preferably be used, and various chemical aids, of which the solid stick of nitrate of silver as recommended by the Vienna School is a good example, have been found to be very valuable.

DIVISION X.

SYPHILIS. ERYSIPELAS. THE EXANTHEMATA.

LECTURE XXII.

SYPHILIS.

THE specific organism which causes syphilis has not yet been dis-
red. The disease as it is manifested in early life appears in two forms,
) *acquired* and (2) *hereditary*.

The former differs in no respect from the disease as it occurs in adults,
is transmitted by direct infection, usually through one of the mucous
branes. Its treatment and general characteristics are the same as in
ts, and I shall, therefore, not do more than refer to so broad a subject
quired syphilis.

HEREDITARY SYPHILIS.—The hereditary form of syphilis, on the
hand, plays an important part in the diseases of the early months of
nd is an affection which in all its phases should be thoroughly under-
by those who practise among children.

inherited syphilis we mean a congenital disease which has been trans-
to the child through one of the parents or through both. It makes
earance either in the early months of life (syphilis of the new-born)
later period towards puberty (retarded syphilis). The stage which is
h at birth usually corresponds to an early stage of acquired syphilis,
at which is delayed until later childhood or puberty corresponds to
tage.

question whether the infant can inherit syphilis from the father
the infection of the mother is one which has not yet been deter-
lly. The weight of evidence is in favor of the view that its oc-
n this way is not possible. The probability is that some mild
ent form of the disease has been overlooked in cases where the
been apparently healthy, especially as the mother of a syphilitic
ways immune to infection by her infant. Instances, however,
e it is impossible to say that the mother of an undoubtedly
fant is also syphilitic. A case of this kind I have here to show

The father of this infant (Case 212) acknowledges having been treated for a primary syphilitic lesion which was followed by pronounced secondary symptoms. The mother (Case 213) is, as you see, a healthy, strong woman, who has always been perfectly willing to give any information required either as to her own or as to her husband's condition, in order to aid in the preservation of her infant's life. She states that she has never had any miscarriages, that she was perfectly well both before and after the birth of this infant, and that she has never had an efflorescence on her skin, a sore throat, or any lesions of the mucous membranes. She came under my observation when her infant was six weeks old, and has since then been seen sufficiently often for me to say that so far as I can determine she has had no symptoms that in any way could be attributed to syphilis. She has always had a plentiful supply of breast-milk, which was evidently of good quality.

The severity of the disease determines the type of the efflorescence, and is also influenced by the time when the infection of the fœtus took place. Thus, the later the period of infection the milder will be the form of the efflorescence which first appears, while the less severe the general symptoms the better will be the prognosis and the greater the amenity of the disease to treatment. The reverse of these rules is found where the infection has taken place early, and where, as a result, the infant is born dead, or at birth shows such advanced stages of the disease as are represented by the more intractable forms of efflorescence and severe general symptoms, making the prognosis exceedingly grave.

It is probably possible for a syphilitic fœtus to infect its mother in utero. This theory of *retro-infection*, however, has not been universally accepted. Fournier believes that there is a class of cases in which the father at the time of marriage has no lesion which would necessarily infect the mother, where the mother never shows any initial lesion and remains free from syphilis so long as she is unimpregnated, and where after impregnation she becomes syphilitic and either aborts or gives birth to a syphilitic infant. In connection with the subject of retro-infection the question arises whether a mother who becomes syphilitic during her pregnancy can infect the fœtus (*post-conceptional syphilis*). There is no doubt that she may abort from her own syphilitic infection, but it has not yet been clearly proved that the fœtus in these cases is also syphilitic.

It has been found that where a woman is syphilitic it is exceedingly common for her to abort. Miscarriage is more frequent when a woman is passing through the early stages of syphilis than later when she has become more or less habituated to the disease. The treatment by mercury in these cases soon after impregnation, and continued during the pregnancy, is a valuable means of averting abortion. You must remember that although the aborted fœtus of a syphilitic woman is usually macerated, yet such a condition of the fœtus may be produced by other diseases as well as by syphilis. Birch-Hirschfeld has found from an examination of a large number of macerated fœtuses that seventy per cent. were undoubtedly syphilitic.

Although the tendency to transmit the disease is greatly lessened by time, yet the thorough treatment of the parents by mercury is the most owerful means of preventing such transmission, and the careful use of this rug in proper doses is never contra-indicated. It is, therefore, evident that when a syphilitic woman becomes pregnant she should be treated with mercury whether she was infected before or after conception. When both parents are syphilitic, and when their syphilis is in the early stages, the nfant is most likely to inherit the disease, and under like conditions the disease is apt to be of a severe type.

Infants entirely free from syphilis, either at birth or later, have been known to be born of parents of whom one or both were undoubtedly yphilitic. Through the courtesy of my colleagues at the Boston Dispenary, Dr. Dixwell and Dr. Greenough, I am enabled to show you some ases of immunity in children born of syphilitic parents.

These children (Cases 214 and 215) are two of a family of five, all of whom were ealthy at birth and none of whom have ever shown any symptoms of syphilis. The father was infected with syphilis before marriage, and later infected his wife. They were both arefully treated with mercury. The wife has never had any abortions. She has had five hildren and has lost none. Both father and mother have had undoubted secondary and ertiary lesions, some of which still exist.

The father of this next child (Case 216) is a rag-sorter, who had a primary syphilitic sion on his hand twelve years ago. This lesion was followed by secondary symptoms. He never had any lesion on the penis. While he was being treated his wife showed sympoms of syphilis and was also treated with mercury. This child has always been healthy, nd is one of three, none of whom have ever developed any syphilitic lesions.

Pathology.—The pathological tissue-changes which take place in the hereditary form of syphilis are of the same nature as those which occur in the acquired form. Diffuse *interstitial hyperplasia* is much more common in the hereditary form than are circumscribed gummy tumors. Changes in the bones are very common in hereditary syphilis, and in fact so much so that it is usually considered necessary to find these osseous changes in order to establish a diagnosis of syphilis in the fœtus.

Liver.—Gubler's description of the alterations which take place in the livers of syphilitic infants is as graphic and as reliable as any which have been since given. The liver is always larger than in the normal condition. He states that the hepatic tissue is harder and more elastic than ual, that it is of a yellow color, and that there are small white granutions scattered throughout the parenchyma. The hepatic acini under rmal conditions are in contact, except at the prismatic spaces formed by eir union, in which spaces the capsule of Glisson forms an envelope to afferent portal vessels of the lobule. It is in these spaces that the and lymph-cells form and collect into small lobules representing micropic gummata. The gummata of the liver which are found in young ildren with hereditary syphilis resemble those which occur in adults.

Spleen.—Parrot states that next to the osseous system the spleen is part most often affected by syphilis. It is enlarged, and the degree of lenic enlargement is usually characteristic of the severity of the disease.

Pancreas.—Birch-Hirschfeld has pointed out the fact that the pancreas is frequently found to be affected in hereditary syphilis. He remarks that the interstitial changes which he found in the pancreas are analogous to those which occur in other organs, especially the liver, and that, while these changes are not constant, they come next in frequency to the alterations in the spleen. The interference with the function of the pancreas, which must occur where it is diseased to any great extent, is probably the cause of the gastro-enteric disturbances so common in hereditary syphilis.

Throat, Upper Air-Passages, Thymus Gland, and Heart.—Extensive lesions are at times found in connection with the pharynx, larynx, trachea, and neighboring parts, and also with the thymus gland and with the muscles of the heart.

Lungs.—In cases of hereditary syphilis born before term, and in those born at term who live but a few days, the lungs present certain pathological conditions represented by nodules or small tumors, usually superficial and varying in size. Sometimes an entire lobe may be involved, and the dense, altered lung-tissue is colorless gray or white, both on its surface and on its section. This condition has been called by Virchow *pneumonia alba*, white hepatization.

Kidney and Testicle.—The kidney and testicle may show the lesions of syphilis. It is to be noted that the lesions of these organs are amenable to treatment. The disease in the testicle is represented by a gradual enlargement, and is usually bilateral.

Osseous System.—The changes in the bones which take place in hereditary syphilis are so important, not only on account of their pathological interest, but also because of their clinical significance, that especial attention should be paid to them.

In this connection it should be remembered that in the latter part of intra-uterine life the long bones are cartilaginous and the process of ossification is intra-cartilaginous. As the cartilage changes to bone the cartilage-cells increase in number and are closely crowded together. Then comes the area of osteoblasts, then the calcareous matter, and deeper down in the ossified portions are the blood-vessels running in from the periosteum. The epiphyses of the bones of the arm are cartilaginous at birth, and they remain separated from the shaft of the bone for some time by a narrow cartilaginous layer. It is in this cartilaginous separating layer, called the zone of proliferation, represented in this drawing of a normal infant's bone (page 1066, Fig. 148), that certain changes are found in hereditary syphilis. This same cartilaginous layer is a marked feature in the changes which take place in the bones of cretins and of rhachitic persons. These I shall describe later, but, as you see, they are represented in this illustration. It is also at this zone of proliferation that the growth in the length of the bone takes place, and here syphilitic changes are most often found. This lesion is an osteochondritis, and may occur together with lesions of the spleen and other parts of the body, or as the only manifestation of the disease.

Osteochondritis is ordinarily the form of bone-disease in infants. Osteoperiostitis belongs almost exclusively to the later forms of hereditary syphilis as they appear in well-grown children and in young adults.

The bones which are affected most commonly are those of the arms and of the legs.

Besides these common osseous lesions a morbid condition of the fingers and toes, called *dactylitis*, occurs quite frequently. In this condition the fingers and toes assume a peculiar pyriform shape.

In addition to these purely syphilitic changes, local thinning of the bones of the skull, called *craniotabes*, occasionally occurs. In this condition the bone-substance is absorbed, leaving only the integuments and membranes.

SYMPTOMS.—In the mild form of the disease the infant may be born apparently healthy and may show no indications of its syphilitic inheritance for some weeks. It is rare, however, for the symptoms to be delayed beyond the first three or four months of life. The earliest symptoms of hereditary syphilis correspond to the secondary symptoms of acquired syphilis. Commonly, unless the infant is born with the efflorescence, it is noticed at birth, or within two or three weeks, to have occlusion of the nares (snuffles), and, soon after, a hoarse cry and an efflorescence of a macular or a papular variety. The efflorescence is general, includes the palms of the hands and the soles of the feet, and is especially prominent on the forehead.

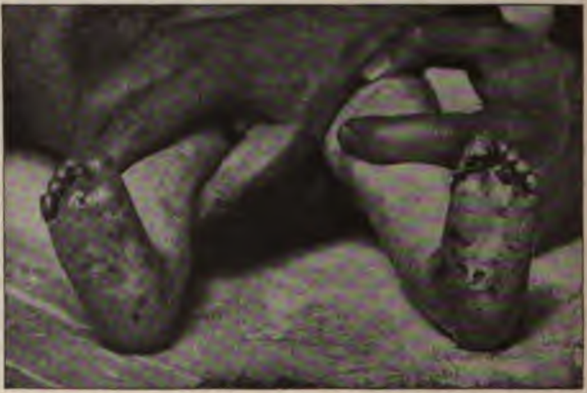
The condition of the infant depends considerably on that of the mother. The rule is that these infants when born are emaciated, presenting somewhat the appearance of these premature infants (Cases 102 and 106, pages 291 and 303), but I have seen them well developed and apparently in good condition, as is shown by this infant (Case 218, page 501), which I shall presently allow you to examine. The disease, with appropriate treatment and good feeding, may in some cases be arrested in this stage, and be cured so that it will not return, or it may advance to another group of symptoms, which are represented by lesions of the mucous membranes. These lesions consist of fissures at the angles of the mouth, mucous patches in the mouth, and condylomata of the anus. In addition to these manifestations, pseudoparalysis of one or both limbs of a greater or less degree may occur. All these symptoms may arise, run their course, and completely disappear, sometimes never to return. Again, they may reappear at various times during the individual's life, but they are especially liable to return during the middle period of childhood and at puberty.

THE EARLY MANIFESTATIONS OF HEREDITARY SYPHILIS.—I have already explained to you that we can judge to a great degree as to the severity of the disease by the type of the efflorescence, and also by the time when it occurs after birth. The mildest and most benign form of syphilitic efflorescence is represented by *maculæ*, the next by *papulæ*, and the next by *pustulæ* and *bullæ*. Another form of efflorescence simulating *psoriasis* is one of the more severe manifestations of syphilis, as is also that form which is called *rupia*, where the efflorescence consists of thick layers of crusts arranged one above the other, forming a conical mass, the skin at the base being somewhat infiltrated. All of these types of the disease have been known to be cured. Finally, you will at times meet with a very dangerous form of the disease, which is almost uniformly fatal no matter what the treatment may be. This is what is called *syphilitic pemphigus*, and is represented by large and numerous bullæ.

These syphilitic efflorescences, unlike most other lesions of the skin, appear commonly on the palms of the hands and the soles of the feet.

Here is an infant which I showed you at a previous lecture (Lecture XV., Case 127, page 367) to illustrate the enlarged spleen which is found commonly in cases of secondary anæmia produced by hereditary syphilis. As seen to-day it well illustrates what I have just said concerning the syphilitic efflorescence appearing on the soles of the feet.

CASE 127.



Syphilitic maculæ, ulcers, and bullæ on the soles of the feet. Male, 2½ months old.

It is a male, two and one-half months old. About one month previous to its birth its mother had an efflorescence limited to the head: her hair fell out, and she had a sore throat. The infant was apparently healthy at birth and during the first six weeks of its life, and had no unnatural appearances on its skin. It was then noticed to have an efflorescence of an erythematous type on the body, face, and arms, including the palms of the hands and the soles of the feet. This efflorescence was in the form of maculæ of a bluish-red color. On examining to-day the soles of the feet, we find, in place of the pronounced maculæ which you have previously seen, pigmented areas. You will also notice on the under side of the toes at their junction with the metatarsal bones a number of bullæ, some of which have burst and, the tissue beneath having broken down, ulcerations have been formed. In other parts of the soles you will also notice ulcers of various sizes, a few papules, some smaller bullæ and the pigmented areas already referred to.

There is marked occlusion of the nares, and an examination of the nose which has been made by Dr. Coolidge shows that the turbinated bones on the left side are swollen and that there is some infiltration of the mucous membrane of the naso-pharynx. There is also a sero-purulent discharge from the left eye.

In a case of this kind, provided that we can eliminate the extreme lesions of scabies, there can be no doubt that the lesions are those of syphilis.

In addition to these general symptoms which I have just described, there occurs in the hereditary form of syphilis the loss of hair which, as you know, is so common in the acquired form of the disease. This *alopecia* may be caused by any of the dermal lesions which occur during the course of the disease, but is probably due mostly to the general lack of nutrition in which the skin participates with the other organs of the body in syphilis. In certain cases the eyebrows and eyelashes are lost, and Barlow believes that the former condition is characteristic of the disease, or at least should excite a suspicion of its presence.

Enlargement of the lymph-glands, *adenopathy*, seems to be less marked in hereditary syphilis than in the acquired form. This enlargement may be due to reflex irritation from the more severe dermal lesions, but in certain cases it is found where no dermal lesion exists. The enlarged glands may be in the inguinal, the axillary, or the cervico-maxillary regions. They are distinct, movable, multiple, and non-inflammatory. The older the child the more likely the glands are to be enlarged.

According to Post, the nails are involved quite frequently in hereditary syphilis, and more frequently than in the syphilis of the adult. The *onychia* occurs in two forms. In the first form a papule or pustule appears on the skin at the side of the nail. This ulcerates and extends along the side of the nail, at times involving the matrix and causing the loss of the nail. The thick and everted edges of the ulcer, its sloughing base and sanious discharge, are somewhat characteristic, and are accompanied by a painful enlargement of the distal phalanx.

The effect of hereditary syphilis on *dentition* is quite marked. The first teeth instead of being cut in the sixth or seventh month may not appear until the fourteenth or fifteenth month, and sometimes even later. These primary teeth are especially liable to decay early. There is nothing sufficiently characteristic to be of diagnostic value in the appearance of the teeth of the first dentition.

Mr. Hutchinson has observed twenty-three cases of *iritis* in syphilitic infants. The average age for the beginning of the iritis was five and a half months. The oldest was sixteen months at the time of the outbreak, the youngest six months. Both eyes were affected in eleven cases, and in fifteen cases the effusion of lymph was copious. The cornea was affected in a few cases. In seven cases the cure was complete, in twelve the pupil was partially occluded. Iritis is one of the rarest of the symptoms of hereditary syphilis, and at times escapes notice on account of the very slight symptoms which usually attend it. The diagnosis in these cases is not dependent on the iritis alone, but the infants always show other well-marked symptoms of syphilis. There is great danger of the disease resulting in blindness if it is left untreated, and mercurial treatment is most efficient in effecting a cure.

In regard to the *digestive disturbances* which arise in these cases of hereditary syphilis, it is well to remember that they may depend upon a syphilitic lesion of the liver, spleen, and pancreas, as well as of the stomach and intestines. It is, therefore, necessary to treat these disturbances of the gastro-enteric tract in a different manner from what is customary where a local non-syphilitic cause is supposed to be present. In fact, mercurial treatment will produce the best results in these cases.

An affection called *syphilis hæmorrhagica neonatorum* is met with at times. Bumstead and Taylor have reported two cases of this kind, and state that the disease is rare, less than twenty cases having been noted. The hemorrhages vary in their extent, and may occur in either the skin or the mucous membranes. This class of cases is difficult to differentiate from the hemorrhagic disease of the new-born which I have already described. There is no doubt that syphilis has in a number of cases an etiological significance in the umbilical hemorrhage which occurs in the early days of life. Dr. Uracek has reported a series of hemorrhages in the different internal organs apparently depending upon a syphilitic taint in the infant.

The course of syphilis is so influenced by treatment that the symptoms must necessarily be irregular. When the disease is untreated, as a rule, all the symptoms grow worse. The infant becomes more and more emaciated, and either it dies in a few weeks of inanition, or the disease progresses still further and serious lesions of the various organs, such as the lung, liver, spleen, and kidney, may finally produce a fatal result. The *occlusion of the nares* may increase to such a degree that the breathing of the infant is seriously interfered with, and, without any other syphilitic lesion, it may die from imperfect oxygenation of the air which enters its lungs.

This occlusion of the nares may cause great loss of sleep. We must, however, understand that, even where this lesion is not of any great extent, syphilitic infants suffer from *insomnia*. This insomnia is usually accompanied by crying, so that it is probable that the restlessness and insomnia are due to pain in the bones, as these symptoms are often present where there is no digestive disturbance.

In connection with these syphilitic lesions of the nose, flattening of the bridge of the nose is at times a noticeable symptom.

There is nothing especial to describe concerning the *condylomata* which are found in the *anal region* and are rare in comparison with the lesions of the mouth. They begin as rounded papules, which sometimes coalesce, and there is more or less infiltration of their edges and breaking down of their centres.

The syphilitic lesions of the *mouth* are found so commonly, and are of so important a character, that an especial description should be given of them. There is no syphilitic lesion of the mouth which is represented by a characteristic stomatitis. The mucous membrane in the course of hereditary syphilis may at any time be in so sensitive a condition that the various forms of stomatitis may be engrafted on it, and we thus may have different lesions of the lips, tongue, buccal cavity, and tonsils, which, while imply representing the lesions of certain non-syphilitic affections, may, by heir peculiar grouping in combination with other symptoms, represent the ereditary form of syphilis. The lesions most commonly appear around he lips and on the mucous membrane lining the cheeks. On the lips ssures are exceedingly frequent; on the upper lip they commonly appear n either side of the median lobule, while on the lower lip they are usually ingle and in the median line. The angle of the mouth is often the seat f condylomata, and these are frequently covered with crusts and at times re deeply ulcerated. A peculiar appearance is in some cases seen at the ommissures of the mouth, caused by cutaneous ulcerations, which make it ook larger than normal, and at times produce a number of lines radiating rom the mouth to the cheeks. Ulcerations may occur on the tongue, the ips, and the fauces. Forchheimer has written more fully on these lesions f the mouth than any other author, and his observations, now so widely nown, leave little additional to be said on the subject. His description f the fissures which occur in syphilitic infants' mouths is very minute. e considers that when they are present they leave no doubt as to the diagosis, since they are infiltrated. The most common place for them to appear at the corner of the mouth. In this place, as a rule, the most striking eature of the fissure is that it is a papule which has been split in or about ts middle, and that it has an infiltrated edge. The fissures sometimes disppear in the mucous membrane, sometimes stop before reaching it, and ometimes run into it. The fissures may or may not be covered by a crust, nd, unlike most syphilitic efflorescences, produce more or less pain when the nouth is opened. These fissures are called *rhagades*. They are characterzed by their persistency and by their lack of tendency to spontaneous ealing. Ulcers and plaques muqueuses may be found upon the mucous nembrane of the lips and cheeks and on the sides and under surface of the ongue. They are superficial, but cover more space than the fissures. The nfiltration is not so well marked, but is present to a greater or less degree. The most common lesions which are found on the tongue are these plaques nuqueuses and ulcers. Both have infiltrated edges, but the plaque in this ituation rises above the level of the tongue, while the ulcerations are onsiderably depressed. They are both characteristic of syphilis. Their ocality is determined somewhat by the presence of such irritants as sharp eeth pressing against a portion of the tongue.

The secretion of all these lesions of the mouth and lips is highly nfectious.

One of the striking symptoms of this early stage of hereditary syphilis esults from osteochondritis. According to Post, the form of lesion is sually that of a tumor at the junction of the diaphysis and epiphysis at the istal end of the long bones, though any part of the osseous system may be nvolved. These swellings are difficult to recognize in fat children. The umors rise abruptly from the bones; they are small and globular, and in ome cases form a ring at the junction of the shaft and epiphysis; in others the whole epiphysis is enlarged. At times only a part of the cartilage is affected, and the external swelling is correspondingly circumscribed. The lesions appear soon after birth, and their development is completed either slowly or rapidly. The termination varies widely. The swelling may be absorbed under appropriate treatment, or suppuration may take place and the skin break down; the disease may end in the separation and destruction of the epiphysis. The result upon the final growth of the bone varies, of course, with the severity of the local disease. When the morbid process is arrested before the destruction of either cartilage or epiphysis, there is no deformity, but the destruction of cartilage puts an end to growth at that point, and a more or less shortened and useless limb results. When the disease takes such a course as to separate the epiphysis while the integuments remain sound, the limb becomes useless for a time and appears to be paralyzed. The disease was first fully described by Parrot, and is known as Parrot's disease, or *pseudo-syphilitic infantile paralysis*. The joints in immediate connection with the diseased bones are sometimes involved. There may be simply an effusion, but, where the bone is destroyed, serious disorganization of the joint must follow. The pain and sensitiveness in these cases of pseudo-paralysis are probably caused by a low grade of periostitis.

The bones of the fingers and of the toes, I have already told you, present at times the peculiar lesion which is known as *dactylitis syphilitica*. The phalanx may be enlarged to two or three times its natural size, giving the fingers a pyriform shape. One or several fingers or toes may be involved, and sometimes the metacarpal bones are diseased. The proximal phalanx is more frequently affected than the distal phalanx. In the early stages the integument is unchanged; later, the overlying parts become involved and abscesses form. If the case is submitted to early treatment the deformity usually subsides, but if untreated the disease may result in permanent deformity and uselessness. Dactylitis, however, is not characteristic of syphilis alone, as it occurs also as a result of tubercular disease of the bone.

Craniotabes is one of the more uncommon symptoms of hereditary syphilis, but, as I have already told you, may in rare cases be found. These softened spots, nearly circular in form and about 1.2 cm. (½ inch), more or less, in diameter, may be recognized by the finger during life. Until lately craniotabes was considered to be exclusively a symptom of rhachitis. It is found especially in the occiput. It is present in rhachitis where no trace of syphilis can be discovered, but it seems to be most common in cases where there is a distinct syphilitic taint. Of one hundred cases of craniotabes collected by Drs. Barlow and Lees, in forty-seven there was satisfactory proof of syphilis.

DIAGNOSIS.—The diagnosis of hereditary syphilis in its more advanced forms, such as I have just described, is not difficult, as no other disease represents such serious lesions of the skin with such a combination of general symptoms and lesions of the mucous membranes.

The milder forms of the disease are frequently mistaken for other seases of the skin which simulate the syphilitic lesions but which are of benign character. I have already spoken of these lesions when describing ich local diseases of the skin as papular erythema, and shall refer to them ain when speaking of the mild forms of syphilis.

Occlusion of the nares caused by swelling of the Schneiderian membrane, persisting during the early weeks and months of life without rise of mperature, should always make us suspicious of the presence of hereditary philis, for a syphilitic efflorescence is often so slight and evanescent as be frequently overlooked.

Marked improvement from the administration of mercury is also usually nsidered of diagnostic value, and, although not by any means conclusive, at least significant.

Periostitis, especially of the lower end of the humerus or the anterior rder of the tibia, is met with in children. It should make us suspicious at syphilis is causing this condition, especially if there is periostitis of a umber of bones at once.

A great deal has been written and much discussion has taken place garding the relationship between syphilis and rhachitis. The two diseases re so distinctly separated that it seems scarcely necessary to dwell, except ery briefly, on the differential diagnosis between them.

Rhachitis is so largely dependent in its osseous changes on a profound disturbance of nutrition that it can fairly be said to result from any disease which from its debilitating nature may interfere with the nutrition of the bones. In this way individuals whose nutrition has been seriously affected by hereditary syphilis may develop rhachitis. This, in my experience, has been a rare occurrence.

In regard to the actual lesions of the bones present in syphilis and rhachitis, there seems to be a concurrence of opinion that the pathological conditions are quite different. Thus, according to Cazin and Iscovesco, syphilitic bones very rarely present the spongy tissue peculiar to rhachitis, and rhachitic bones never show the osteophytes of syphilis.

PROGNOSIS.—From what I have already told you, the prognosis in any case of hereditary syphilis is a serious one. In addition to the results which we are likely to have from the syphilis of the parents being early or late in regard to the impregnation, and from their having been thoroughly treated or not, there are certain facts to be remembered concerning the infant itself.

The prognosis is grave inversely to the number of weeks after birth when the disease first shows itself. The milder forms of the efflorescence justify us in giving a better prognosis than the more severe ones. In addition to these conditions which render the prognosis more favorable are the possibility of the infant being fed with good breast-milk or with a carefully prepared substitute food, and good hygienic surroundings.

The cases in which the spleen is much enlarged are evidently so profoundly affected by the secondary anæmia by which the enlargement is caused that the prognosis is almost invariably bad, and the degree of splenic enlargement may almost be taken as an index of the severity of the disease.

The opinion which we give to the parents should, however, always be very guarded, as, even though the disease may for the time apparently be entirely cured, it is always liable, as I have already stated, to appear again in later childhood and at puberty. When the disease is amenable to treatment these secondary symptoms almost always disappear by the second year, and in quite a large number of cases, where proper treatment has been thoroughly carried out, the infant recovers entirely and is as well and strong as though it had never had syphilis. In another set of cases, however, although the disease is apparently eradicated, in later years it is found to have left its marks in disturbances of the different functions and in the general lack of vigor of the various tissues.

TREATMENT.—The treatment of hereditary syphilis is first to adapt at once as nourishing a food as is possible to the infant's digestion. A healthy mother with plenty of good breast-milk will, as a rule, provide the best food for her infant.

If the mother's nutrition is reduced by syphilis or by any other chronic disease, the infant should be fed on a properly adjusted substitute food, while the general hygiene, such as fresh air, sunlight, and warmth, should be carefully regulated. A wet-nurse should not be employed unless she has herself had syphilis, in which case the same rules will apply to her nursing as to that of the syphilitic mother. A syphilitic infant does not infect its mother (Colles's law). It readily infects a woman who either has never had syphilis or who has never given birth to a syphilitic infant.

It should be remembered that the secretions from a syphilitic infant's mouth are very infectious, whether the disease is of the hereditary or of the acquired form. If, therefore, the mother is not syphilitic and the infant has acquired in any way a syphilitic lesion, the nursing must be discontinued and the infant fed on a substitute food.

The only drug which can be depended upon in the treatment of the early lesions of hereditary syphilis is mercury. This drug naturally would be employed from our experience with it in acquired syphilis, where, as you know, it is more valuable in the early stage of the disease than at any other period. In like manner iodide of potash is of little use in the early stages of hereditary syphilis, while it becomes useful in the retarded form, which corresponds to the later stage of acquired syphilis.

It is important carefully to adapt the form of mercury which you give to the syphilitic infant according to its special idiosyncrasy for the drug, and also to regulate the means of its administration according to the necessity of having it act quickly, as is indicated in the more severe forms of the disease, and according to the sensitiveness of the individual's stomach or skin. Thus, mercury may be administered either through the mouth or through the kin. In the latter case it may be applied directly in the form of liquid or intment or by means of subcutaneous injections. The last method should e used in very urgent cases only, for the tissues and skin of the syphilitic nfant are especially liable to be irritated to such an extent that sloughing nay take place, and the tissues under these circumstances are readily destroyed. When used, it should be in the form of corrosive sublimate.

The corrosive sublimate should never be given subcutaneously in larger oses than 0.0006 gramme ($\frac{1}{100}$ grain). Where the mercury is to be applied irectly to the skin it may be in the form of corrosive sublimate baths, .3 to 0.6 gramme (5 to 10 grains) to each bath once daily, but practically t is found better to introduce it into the system by means of an ointment. his ointment may be the official mercurial ointment, either in full strength r diluted with some simple ointment, and this is very often applied by neans of inunction, as is the custom in the acquired syphilis of adults. fter the infant's skin has been thoroughly washed, a small portion of the intment should be applied to its back and rubbed carefully and gently nto the skin for ten minutes. On the next day the same procedure can e carried out on the front of the chest; on the third day in the axilary regions; and on the following days respectively on the outer suraces of the arms and thighs. I have found that the most practical way f applying inunctions to these infants is, after having thoroughly washed he abdomen, to spread the ointment thickly on a piece of thin soft flannel ut so as to reach from the ensiform cartilage to the pubes and to extend round the entire abdomen. This ointment is made in the following way (Prescription 66):

PRESCRIPTION 66.

Metric.	Gramma.		*Apothecary.*
℞ Unguenti oleati hydrargyri,			℞ Unguenti oleati hydrargyri,
Unguenti lanolini . . . . . āā	60	00	Unguenti lanolini . . . . . . āā ℥ii.
M.			M.

The band should be allowed to remain in place for forty-eight hours. It should then be removed, and, after the skin has been thoroughly washed with warm water and soap and dried with a soft towel, the flannel should again be spread with the ointment and reapplied.

In giving mercury by the mouth I am in the habit of using the official hydrargyrum cum creta. I usually begin with 0.06 gramme (1 grain) of the drug, administered three times in the twenty-four hours. Within a few days I increase the dose to four times in the twenty-four hours, and if no unfavorable symptoms appear I again raise the dose to 0.12 gramme (2 grains) three or four times in the twenty-four hours.

The unfavorable symptoms which I have just referred to as possibly being caused by the drug are represented by diarrhœa. We must remember that the infants whom we are treating for hereditary syphilis are so young that the salivary secretion has been very slightly developed, and that therefore we naturally do not salivate an infant of this age so readily as we would a child or an adult. We must not, however, think that we can be guided as to the amount of mercury we are introducing into the infant's stomach by salivation, which is usually relied upon to indicate the physiological action of mercury. I have found it a safe rule to continue with the mercury until diarrhœa is caused, when the drug can be reduced in quantity, or even be omitted for a few days. When the intestine has become less sensitive we can again begin with a smaller dose, and one which by experiment has been shown not to cause diarrhœa in the especial infant.

Other forms of mercury, such as calomel in doses of 0.006 gramme ($\frac{1}{10}$ grain) three or four times daily, may be given by the mouth in these cases.

These various forms of mercury should be tried where for any reason one of them is found not to suit the case.

For the treatment of the fissures which occur around the lips and the lesions of the mouth, as well as those which occur at the anal orifice, I am in the habit of using a simple powder of calomel, which is dusted on the part affected. The mouth should be carefully cleansed several times during the day and a wash of chlorate of potash used at least twice a day. In some cases, though rarely, nitrate of silver is needed as an application to the ulcers when they are intractable. Where there are crusts around the lips and in the neighborhood of the fissures, or where anal condylomata are present, the ointment (Prescription 66) which I have just shown you is of much benefit. The crusts should be carefully removed from the nose and this same ointment gently applied to the lesions. The application of this ointment to the abdomen is at times followed by an eczematous irritation of the skin of the abdomen, as has happened in this case (Case 127, page 367). Under these circumstances any simple ointment should be applied in place of the mercurial for a few days until the skin has recovered, and the ointment can then be further diluted with lanoline or some simple ointment and reapplied, thus finally adjusting the strength of the mercurial to the vulnerability of the infant's skin.

In addition to the mercurial treatment, tonics in some form, especially iron, are at times required. It is usually in the later stages of the disease that they are indicated, and in cases where the persistence of the splenic enlargement shows the presence of profound secondary anæmia.

After all the symptoms of syphilis have disappeared and the infant is entirely well, the mercurial treatment should be continued for some months, and also later during the first three or four years of its life, at intervals of three or four months, even where there is no return of the syphilitic symptoms. It should likewise be given at intervals during the period of the second dentition, and again at puberty. This treatment is especially important whether the infant appears to be in good health or not, as it tends to prevent a recurrence of the disease, and you should understand that a recurrence often proves very intractable to treatment.

I have some infants here to-day who illustrate the different phases of early hereditary syphilis and the different conditions which you are liable to meet with in this disease.

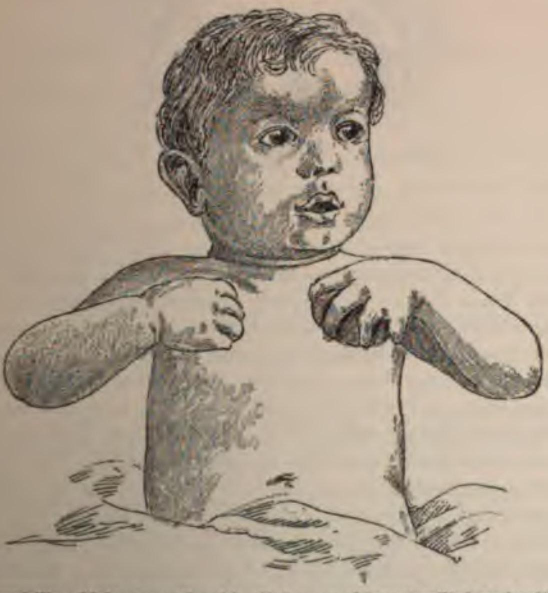
This first infant (Case 217) is three weeks old. Its mother looks well and strong, denies having had any miscarriages or disease of any kind, and asserts that the father is also healthy. Both of these statements are probably untrue, as you will presently see; but we have an excellent opportunity for making a diagnosis simply by inspection and by a physical examination.

At birth the infant was puny and atrophic. It soon began to have occlusion of the nares. When one week old, an efflorescence of papules appeared on its arms, legs, and feet, with pustules on the palms of the hands and the soles of the feet. It does not vomit. The fæcal movements, as you see on the napkins (Plate III., 4, facing page 112), are of a good color and fairly well digested. The heart and lungs are normal. The splenic area of dulness is slightly increased, but the spleen cannot be felt. You see that there are marked fissures at the angles of the mouth, a muco-purulent discharge from the nose, and crusts forming on the eyebrows. The mouth and throat show nothing beyond a pronounced erythema. There are papules and pustules on the body, and a squamous as well as a pustular efflorescence on the palms of the hands and the soles of the feet. There are maculæ on the buttocks. The anus shows nothing abnormal. The temperature is normal. The infant looks fairly well nourished.

There can be no question about the diagnosis in a case like this, and the statements of the mother regarding herself and her husband can be entirely ignored, for by simple inspection we see at once that we have a case of hereditary syphilis to deal with.

Before referring to the treatment of this case, I shall ask you to examine another infant.

Case 218.



Hereditary syphilis. Male, 6 months old. Fed on good breast-milk by a healthy mother.

This infant (Case 218) is six months old. The mother, a healthy-looking woman with plenty of breast-milk, nurses the infant. She has had one miscarriage, in the third month, and this is her first child. The father denies having had any venereal disease.

At birth the infant was rather atrophied and had a general papular efflorescence all over it, and later a squamous efflorescence on the palms of the hands and the soles of the feet. It always had marked occlusion of the nares (snuffles).

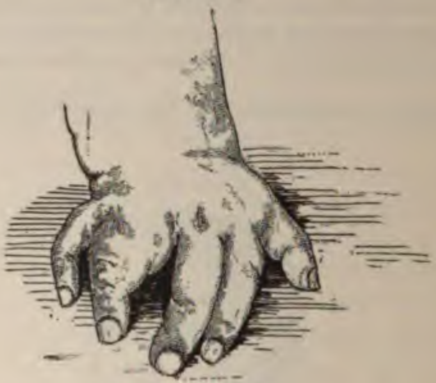
The infant was immediately placed under treatment, and now looks well nourished.

It is also a case of hereditary syphilis, and shows the beneficial result of good breast-milk and mercury, for you see that it is very large for its age and is fat and strong-looking. It has, however, certain lesions of the bones which are the result of the syphilitic manifestations which it presented at birth. One of these lesions is represented in the marked prominences which you see on either side of the frontal bone, with a somewhat depressed sulcus between them.

On examining the infant's hands you will notice a still more characteristic lesion of the bone. You see that the first phalanx of the left little finger and that of the left third finger are swollen and somewhat reddened, and that the tissues have a tendency to break down. This condition is called *syphilitic dactylitis.* It is not, however, characteristic of syphilis alone, for cases of tuberculosis of the bone often simulate this condition, and in fact so nearly approach it in appearance that the two diseases cannot be distinguished by simple inspection.

To show you the close resemblance between syphilitic dactylitis and tubercular dactylitis, I have here an infant (Case 219) on whose hand the same general characteristics will be found. In this case the third finger of the left hand is affected.

CASE 219.



Tubercular dactylitis.

In connection with the first of these cases (Case 217) I have stated that while the syphilitic infant is described essentially as atrophic, this is, as a rule, the case only when it is deprived of good breast-milk or of a properly proportioned substitute food, the atrophy being usually a fault in diet, provided that the intra-uterine nutrition has been good. You see that neither of these cases is suffering from malnutrition. They are being nursed by strong mothers, who are giving them a plentiful supply of milk. The second case (Case 218) is rapidly recovering, and will soon need only to be seen and treated at intervals. In fact, it illustrates remarkably well how healthy an infant may look who is but just recovering from the more severe symptoms of infantile syphilis. In the first case (Case 217) the prognosis is not quite so good, as, although the infant has been under treatment for two weeks, the lesions are marked and numerous. What inclines me, however, to look upon the case favorably is the improvement which has occurred in the mother's milk, and which will naturally find its counterpart in the infant's nutrition. The insomnia and restlessness which were present in this case have also greatly lessened, showing that the infant is improving.

An interesting and important point to be noticed in this case was that when the mother first noticed the efflorescence and brought the child to me she was so much frightened that her milk had considerably lessened in quantity, and she was sure that she would lose her ilk entirely and that her infant would die. Judging that the milk was affected by the ental condition of the mother, I at once caused a marked revulsion in this condition by tating decidedly that her milk would soon become plentiful, and that in the mean time she ould give her infant in addition to her milk about an ounce of the following mixture Prescription 67):

PRESCRIPTION 67.

Fat	2.00
Sugar	6.00
Proteids	1.00

This substitute food suited the infant's digestion so well that the mother soon ceased to believe that it would die, and the desired mental revulsion was so effective that in twenty-four hours the infant was receiving its natural supply of breast-milk and the substitute food was omitted.

Syphilis is so prolific a source of miscarriage that a history of miscarriage in the mother justifies us in looking with suspicion on a doubtful lesion of the skin in her infant. A woman may have a number of miscarriages caused by syphilis, and may then, if she has been treated with mercury, give birth to a living syphilitic infant, or to one that is healthy. These facts are important for us to remember when we are considering the prognosis in a case of hereditary syphilis. For instance, the mothers of both of these infants deny having had any disease, and the mother of the first case (Case 217) says she has had no miscarriages, while the mother of the second case (Case 218) acknowledges that she has had a miscarriage. We may take it for granted from the healthy appearance of these mothers that they have been treated. This opinion, of course, is merely provisional, and does not deal with the additional argument which might be brought up, that both infants were infected by the fathers through healthy mothers. These two infants have both had the same treatment, and that treatment has been essentially good food and mercury in the form of oleate of mercury ointment diluted one-half with rose-water ointment and applied on a flannel to the abdomen. In addition to this external treatment, hydrargyrum cum creta in doses varying from 0.12 to 0.24 gramme (2 to 4 grains) three or four times a day has been given.

The next infant that I shall show you illustrates the trouble that may arise from the physician in general practice not thoroughly understanding the varied forms in which syphilis may manifest itself in infancy.

This infant (Case 220), a male, four months old, was brought to my clinic three weeks ago with syphilis of a rather aggravated type, and among other lesions this condyloma, the remains of which you now see at the anal orifice.

It had a general papular efflorescence on the face, body, and limbs, including the palms of the hands and the soles of the feet. The left arm hung helpless by its side. You see that it can now move it a little. The left leg was also somewhat affected. On examining the arm I found that there was a small, hard, painful, circumscribed swelling at the lower end of the humerus. No crepitation was detected. The infant was treated with mercury, and a carefully proportioned substitute food was given to it. The mother was cautioned to be very gentle when she moved the arm, and to come frequently to the clinic for observation. She did not bring the infant again for two weeks, but when she did she was very indignant, because she thought her infant had not been properly treated at the previous visit. She said that she had been to a surgeon, who had told her that the infant had a broken arm, and that the hard swelling was the resulting callus. The mercury was therefore omitted, and a splint applied. It is needless to say that the arm and the infant grew rapidly worse, the left arm also becoming helpless.

The true nature of the disease was then explained to the woman, the splint was removed, and a vigorous course of mercurial treatment was carried out with the infant; and to-day you see the rapid improvement which is taking place.

Here again we had to deal with one of the osseous lesions of syphilis, an *osteochondritis* accompanied by periostitis, which caused so much pain on movement as to disable the limbs and simulate both paralysis and fracture.

The next case (Case 221, Plate VI.) is of remarkable interest, owing to the form and appearance of the efflorescence, which, though unusual, is so characteristic that it could represent no other disease than syphilis.

The infant is six weeks old. The mother states that she has been married about three years, has had two children, and has had no miscarriages. She says that the father is well and strong, and that neither of them have had any efflorescence on their skin.

The older infant is fourteen months old, and is healthy.

The younger infant is being nursed by its mother. At birth it was apparently healthy and well nourished. Its skin was clear, its body fat, and there was no occlusion of the nares. This condition continued until it was eight days old. It then began to have occlusion of the nares (snuffles), a slightly hoarse voice, and an efflorescence on its back. To-day you see that it has an efflorescence on various parts of the body and limbs. This efflorescence consists mostly of maculæ, many of which are circumscribed by healthy skin. They vary in size from 0.6 to 1.25 cm. (¼ to ½ inch).

The lesions can be studied well by examining the right leg and foot, where their appearances are most clearly depicted. In order to see the exact color and distribution of these lesions, which at present are much obscured by dirt, I shall first have the leg and foot thoroughly washed with soap and water. This can be done without removing their characteristic appearances, since they are but slightly squamous, and, being mostly macular, can best be studied when the skin has been washed clean.

In addition to the maculæ, which you see varying from a delicate pink to a yellowish-white color, is a pustule on the outer side of the leg just below the knee. On the inner edge and almost on the back of the foot are the remains of a bleb which has broken down and has been emptied of its contents. There is also on the inner side of the foot, nearer to the heel, a small ulcer. All the other lesions are maculæ, and you see how distinct are these red maculæ on the sole of the foot. The entire skin of the heel is reddened and has a shining appearance. The erythematous lesions in places on the leg are surrounded by normal skin presenting a mottled appearance, and there are white spots on the skin. These latter, however, are caused merely by the peculiar distribution of the syphilitic maculæ.

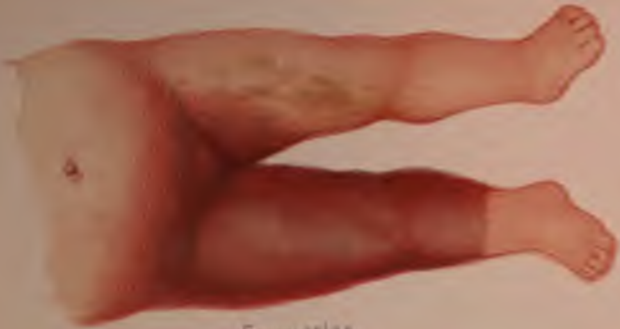
In addition to these lesions on the leg there are a few ulcers on the buttocks, and in addition to the maculæ on the soles of the feet there are some on the palms of the hands. A few scales showing a squamous condition can be seen on the left leg, but this lesion is not a prominent one.

The eyes are not affected. There are a few fissures about the mouth, but no lesions on the buccal mucous membrane, and there are no gummata around the anus.

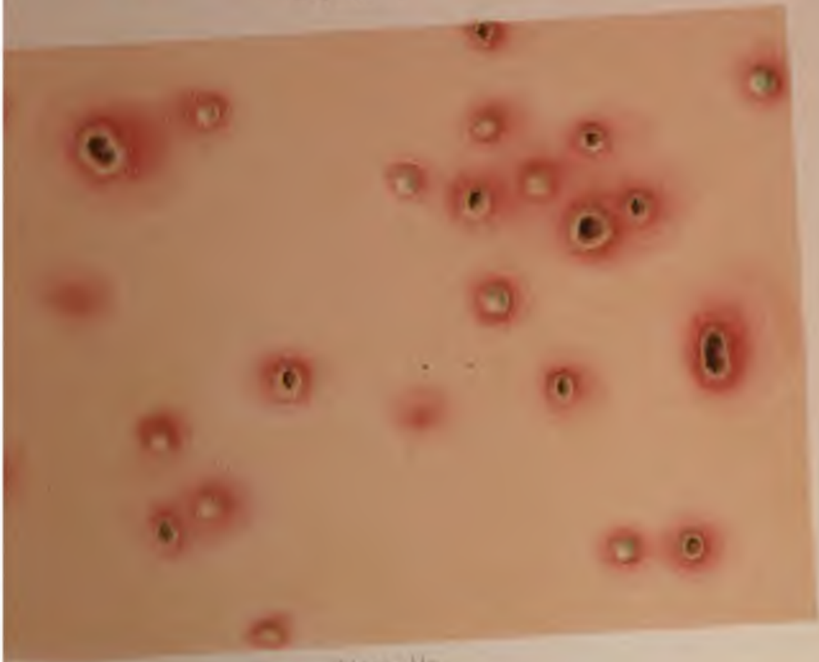
The treatment of this case will be by inunction with the oleate of mercury ointment which I have already described (Prescription 66, page 499), and by the administration of hydrargyrum cum creta.

THE LATER MANIFESTATIONS OF HEREDITARY SYPHILIS.—I have already told you, in speaking of the manifestations of hereditary syphilis which appear at birth, that these symptoms usually develop in the first three or four months of the infant's life. In certain cases of syphilis which are without doubt of the hereditary form, either no symptoms whatever are

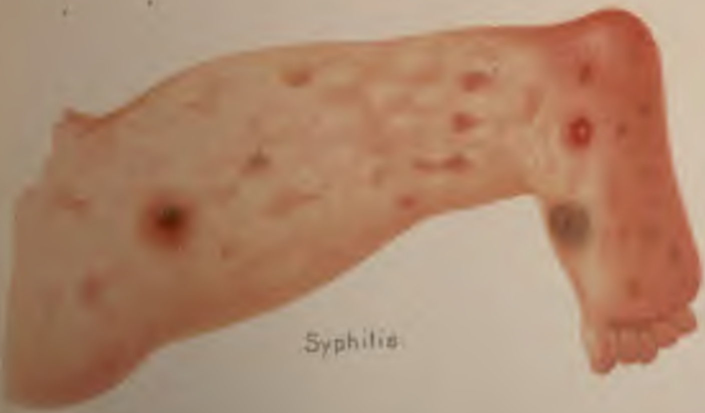
PLATE VI.



Erysipelas.



Varicella.



Syphilis.

ced in the early years of life, or they are so slight, or so lacking in the racteristics of syphilis, that it is sometimes impossible to recognize them yphilitic lesions. The lesions of this late hereditary form correspond to tertiary lesions of the acquired form. They appear in different periods hildhood or at puberty. These periods correspond to what I have ady stated to be the time when a fresh outbreak of an attack of syphilis ch has occurred in the early months of life is apt to take place. This gnificant as leading us to suspect that the early symptoms of the disease e been overlooked rather than to believe that they did not occur.

The lesions of the bones hold a prominent place in these later manifesta- s of hereditary syphilis. These lesions may be in the form of a peri- tis, or an actual necrosis of the bone may take place either in connection h a dactylitis or with a simple lesion of the osseous tissue in any of the es.

As these later forms of hereditary syphilis merely represent the same ditions as are met with in tertiary acquired syphilis, we should expect most varied lesions. In this late form of hereditary syphilis the bones the nose are frequently involved, and a flattening of the bridge of the e is not uncommon. The cranial bones show certain alterations which times are quite characteristic. The frontal bone may present a promi- ce on either side, which, with a depression more or less deep between the minences, causes such a peculiar conformation of the head as to be almost racteristic of syphilis. This is well represented in the case (Case 225, e 510) which I shall presently show you. In addition to these frontal minences, at times there is a prominence of the centre of the frontal e, which, with the apparent flattening on either side, causes a peculiar e simulating the keel of a ship. Sometimes protuberances similar to e which I have described of the frontal bone may appear on the parietal es. When they are bilateral the sagittal suture appears as a depressed us between them, and this deformity of the skull, from its resemblance he shape of the nates, has been designated by Parrot as the *natiform* ll.

These tuberosities which I have just described as appearing on the ll may also appear upon the long bones, either in the diaphysis or in epiphysis. When the tibia is affected there is often so marked an in- ase in parts of the shaft of the bone, especially its middle third, that, the enlargement is chiefly in the anterior portion, the swelling when minent gives an appearance of curvature to the bone. This is, however, ly a seeming curvature, as the posterior portion of the bone is not affected.

An interference with the growth of children who are affected by these ious osseous lesions of syphilis is not uncommon. There is frequently a k of development, which shows itself usually in a failure of the individual attain the ordinary height. The mental development is retarded, the ldren often appearing to be a number of years younger than they really . This condition Fournier has designated as *infantilism*.

The first set of teeth in infants with hereditary syphilis have nothing characteristic about them; they show a lack of nutrition, a condition which may arise from many other morbid processes.

The second set of teeth, however, present certain characteristics. These characteristics are shown especially in the two middle upper incisors, in which the cutting edge of the tooth is worn away, leaving a convex surface with the convexity upward. The teeth are also apt to be somewhat far apart, and, as the child grows older, to assume a peg shape. The especial characteristics of syphilitic teeth were first described by Hutchinson. This peculiar shape of the teeth is not always present in syphilis, but when it appears it is certainly very suggestive of the disease. As was pointed out by Coleman, the dentist who examined Hutchinson's cases, in nearly every one of them there was a deficiency in the superior alveolar arch at the anterior portion, so great in some cases that when the jaws were closed the upper and the lower incisors did not come together.

I have already described the *onychia* which occurs as one of the earlier manifestations of hereditary syphilis. In the late form of syphilis another form of onychia is met with, characterized, according to Post, by a swelling at the base or the side of the nail, which becomes thickened, fissured, and brittle, with more or less deformity of the phalanx.

In the late form of syphilis a peculiar inflammation of the cornea at times appears. It usually begins with a cloudiness of the substance of the cornea, with ciliary congestion. The entire cornea in this way becomes clouded. The affection is not accompanied usually by pain, and does not show any special congestion of the conjunctivæ. Hutchinson says that it is always symmetrical, although at first it is apt to begin with one eye and later to attack the other. The interval between the two attacks may extend over several years. This disease is called *interstitial keratitis*, and may for a few weeks almost entirely abolish sight. It usually disappears under treatment without leaving any trace behind it. On the other hand, opacities are sometimes left and interfere with vision. The total duration of the disease varies from six to eighteen months. Interstitial keratitis, according to Post, occurs most frequently in female subjects, and is most common between the ages of ten and fifteen, although it may occur much earlier, and, according to Fournier, may even be met with at birth.

Complications may arise in the shape of iritis, choroiditis, and retinitis.

Disturbances of hearing may occur from a number of causes, especially as secondary to diseases of the pharynx. An especial form of *deafness*, however, without any special lesions to explain it, occurs in the syphilis of childhood, is usually intractable to treatment, and persists into later life. Extensive ulcerations produced by syphilis may occur in the *nose* and *pharynx* at any time during childhood.

Treatment.—The treatment of the lesions which usually occur in the retarded form of syphilis is essentially with iodide of potash, either alone or in combination with some mercurial. The iodide of potash should be ven at first in doses of 0.12 or 0.18 gramme (2 or 3 grains), and this dose ould be gradually increased to 0.36 or 0.6 gramme (6 or 10 grains), or en more, as children often tolerate this drug remarkably well, and large ses are usually indicated.

When iodide of potash is given in combination with mercury, you can gin with corrosive sublimate in doses of 0.0006 gramme ($\frac{1}{100}$ grain) and adually increase the dose. Corrosive sublimate is, however, so apt to use disturbance of digestion that I prefer to treat these cases by giving e iodide of potash uncombined with any other drug, by the mouth, and plying mercurial ointment to the skin.

The treatment of these later manifestations of syphilis must often be ntinued for long periods.

I have here, to illustrate the retarded form of syphilis, a girl (Case 222), thirteen years d. This case shows the importance of carefully reviewing the previous history not only the child, but also of its parents.

The mother has had only this child, has never had any miscarriages, has always been ell, and has never shown any manifestations of syphilis.

The father, so far as I can ascertain, until recently has lways been well and strong, and has shown no signs of syphi-is. About one year ago he began to have cerebral symptoms, which rapidly increased, were accompanied by paralysis, and were undoubtedly of syphilitic origin.

I was first called to see this child when she was suffering from a mild attack of appendicitis, which did not come to operation. At that time I noticed a peculiar conformation of the upper incisors, which made me at once suspect that I had under my care a case of hereditary syphilis. On further inquiry I learned that she had been treated some years earlier by an oculist for keratitis. The upper incisors, as you see (Diagram 7), are abnormally far apart and stunted in their growth. They are notched, as is also the left lateral incisor, which is peg-shaped and by its clearly-cut notch represents nore nearly than the others the characteristic syphilitic teeth. The right upper lateral incisor has a peculiar shape, the crown f the tooth coming down almost to a point. The other teeth re, as you see, in many places deprived of their dentine, and re in various stages of disorganization.

On recovering from the appendicitis the child remained in weak condition during the following year, looked sallow, and had continual headaches, which did not improve under the usual remedies. Treatment with iodide of potash has not only een followed by the disappearance of the headaches, but also as resulted in this healthy appearance of the child, who is erfectly well.

CASE 223.

Probably retarded syphilis.
Male, 7 years old.

Here is another illustration of what is probably he retarded form of syphilis.

This boy (Case 223) is seven years old, and is a negro. is mother, who is said to be white, has had two miscarriages. he history of the father is not known, except that he was a negro. The boy has never d any disease, except measles when he was two or three years old. There is no history of his ever having had any of the earlier manifestations of hereditary syphilis. When he was four years old he had what were described as epileptiform convulsions, and since then he has had three or four of these attacks. The attacks come on suddenly, and he is very somnolent after they have passed off. Ever since he was four years old his abdomen has been more or less distended. His appetite is good, his bowels are regular. He has lately been brought to the hospital to be treated for headache, a distended abdomen, and dyspnœa.

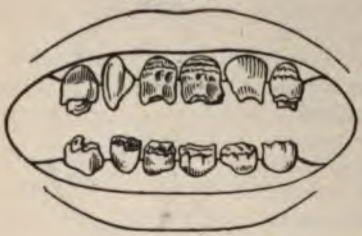
He has no enlarged glands, is not rhachitic, has no enlargement of the spleen and no ascites. The liver is found to be much enlarged, and, as you see, comes as low as the level of the umbilicus. Below the line of liver dulness the abdomen is resonant. The boy is mentally weak, and is very anæmic. On examination of his teeth you see that there are marked abnormal changes in the incisors. The upper four incisors are notched, dwarfed in size, and unnaturally far apart. The lower two middle incisors are also small and notched.

I have been treating this boy for the past month with hydrargyrum cum creta by the mouth and with mercurial inunctions. Under this treatment his general health has much improved, and he does not display the same degree of mental hebetude that he did on entering the hospital; he has also ceased to have the epileptiform attacks already referred to.

(Under the mercurial treatment the boy made a most decided improvement in his general health. The liver decreased in size, the digestion and appetite improved, and he gained steadily in weight and in mental development. He was discharged four months after entering the hospital, apparently perfectly well, except that there was still a slight enlargement of the liver.)

I have in this diagram represented twelve syphilitic teeth of the second dentition. They are all, as you see, more or less disorganized in a way which might occur from any cause which would interfere with the normal development of the teeth and cause their early decay. The middle two and left lateral upper incisors show the notched and somewhat peg-shaped condition which is supposed to be characteristic of syphilis, and which you see I have copied from the mouths of the girl and boy whom I have just shown you (Cases 222 and 223).

DIAGRAM 7.

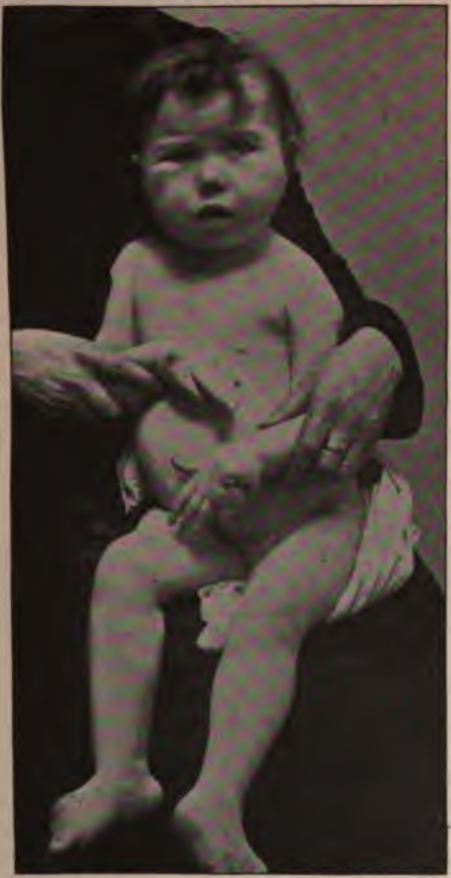


Syphilitic teeth of the second dentition.

As an illustration of these various tertiary lesions of syphilis, I will now show you a child who has been treated here in my clinic for some months.

It is a girl (Case 224), three and one-half years old. You will notice certain lesions on the face, arms, hands, and feet, which are the result of congenital syphilis. When this child was born it was apparently healthy. When it was three months old it was noticed to have occlusion of the nares, and at that time it had an attack of bronchitis lasting for three weeks. It is said that no efflorescence was ever noticed on its skin. When it was seven months old its hands began to swell, and at fourteen months the tissues around the metacarpal bones of the little fingers of both hands became reddened and ulcerated and the fingers assumed the pyriform shape characteristic of syphilitic dactylitis. When the child was about sixteen months old, the feet began to swell, and in certain parts, especially the metatarsal bones of the right foot, the skin became reddened. When the child was three years old, pieces of dead bone began to come away from the hands, and this has since continued. At this time also swellings began to appear over the upper maxillary bones, and, as you see, an extensive reddened and swollen condition of the tissues exists under the right fontanelles are closed. There are evidently a periostitis and an osteochondritis t arm, and there is also an enlargement of the left ankle, accompanied by ulcera- e outer side of the malleolus.

CASE 224.



Late manifestations of syphilis. Female, 3½ years old.

child has been treated with the combination of mercury and iodide of potash such in this prescription (Prescription 68):

PRESCRIPTION 68.

Metric.	Gramma.		*Apothecary.*	
rg. chloridi corrosivi . . .	0	03	℞ Hydrarg. chloridi corrosivi .	gr. ss;
ii iodidi	3	75	Potassii iodidi	ʒi;
estil.	60	00	Aq. destil.	℥ii.
			M.	

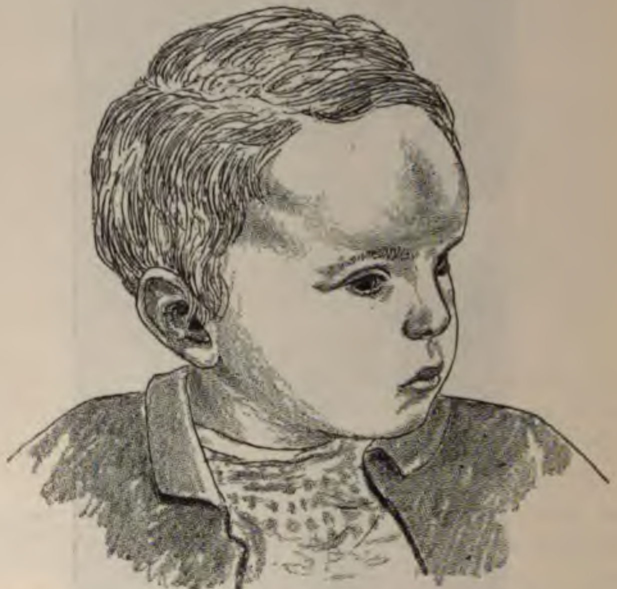
S.—2 c.c. (½ drachm) 3 or 4 times in 24 hours.

ve noticed that while it was taking this combination of drugs all its symptoms seemed better and brighter, and the lesions showed a tendency to heal. When- nedicine is omitted all the previous symptoms return. I should advise giving the h larger doses of the iodide than are contained in this prescription.

The following case illustrates, among other interesting points, this same lesion of the bones.

The boy (Case 225) is six years old. You will notice that he is rather pale, and that he has a somewhat peculiar frontal development, which well illustrates the form of syphilitic head to which I have already referred (Case 218).

CASE 225.



Hereditary syphilis. Male, 6 years old. Abnormal prominences of frontal bone.

You see the slight depression of the bridge of the nose and the bulging of the forehead on either side just above the orbital ridges. These prominences are accentuated by the deep sulcus between them, extending from the depressed nasal bones upward almost to the margin of the hair. This condition represents the typical syphilitic head.

The boy is in fair health, and I can detect nothing abnormal about him on careful physical examination. His mother brings him to the clinic by my direction to receive, now that he is entering upon the period of the second dentition, a course of mercurial treatment for a few months. Possibly some iodide of potash may be given with benefit.

He is a case of probable recovery from hereditary syphilis, as up to the present time he has practically been cured. The various lesions of the bones and in the organs which it is necessary now to guard against correspond to the later lesions of acquired syphilis, and hence my reference to the use of iodide of potash, which in conjunction with mercury is of great value in these later manifestations of syphilis, and will be given to him in the form of the stronger chloride of mercury in combination with the iodide of potash.

The mother of this boy first brought him to see me at the Children's Hospital when he was six weeks old. The mother had been well and strong, and had never had any other children nor any miscarriages. The father had had a primary syphilitic lesion one year previous to the birth of the child, which was followed by secondary manifestations. The mother had plenty of good breast-milk, and nursed her infant until he was nineteen months old. The infant was never atrophic, and though pale was apparently well nourished. At birth he showed a bullous efflorescence of medium grade. During the early weeks of his life he did not receive any medical treatment, although he had a general efflorescence of ed to be considerable pain in the arm, which made the infant restless and fretful. Hy- ia was a prominent symptom. The arm was put in a light splint, and the oleate of ury ointment (Prescription 66, page 499) was ordered.

The infant was then not seen for a week. On being brought back to the hospital the arm was found to be helpless, and the mother stated that the ointment had been dis- inued, as it caused excoriation of the skin. The ointment was then reduced one-half lanoline, and hydrargyrum cum creta was given three times daily in doses of 0.06 me (1 grain).

In three days the infant was much better, the paralysis soon disappeared, and nothing rmal was detected about the arms. The hydrargyrum cum creta was increased to 0.24 me (4 grains), but, as this caused diarrhœa, the dose in a few days had to be reduced to gramme (3 grains). In the course of the next month the nasal symptoms and the escence had disappeared, and the infant seemed perfectly well.

Six months later it was brought back to the hospital with a return of the condylomata a slight papular efflorescence. The same treatment as before was carried out. The ilitic manifestations disappeared, and have not returned since.

The child was kept under observation and treated from time to time for three or four . The first teeth were cut at nine months, and, as you see, are in fair condition to-day.

In connection with this case, and for the purpose of aiding you in your differential nosis where an apparent paralysis is present, I would state that the possibility of the lysis being a poliomyelitis anterior acuta was considered, but hardly seemed to explain ymptoms and the result, both arms being affected and entire recovery taking place three after the mercurial treatment was properly carried out. Pain, also, would not have present in a poliomyelitis. A central lesion was then thought of, but the rapid re- ry from the paralysis before the efflorescence or the occluded nares had begun to be ted seemed to show that such a lesion did not exist. The evident pain experienced by hild when the arms were touched, and the speedy disappearance of this sensitiveness, ell as of the paralysis, under mercurial treatment, pointed towards a lesion in the arms nselves. The infant did not choose to lift or use its arms, because moving them caused . No traumatic history could be obtained. Rheumatism occurring at six weeks of and affecting a child in this peculiar way would be very uncommon.

LECTURE XXIII.

ERYSIPELAS.

THE term erysipelas is applied to an inflammation of the skin, subcutaneous tissue, and mucous membranes which has the following characteristics. It especially involves the lymph-spaces and lymph-vessels. It has a tendency to spread, and is attended by unusual swelling of the subcutaneous tissue and an intense red color of the skin or the mucous membrane. In addition to these local appearances it is accompanied by constitutional symptoms, which are mostly the result of a heightened temperature.

It is caused by a micrococcus which is found exclusively in the lymph-spaces of the skin. This organism is a streptococcus, and in all probability is identical with the streptococcus pyogenes. The former belief that there existed a special organism which caused erysipelas has not been supported by recent investigations.

The disease runs an acute course, is contagious, enters the individual through some abrasion of the skin or mucous membrane, and is self-limited. The most careful and complete work which has been done in studying this disease is by Fehleisen.

PATHOLOGY.—According to Delafield and Prudden, the tissues may be swollen by an accumulation of serous fluid. This fluid may be nearly transparent, or may be turbid from admixture with pus-cells. The pus-cells may infiltrate the tissues either sparsely or in dense masses. Sometimes vesicles are found on the surface, or there may be crusts. Sometimes more or less of the affected region is filled with abscesses or becomes gangrenous. In some cases, aside from the local lesions petechiæ are found in the serous membranes, and swelling of the spleen and parenchymatous degeneration of the kidneys and liver. When the mucous membranes are affected they show the same appearances as the lesions of the skin, except so far as these are modified by the different structure of the tissue. The disease may attack the larynx and upper air-passages and may result in œdema. Pneumonia may occur as a complication.

Although the different organs, such as the spleen, kidney, heart, and liver, at times show pathological changes, nothing characteristic of erysipelas has been found in these organs, but only such changes as may occur from a continued high temperature or as the result of sepsis.

Erysipelas may be divided into two forms, (1) *migrans*, extending from surface to surface, and (2) *ambulans*, occurring in different parts of the skin. It may also be *acute* or *chronic*.

In erysipelas migrans, which is the most common form, the whole surface

body may be attacked. It is very prone to return, passing over the
urfaces of the skin again. The face and head are not so commonly
d in infants as in adults, and the disease seldom spreads from another
the body to the head. When it does attack the head, it is apt to be
om a secondary purulent meningitis. It at times causes great swell-
l tension, and may go on to gangrene in certain localities, such as the
n.

ter the first year erysipelas so closely resembles the disease as it occurs
lts that we need not consider it in this later period of life. It is a
hat frequent disease in infants up to six months of age. It then be-
less frequent up to the first year, and after that and in childhood is
rare. I shall, therefore, speak of erysipelas as it affects infants only.

e erysipelas of infancy may be divided into (1) erysipelas of the new-
nd (2) erysipelas of sucklings.

YSIPELAS OF THE NEW-BORN.—Where erysipelas occurs
the end of the third week the infant seldom lives, and indeed it is a
langerous disease up to the end of the third or fourth month. Ery-
of the new-born is apt to occur during an epidemic of puerperal
If the mother has any septic symptoms, the infant should be imme-
taken away from her. I have seen a case where the mother had
ral peritonitis following her delivery and where the infant (Case 226),
as allowed to nurse her, was attacked by erysipelas.

many cases occurring in the early days of life the disease starts on
nitals, and may be complicated by other diseases, such as empyema
pecially pneumonia. During the course of the disease the fontanelle
the spleen is enlarged, convulsions may occur, and peritonitis accom-
by vomiting may arise as a complication. The disease is liable to
the tissues at any point of abrasion, whether from the forceps or
accination, or at the point of separation of the umbilical cord. The
is the most common locality for the infection to take place. From
int the infection may extend and produce a gangrenous condition of
mach or abdomen.

though the temperature in the early hours or even days of the disease
ot be raised, yet, as a rule, fever soon appears, the temperature varying
39° to 41° C. (102.2° to 105.8° F.). Reddening and swelling, not
igh grade at first, appear on the parts affected. The infants show
oms of a general sepsis. Vomiting frequently occurs, followed by
e and almost without exception by death.

EATMENT.—The treatment of this severe form of erysipelas is by
ants and a substitute food adapted to the infant's digestion.

YSIPELAS OF SUCKLINGS.—The stage of the *incubation* of
elas lasts, according to Osler, from three to seven days.

hen the disease occurs in the early months of life, its beginning is
y accompanied by cold extremities and collapse. The temperature is
and the higher its degree the graver the prognosis. The temperature

curve, as a rule, shows a zigzag course, except in the more severe forms, where there is continued high fever with which icterus is apt to be combined.

The efflorescence, although very similar to that which is seen on the adult's skin, differs somewhat on account of the more delicate structure of the infant's skin. It begins as a faint erythema, which spreads rapidly and as quickly disappears, perhaps in twenty-four hours, and twenty-four hours later desquamation may occur. The light color of the efflorescence soon becomes darker and more intense, and is accompanied by swelling, heat, and tension of the subcutaneous tissue. After the efflorescence has continued for a certain number of days, depending upon the amount of the surface of the skin involved, the extension of the disease ceases and the temperature falls. The redness gradually disappears, and the skin becomes covered with yellowish-brown crusts. Finally, desquamation takes place, and the skin recovers its normal appearance, the disease extending over a variable period according to the greater or less extent of the surfaces invaded.

Although the disease when involving large surfaces is dangerous, yet cases in the later months of infancy recover even where the attack has been a severe one. An instance of this kind came to my notice where an infant ten months old was attacked with erysipelas, the point of infection being the right labium.

In this case (Case 227) the whole vulva shortly became very tender and the disease extended to the pubes and abdomen. It invaded every part of the body and extremities and the head and neck. The eyelids and lips were the last points of attack. Even the palms of the hands and soles of the feet were affected. From the time that it appeared at one part of the body until the skin of that part assumed its normal color again was four days. When the erysipelatous inflammation extended to the feet there was marked œdema. The duration of the attack from its first appearance at the vulva to its disappearance at the eyes and mouth was about fifteen days. The infant was treated with small doses of iron and quinine, and recovered entirely.

TREATMENT.—No treatment of which I know is of any avail in cutting short the disease. Where large surfaces are affected, the application of cold compresses tends to depress the vitality of the infant, which it is so important to sustain. During the height of the disease the infant's strength should be supported by stimulants and by the frequent administration of a food adjusted to its digestion.

I have here an infant (Case 228, Plate VI., facing page 504) six months old which represents the typical efflorescence of the erysipelas of sucklings.

It is a female, has always been healthy, and was nursed by its mother until within the last three weeks, when it was weaned from the mother and nursed by another woman. It is of normal weight and general development.

The first symptoms which were noticed were that it began to vomit and to have a raised temperature, 39.5° C. (103.5° F.) in the axilla. It seemed weak and languid, looked badly, and refused to take the breast. An examination of the breast-milk showed a peculiar green color, which not only appeared in the milk when drawn from the breast, but also, when the analysis was made, appeared in the curd resulting from the precipitation of the proteids.

The analysis (Analysis 60) of the milk was as follows. The nature of the micro-organism which produced the green color was not determined.

ANALYSIS 60.

Fat	4.56
Sugar	6.36
Proteids	3.46
Ash	0.13

Later in the day a pink efflorescence appeared just above the pubes, and there was found o be considerable irritation in the neighborhood of the vagina. The redness extended from he vagina to the supra-pubic efflorescence. The efflorescence was of an erythematous type.)n the following day it spread to the left thigh, and then to the left lower leg. The temerature continued to be raised, and the infant refused to nurse. Small quantities of a ubstitute food with the following percentages (Prescription 69), which had to be varied from lay to day, were given to it:

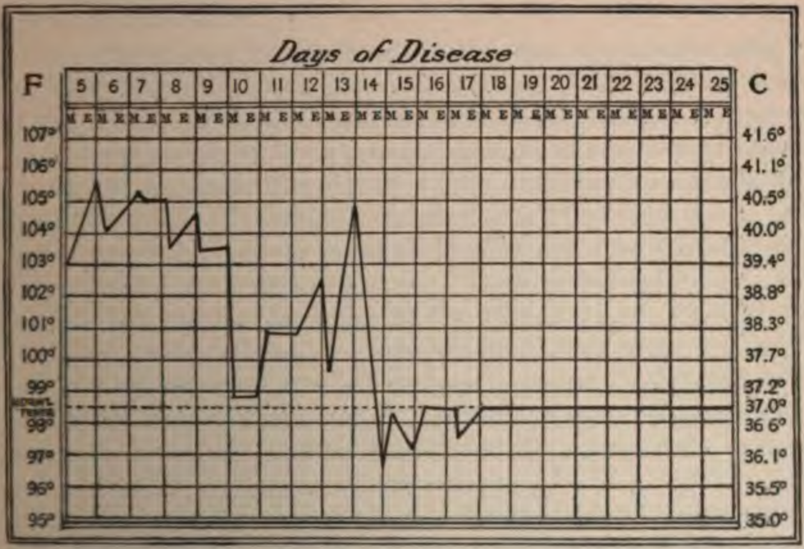
PRESCRIPTION 69.

Fat	2.50
Sugar	6.00
Proteids	1.50

There were no convulsions or other symptoms, but the infant lost somewhat in strength nd weight and its face looked pinched.

The efflorescence on the left leg began to fade on the ninth day of the disease, and on he tenth day the temperature became almost normal. On the following day, however, it gain rose, and a fresh efflorescence began to appear on the right thigh continuous with the efflorescence of the supra-pubic region. This efflorescence extended down the right leg to the ankle, and you see the condition of it to-day.

CHART 10.



Erysipelas of legs. Female, 6 months old.

You will notice certain points in regard to the efflorescence on the left leg. A slight amount of redness is present, but it has mostly disappeared, leaving the skin in parts in an almost normal condition, in other parts covered by thin brownish-yellow crusts. The supra-pubic region and the right leg as far as the ankle are, as you see, covered with a bright red efflorescence sharply bounded by normal skin below, just above the ankle, as though it were a stocking. The whole leg is swollen, is hotter to the touch than the sound skin, and presents a somewhat raised, glistening appearance.

No external applications and no drugs have been employed in this case. The milk has been carefully modified, and small doses of brandy have been given.

(The subsequent history of the case is as follows. A few days later the temperature became normal, the efflorescence began to fade, desquamation subsequently took place, and the skin finally recovered its normal appearance. The infant gradually regained its strength, became perfectly well, and has had no return of the disease.)

Here is the chart (Chart 10, page 515) showing the temperature during the course of the erysipelas in this case.

This form of erysipelas may become chronic, and this is more apt to occur in children than in infants. It is also most common in children who are in a debilitated condition, and may occur at intervals of three or four years. It is frequently in older children connected with chronic inflammations of the Schneiderian membrane, and in these cases is peculiarly intractable to treatment.

LECTURE XXIV.

THE EXANTHEMATA.

VARIOLA—VARICELLA.

ntradistinction to the various diseases of the skin which dermatolo- accustomed to designate as exanthems of local origin are certain cific, infectious diseases which they call the exanthemata. This ases is of especial interest in connection with diseases which arise n, as it is among children that they most frequently occur. They ver, attack individuals of any age. Although none of these dis- entirely self-protective, yet the instances in which they develop in dual more than once are rare.

xanthemata comprise five diseases,—variola (small-pox), varicella pox), scarlet fever, measles, and rubella. In regard to the latter question whether it is a disease distinct from measles.

group of diseases is characterized by certain conditions common Besides being infectious, each disease runs a definite course and is ed, facts which should be remembered when we are studying its and treatment.

ourse of these diseases from the time when the infection takes place appearance of their later manifestations may be divided into dis- es. In the first of these certain micro-organisms are supposed to system, and, so far as external appearances and general symptoms rned, to remain dormant for a time, constituting what is called the *ncubation.* This stage of incubation is followed by certain general s resulting from the supposed development of the special organisms tituting the *prodromal stage.* These prodromal symptoms are, rvals varying according to the special disease, followed by an ce on the skin, which marks the third stage of the disease, called *of efflorescence.* The efflorescence in its turn is followed by what the *stage of desquamation,* this desquamation being more or less ed in proportion to the intensity of the lesions of the skin which rred during the stage of efflorescence.

ugh in a large number of cases the diagnosis of these diseases can ined by the appearance of the efflorescence and its location, yet occur not infrequently where the efflorescence is very misleading. d, therefore, be familiar with the characteristics of the other stages diseases, for it is by carefully considering the pictures which they us as a whole that we are enabled to make a correct differential of the especial case. Thus, a papular efflorescence, although significant in most cases of measles, may also be present in any other member of the group, while an erythema closely resembling scarlet fever may occur in variola, measles, or rubella.

VARIOLA (Small-Pox).—The first disease of this group which I shall speak of is variola. Variola is one of the most virulent of the infectious diseases with which we have to deal, and is particularly fatal among infants and young children. It is an acute disease, caused evidently by a micro-organism. It is characterized by severe constitutional symptoms, accompanied by a progressive efflorescence from macules and papules to vesicles and pustules, followed by the formation of crusts, these lesions having a tendency to result in cicatrices. As I have stated to you in a previous lecture (Lecture V., page 147), since vaccination has been established, variola, in contradistinction to varicella, scarlet fever, and measles, is an extremely rare disease among infants and young children who have been vaccinated.

Although there are no characteristics of variola which are distinctive in children from those of the disease occurring in adults, it is important to recognize its chief features for the purpose of differential diagnosis. It is possible for the fœtus to contract the disease in utero. This, however, is rare, and it is well known that infants whose mothers are affected with variola can, even when born in small-pox hospitals, be protected from the disease if vaccinated immediately. It is rather remarkable that the micro-organism which causes variola has never been discovered, when we consider for how long a time the disease has been known to be highly infectious. The contagium is supposed to exist in the secretions and excretions, and to emanate from the exhalations of the lungs and from the skin. It is in all probability transmitted principally by means of particles of the crusts. It has a wonderful tenacity for clothing or any like means of conveyance. It has been proved that the contagium is active before the efflorescence occurs, though not so much so as later. It has also been fairly well proved that its activity ceases when all the crusts have fallen off and when the entire skin has become smooth. The most virulent form of the disease can be contracted from a mild form, such as varioloid.

PATHOLOGY.—The pathological conditions found in variola are chiefly those of the skin and the mucous membranes.

According to Weigert, the progressive changes of the lesion of variola are as follows. The lesion begins as a round, somewhat raised macule. This develops into a hard papule, and later a small vesicle arises on its summit. This vesicle enlarges very rapidly and changes to a tensely filled pustule with a central depression. The size of this pustule corresponds to that of the original macule. Microscopically the macule consists of a circumscribed spot of hyperæmia in the capillary layer of the skin. The papule is formed by a sharply defined necrobiotic degeneration of the under layers of the rete mucosum, by which process the nuclei of the epithelial cells are destroyed. By the transudation of fluid into these areas the cells are pushed apart and the epithelial layer is lifted up as a whole, covering the area affected, and forms a vesicle the inner part of which is composed of a mesh-work filled with lymph. In the vicinity of the necrobiotic focus an inflammation is set up, causing an increased growth of the cells of the rete which surround and wall in the focus on all sides. The developed pustule extends through the whole thickness of the cutis to the subcutaneous tissue. A net-work inside the pustule, which is most tense in the central part, connects the roof and floor of the pustule, and, in conjunction with the above mentioned growth of the cells of the rete around the focus, causes the central depression. If the vesicle is pricked, only a part of the lymph flows out of the mesh-work within. The lymph is clear, and contains some white and red blood-corpuscles, streptococci and staphylococci, fibrin-flocculi, and molecular granules. The contents of the pustule are purulent, and those in the hemorrhagic form contain blood. Clumps of bacteria with analogous localized degeneration and its associated changes are found in the neighborhood of the pustules, also in the parenchyma of the internal organs and lymph-glands, as well as in the skin. When the variola has reached its height the central depression in the pustule disappears, because the increased tension in the contents tears away the mesh-work. The vesiculation begins in the upper central part and spreads downward towards the periphery. The pustule then collapses and changes to a crust, which after a certain number of days falls off, leaving a more or less deep scar covered with young epithelium. A distinct difference in the anatomy of a pustule of variola vera and one of varioloid does not exist.

On the mucous membranes of the mouth, nose, conjunctivæ, bronchi, œsophagus, rectum, sometimes the vagina, and also on the tonsils and the tongue, the same pustular efflorescence may be found, and is either superficial or extends more deeply. At times also a pseudo-membrane is found on the ulcers.

According to Osler, the papillæ of the true skin below the pustules are swollen and infiltrated with embryonic cells to a variable degree. If the suppuration extends into this layer, scarring invariably results; it does not necessarily follow if the suppuration is confined to the upper layer.

In the intestines swelling of Peyer's follicles is not uncommon. In the larynx the efflorescence may be associated with a fibrin exudate, and sometimes with œdema sufficient to cause death. Occasionally the inflammation extends deeper and involves the cartilages. In the trachea and bronchi there may be ulcerative erosions, but the characteristic lesions seen on the skin do not occur. There are no special lesions of the lungs, but congestion or broncho-pneumonia is very common.

According to Gardner, in addition to the conjunctiva almost every part of the eye may suffer, the lids, lachrymal sac, cornea, choroid, and even the retina and extrinsic muscles.

These complications may occur either during the course of the disease or afterwards.

According to Adler, keratitis may develop from a purulent conjunctivitis, or quite independently of it, never, however, earlier than the twelfth day. It may occur as a circumscribed superficial inflammation which, even under atropine and hot fomentations, may take the form of an ulceration very dangerous to the eye.

In the ear, according to Wendt, complications are more frequent than in the eye. The milder forms of hyperæmia are generally overlooked, as they cause no symptoms. Congestion of the middle ear is common, and is generally directly due to swelling of the naso-pharyngeal mucous membrane closing the Eustachian tubes. Sometimes this progresses to acute inflammation of the middle ear, which may end in extensive destruction of the soft parts, with subsequent permanent deafness.

According to Osler, in exceptionally rare cases the eruption extends down to the œsophagus and even into the stomach.

The pathological changes in the other organs consist of enlargement of the spleen and fatty degeneration of the liver, kidneys, and heart. Metastatic processes in the various organs and in the joints sometimes occur. In the hemorrhagic form hemorrhages in the various cavities in the different organs, and, according to Golgi, in the medullary cavities of the bones, may occur, also in the serous and mucous surfaces and in the muscles.

INCUBATION.—The incubation of the disease varies from twelve to fourteen days, the latter being the most frequent period.

SYMPTOMS.—According as the symptoms of variola are mild or severe the disease has been divided into a number of forms, designated as follows: (1) *discrete*, (2) *confluent*, (3) *hemorrhagic*, and (4) *modified*. In all these forms the initial fever, convulsions, and general symptoms may be severe, and do not necessarily indicate which type of the disease is about to follow.

(1) DISCRETE.—The mildest and most typical form of the disease is that which is called *discrete*.

Prodromata.—In this form, the invasion, though sometimes less severe than in the confluent and hemorrhagic forms, as I have just stated, in infants and young children is almost always of a grave type. In infancy and early childhood the disease commonly begins with convulsions. There may be vomiting, great restlessness, quick pulse, high temperature, and in a number of cases the children quickly succumb to the disease from the virulence of the toxæmia. If they survive this early stage of the disease they usually present the same sequence of symptoms as in cases occurring in later life, but may eventually die from the exhaustion which often rises from a prolonged suppurative fever. In the prodromal stage the pulse is much quickened and the temperature may be as high as 40°, 40.5°, or even 41.1° C. (104°, 105°, or 106° F.). In this stage we at times, especially among children, meet with an evanescent erythematous efflorescence. According to Simon this manifestation is distinct from that of scarlet fever. It has a peculiar distribution and generally a limited extent, usually affecting the lower abdominal areas, the inner surface of the thighs, the sides of the thorax, and the axillæ; sometimes, however, it involves the whole surface. This efflorescence is distinct from the typical lesions of variola which occur later.

Efflorescence.—On the third or fourth day of the prodromal symptoms an efflorescence appears on the skin, and at this time the frequency of the pulse lessens, the temperature usually falls considerably, and the more severe symptoms improve, so that the patient appears much more comfortable. The efflorescence is at first represented by small red macules or papules, which, as a rule, first appear on the forehead, or on the face and mucous membranes, and later on the trunk and limbs. The papules are rather scattered in their distribution, and have a feeling as of shot under the skin. The macules when present soon become papules. On the third day by means of a good light a small vesicle can be seen at the apex of the papule, and by the fifth or sixth day the vesicular stage is well established and the vesicle becomes distinctly umbilicated. This appearance on careful examination can also be seen in the lesions of the mucous membranes. At about the eighth day the vesicles become pustules, the tops soon flatten, and the umbilication disappears, leaving an areola of injection and the intervening skin swollen.

The temperature at this time rises, from the suppuration which is taking place in the pustules. This rise of temperature is called the secondary fever, or fever of suppuration. The temperature remains high for from twenty-four to forty-eight hours, and then gradually falls until by the twelfth or thirteenth day it usually becomes normal. The contents of the pustules dry up, and crusts are formed. On the palms and soles small hard disks form, which may of themselves fall off in infants, but in children as old as ten years would remain for a long time unless removed with the point of a knife.

Desquamation.—By the fourteenth or fifteenth day the stage of desquamation is established. In some cases extensive scars are left on the skin where the crusts have fallen off. This is most apt to occur in severe cases.

(2) CONFLUENT.—In contradistinction to the mild or discrete form of variola is the more severe form, called *confluent*, on account of the tendency of the lesions to coalesce. In the confluent form of variola the efflorescence usually appears at the same time as in the discrete form. At about the fourth day the lesions become confluent, the skin becomes reddened and swollen, and the face may be much distorted by the severity of the lesions. In this form the initial temperature does not fall to the same degree as it does in the discrete form, and, according to Sydenham, diarrhœa is likely to occur, particularly in children. The pharynx and larynx are especially apt to be involved, and the cervical lymphatics to be enlarged. The crusts adhere longer in the stage of desquamation than they do in that of the same stage of the discrete form.

(3) HEMORRHAGIC.—The third or *hemorrhagic* is the most virulent form of variola, and may occur in children as it does in adults, though not so frequent in the former as in the latter. Its symptoms in children are so severe that in almost every case it very quickly proves fatal. It is charac-

terized by punctiform hemorrhages in the skin, appearing from the first to the fourth day of the prodromal stage, ecchymoses in the conjunctivæ, and hemorrhages from the mucous membranes. According to Osler, hæmaturia is the most common form of hemorrhage, hæmatemesis the next.

(4) MODIFIED FORM.—The fourth or *modified* form of variola is where the disease attacks individuals who have been successfully vaccinated. This form is called varioloid, but would be better termed "modified small-pox." Modified small-pox is usually much milder in its symptoms than any of the other forms of variola, although the initial fever may be as high as in a severe case. The papules are fewer in number, the temperature becomes normal sooner, and the child seems comfortable in a shorter period of time, since there is usually no secondary fever from suppuration. The nearer the attack comes to the time when the child was vaccinated, the less severe will be the symptoms.

In any of these forms of variola the prodromal symptoms may be of a very severe nervous type, and this is especially characteristic of the disease as it occurs in children. For this reason variola may simulate other diseases in its prodromal stage, and may often cause death before the efflorescence has appeared. This is especially the case with the prodromal symptoms of the hemorrhagic form.

COMPLICATIONS.—The most common complications of variola are those of the larynx and the lungs. Where the larynx is affected, œdema of the glottis may suddenly arise and death take place from suffocation.

In the throat the presence of the efflorescence occasions great irritation and the accompanying secretions cause nausea and at times dyspnœa, with a cough which in weak children is very exhausting.

Where acute inflammation of the middle ear has taken place the pain during the formation of the pus is very intense, but it subsides as soon as the sac bursts or is incised. This complication, therefore, requires early and careful treatment.

Where a lesion of the lung develops, it is usually in the form of a broncho-pneumonia. Lobar pneumonia rarely complicates the disease.

Although albumin is very frequently present in the course of the disease nephritis is rare.

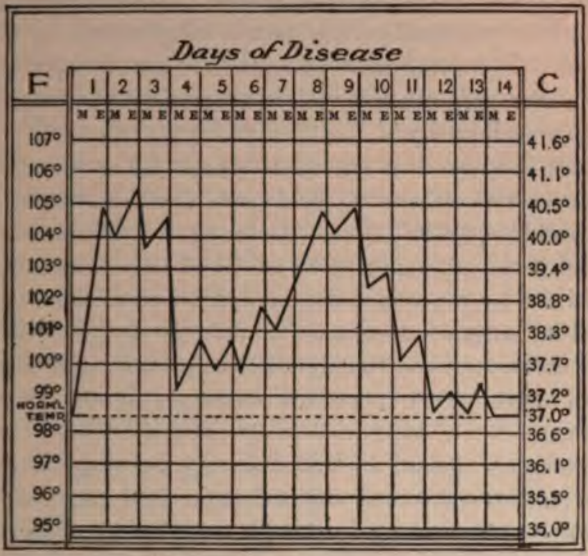
This chart (Chart 11, page 523) represents the usual temperature curve of the initial fever and suppurative fever of a typical case of variola.

DIAGNOSIS.—There is no other constitutional disease accompanied by an efflorescence on the skin which in a typical case would be likely to be mistaken for variola. The severe constitutional symptoms, the slowly developing and rather scattered macules and papules, with the shotty feeling of the latter, the umbilicated vesicles gradually becoming pustules, the extensive crust formation, and the initial and suppurative fever, all render the diagnosis in most cases quite plain.

In making the diagnosis of variola we should consider that the disease differs materially in its prodromal symptoms from varicella and measles.

The almost complete absence of prodromal symptoms in varicella, and the pronounced catarrhal symptoms of the nose and eye in measles, make the differentiation from these diseases comparatively easy. Although the prodromal symptoms of scarlet fever and of variola are often of equal

CHART 11.



Fever of invasion. Fever of suppuration.
Variola.

everity and somewhat similar, such as the convulsions and vomiting, yet the pronounced symptoms connected with the throat in scarlet fever, and the appearance of an erythematous efflorescence instead of the scattered papules of variola, serve to differentiate clearly the two diseases. We must, however, be careful not to mistake the evanescent efflorescence which I have already referred to as occurring in the prodromal stage of variola for the erythema of scarlet fever. The distinction can usually be made by remembering that this efflorescence in variola affects the particular areas of the skin already referred to, and that these areas in scarlet fever, measles, and varicella are unlikely to be affected early in the stage of efflorescence. The typical location of the efflorescence of scarlet fever is first on the neck and chest, that of measles on the face, and that of varicella on the back, face, and head.

In making the diagnosis of variola we must, of course, bear in mind the efflorescence which appears on the skin as the result of *vaccination*, and that which occurs in the course of the disease *vaccinia*. In vaccination the single lesion and the absence of severe constitutional symptoms make it hardly necessary to do more than refer to it in this connection. The differential diagnosis from vaccinia is not difficult, and yet this disease is so rare that when it appears it almost always creates a suspicion that we may be dealing with variola. As a rule, in vaccinia the general symptoms are not severe, the disease being represented almost entirely by a slight malaise and loss of appetite, in conjunction with the appearance on the third or fourth day of an efflorescence on the skin. This efflorescence, as I have stated in a previous lecture (Lecture V., page 152), is represented by papules, vesicles, and pustules, few in number and irregularly distributed, some on the face and nose and a few on the body and extremities. As the disease almost invariably appears after vaccination, this fact is of great aid in differentiating it from variola. The subsequent course of a case of vaccinia is so much milder and shorter than that of variola that in a few days the differential diagnosis can be made easily.

TREATMENT.—There is no specific treatment for variola, but it is of the utmost importance that the best hygienic care should be employed. The air of the room should be perfectly fresh. The crusts should be kept softened with a mixture of glycerin, oil, and carbolic acid, and the odor arising from them should be modified by the application of a dilute solution of carbolic acid.

In the initial stage of the disease stimulants should be freely given if the symptoms are severe, and the high temperature should be controlled by sponging with water at a temperature corresponding to the power of the child's reaction.

The greatest care should be taken during the stage of convalescence, and when the child is considered well the most rigid measures for preventing the spread of the contagium should be enforced. The clothing and everything connected with the child and its attendants, and the room in which they have been kept during the sickness of the child, should be thoroughly disinfected, the same precautions being taken to prevent the spread of variola that I shall presently describe to you in speaking of scarlet fever (Lecture XXV., page 549). The immediate transferrence of a patient from its room to a small-pox hospital is in most communities considered the wisest method of dealing with the disease, and is usually enforced by law.

VARICELLA (Chicken-Pox).—The next member of the group of exanthemata which I shall speak of is varicella. It is the mildest in its symptoms and the most favorable in its prognosis of the whole group. It is highly infectious, and is characterized, in distinction from the other exanthemata, by its long stage of incubation, the shortness or absence of the prodromal stage, vesicular efflorescence, and absence of sequelæ. Varicella has been known as an independent disease for the last two centuries. At one time it was not clearly differentiated from measles and scarlet fever, and in some parts of the world it is supposed to be closely allied to variola. This opinion, however, is not generally substantiated, and we can accept varicella as a distinct disease.

It can occur at any age, but the most common time for its appearance is in the middle and latter part of the first year. It continues to be a common disease all through the early and middle years of childhood. The susceptibility to the contagium of varicella lessens after ten years of age, and almost disappears at puberty. It is sometimes sporadic and sometimes epidemic.

t occurs with equal frequency at all periods of the year. The vehicle of ontagium is not known, but it probably enters the system by the lungs. he specific organism which produces varicella has not yet been determined.

Pathology.—Deaths from varicella are so extremely rare that our nowledge of the pathology of the disease is necessarily limited. It is vident, however, that the efflorescence of vesicles, which represents the rincipal morbid lesion of the disease, is of a somewhat different type from hat which occurs in variola. The vesicle is much nearer the surface than n the latter disease, being formed mostly by the upper layers of the pithelium. The vesicle itself is seldom multilocular, a condition which is requently present in variola. The contents of the vesicles are usually a lear serum, the progression to a pustule being rare in comparison with the sion of variola. The lesion so rarely involves the deeper layers of the kin, and the process is usually so very mild, that it is seldom that suffiient destruction of the tissue takes place to produce a scar.

The lesions may appear on the mucous membranes as well as on the kin. At times the lesions assume a much more serious form and may ecome gangrenous. In *gangrenous varicella,* according to Eustace Smith, he vesicles, instead of drying up in the ordinary way, become black and arger, so that a number of rounded black crusts are scattered over the urface of the body. If a crust be removed, it is found to cover an ulcer nore or less deep. Around it the skin is of a dusky red color. All the esicles do not become gangrenous, so that we find crusts of the ordinary ppearance mixing with the blackened crusts. The gangrenous process ften penetrates deeply through the skin to the muscles. The lesions at imes are so extensive as to form ulcers which may invade and destroy arge areas of tissue.

Incubation.—The stage of incubation is variable, but lasts from eight r ten days to three weeks, the usual time being about seventeen or eighteen ays.

Symptoms.—**Prodromata.**—There are rarely any prodromata in variella, beyond a slight malaise for a few hours. At times, however, especially n young infants, the onset of the disease may be severe: it may be characerized by vomiting, and, where the temperature is high, even by convulsions. n rare cases the prodromal stage is of considerable length and the prodronata resemble somewhat those of the other exanthemata.

Efflorescence.—The disease usually shows itself in the form of an fflorescence, the characteristic and most common lesion of which is a esicle. The lesion, however, is in the beginning a macule, which quickly ecomes a papule, and the papule so rapidly develops into a vesicle that it s in the vesicular stage that we usually first notice the efflorescence. These nacules and papules are so superficial that they are soft to the touch and do ot give the shotty feeling which is so common in these lesions when they ccur in variola. The vesicle of varicella, as a rule, is not umbilicated, nd but rarely do its contents become pustular. It may be surrounded by a light red areola, but this is not present in all the lesions. The usual course of progression in the lesions is that the vesicle flattens, its contents are dispersed on the skin or absorbed, and a small crust is formed, which finally falls off, leaving the skin smooth and without a scar. Occasionally a scar results from some individual lesion in which the inflammatory process has involved the deeper layers of the skin. The efflorescence is irregular and general in its distribution, the lesions appearing on the face and head, in my experience especially behind the ears, on the body, usually first on the back, and finally on the extremities. It comes out in successive crops, so that very different lesions may be found on the skin at once, representing the early and late manifestations of the efflorescence. It, however, may first appear in the throat, but is not so often seen in this location as is the efflorescence of scarlet fever or measles. It is possible that the efflorescence always appears first in the throat, but that in many cases it is not seen early enough to be recognized, as the manifestations are very evanescent.

This efflorescence of varicella is almost the only one which is characteristic of a specific disease. By this I mean that while a vesicle does not necessarily allow us to diagnosticate any disease of the skin, yet when these vesicles with their areolæ, in combination with constitutional symptoms, appear in groups in different parts of the body, there is no other disease with which we should be likely to confound it, with the exception of variola, vaccinia, and possibly herpes zoster.

The course of varicella is rapid. It is characterized by a sudden onset of constitutional symptoms, with the almost immediate appearance of the efflorescence. The efflorescence runs a rapid course, appearing quickly on different parts of the skin, and disappearing almost as quickly as it appears. The disease lasts about a week or ten days, and, as a rule, has no serious sequelæ. It is rarely complicated by any other disease.

COMPLICATIONS.—During the course of certain epidemics, however, it has been noticed that the kidney is affected. This complication usually occurs after the efflorescence has almost disappeared, and in the second week from the time of the beginning of the attack. In these cases albuminuria is present, and in all probability is caused by some form of nephritis, although nothing definite is known about this class of cases.

GANGRENOUS VARICELLA.—A complication which at times arises in varicella is what is called the gangrenous form of varicella, the pathology of which I have already described. Although it is most common in ill-nourished children, yet it does not necessarily attack this class of cases, and it seems to have some connection with the gangrenous processes which certain individuals show a tendency to develop.

PROGNOSIS.—The prognosis of varicella is usually, unless the above-mentioned complications arise, extremely favorable. Cases occur where the prognosis is rendered unfavorable by lack of proper care during the convalescence, resulting in broncho-pneumonia and other diseases. In some cases ognosis is rendered unfavorable by the anæmia which is apt to follow ck of varicella, and is at times pronounced.

AGNOSIS.—The diagnosis of varicella is not difficult if we bear in he characteristics of the diseases which it is most apt to simulate.

differentiating it from variola we must consider the great difference rapidity of the development of the efflorescence in the two diseases. iola it is essentially slow, in varicella it is characteristically quick. apules of variola are hard to the touch, those of varicella are soft. sicle of variola, as a rule, is umbilicated and soon becomes a pustule; haracteristics are absent in varicella. The whole course of variola s a period of from two to three weeks; the course of varicella is shorter, and is often limited to one week. Finally, the severe con- nal symptoms and the long prodromal stage in variola differ essen- from the lack of prodromata and the mild constitutional symptoms in la.

vaccinia the slow progression of the lesions from papules to pustules, e rather limited areas affected, serve to distinguish it from the succes- ops of vesicles, with their rapid development and extensive areas, are met with in varicella.

e differential diagnosis of varicella from herpes zoster is not difficult, consider that the vesicular efflorescence in herpes zoster follows the of some set of nerves, while that of varicella is perfectly irregular in no way connected with the distribution of the nerves.

this table (Table 93) I have arranged the chief points of difference n varicella and variola:

TABLE 93.

	Varicella.	Variola.
ion	Two to three weeks.	One to two weeks.
nata	None or slight.	Three to four days in length. Active. Severe.
ence	On the skin. Rapidly becomes vesicular. Not umbilicated. Unilocular. Irregular. Numerous. Universally distributed in successive crops. Vesicles differ greatly in size On pricking, collapses entirely.	Under the skin. A slow progressive development from a macule to a papule, from a papule to an umbilicated vesicle, then to a pustule. Multilocular. Regular. Not numerous. Defined in its localization. Lesions, as a rule, of uniform size. On pricking, collapses partially.
nation . . .	Slight crust formation.	Pronounced crust formation.
a	Short, one week to ten days.	Long, three to four weeks.
. . . . . .	Mild.	Severe.
ature	Irregular, not high.	Rises suddenly. Remains high until papules are developed, when it falls considerably. Rises again during the development of the pustules.

I have here a boy (Case 229) who was brought to the hospital a few hours ago, and who illustrates very well what I have told you concerning the efflorescence of varicella as it occurs in the throat.

He is said to have been well until yesterday, when towards evening he began to feel feverish, to have loss of appetite, and to complain of sore throat. He was brought to the hospital to be treated for a supposed cold. On examination nothing abnormal was found except these lesions which I shall show you in the throat, and a few vesicles behind his ears and on his back. These lesions on the skin have appeared since he came to the hospital, subsequent to those which were seen in his throat a few hours ago. On making the child open his mouth and depressing his tongue you will see certain lesions of the mucous membrane of the entire throat (Plate VIII., Varicella, facing page 781). The tongue, you see, is very slightly coated. The tonsils are not enlarged. The mucous membrane of the hard and of the soft palate and of the pharynx is slightly hyperæmic. On the upper and right side of the hard palate and very near where it joins the soft palate you will notice two small vesicles surrounded by a distinct red areola. To the left and below these lesions are three minute macules, two of which have almost become papules. You must remember that the difference between a vesicle and a pustule is simply one of degree. On the skin behind the ear and on the back you will notice that these lesions are purely vesicular. There are not so many leucocytes in the vesicles on the skin as are evidently present in the two lesions on the hard palate, which give the latter a yellowish color in contradistinction to the pearly white color of the dermal lesions.

This case illustrates very well the importance of making a thorough examination of the throat in children, which I have referred to in a previous lecture (Lecture XIII., page 323), for unless the throat had been examined the child would have been supposed to have a cold and would have been allowed to remain in the clinic and thus spread the contagium.

To illustrate still further the efflorescence of varicella, I happen to have in the isolating ward of the Children's Hospital a case in which the varicella is at its height and has been running its course for two days.

This child (Case 230, Plate VI., Varicella, facing page 504), a girl, was attacked with headache and malaise three days ago in the morning. In the afternoon an examination showed an efflorescence in the throat, but there was also a well-marked vesicular efflorescence on the back. This efflorescence soon began to come out in crops in different parts of the body, on the limbs, behind the ears, and on the scalp. There are also a few lesions on the face. Here on the back you will notice a number of lesions, some of which are simply macules, and again a few of the macules have become papules. In most cases, however, the lesions are distinctly vesicular, varying in their contents to such a degree that we sometimes see the pearly white appearance and again the yellowish color of a vesicle which has become somewhat pustular. In other places the vesicles have broken down and little crusts have formed in their centres, which are somewhat indented. On pricking one of these vesicles you see that it collapses and is emptied of its entire contents, showing that it is unilocular. The vesicle of variola when pricked in this way would in most cases be only partially emptied, showing that it was multilocular.

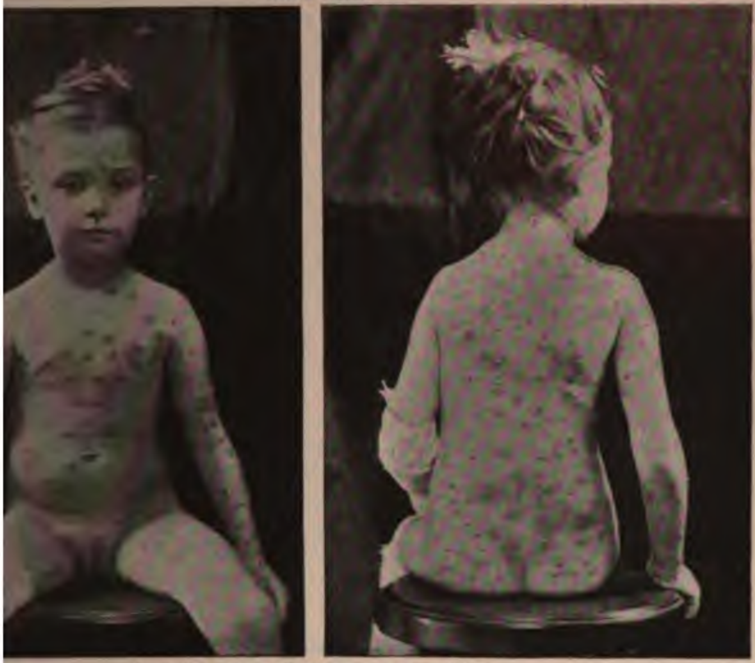
In this next bed is a little girl (Case 231) who shows the lesions of varicella in all these stages.

This child was brought from the surgical ward three days ago, and, as you see, was being treated with plaster-of-Paris bandages. The efflorescence, chiefly vesicular in character, first appeared behind the ears, and one or two of these lesions which have mostly run their course and have become crusts are, as you see, still present. The whole of the child's back is thickly covered with the efflorescence. The lesions are also on the arms, legs, and abdomen, and, although not so numerous on the front of the chest as on the back, they are very prominent in this area. The lesions have attacked the chin, lips, face, nose, and forehead, and can also be found on the scalp.

I shall order the plaster bandage to be removed, as a fixed bandage should never be

he course of any of the eruptive diseases, owing to the probability that exten-
ns will develop under them.

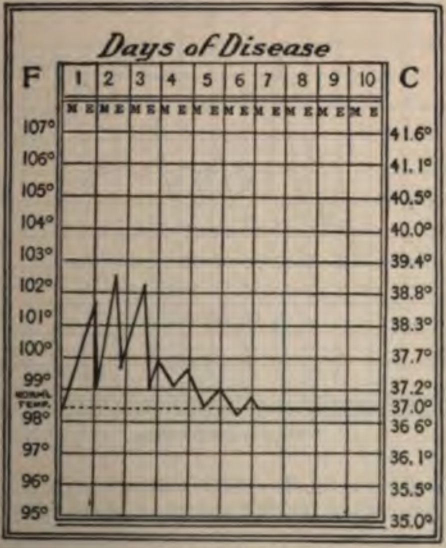
CASE 231.



Varicella. Stage of efflorescence, third day.

mperature in varicella is in most cases not high, and is very
It usually rises when a crop of lesions of any considerable
velops, and falls again at the outbreak. This chart (Chart 12)

CHART 12.



Varicella simplex.

shows the usual variations which you may expect to find in the temperature of varicella.

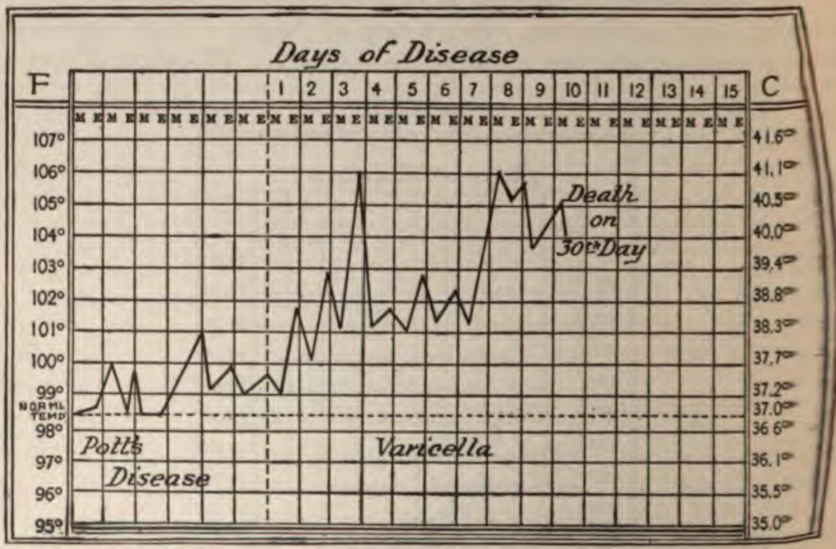
I have also here a child (Case 232), three years of age, who was brought to the hospital with Pott's disease, and with a paraplegia arising from a transverse myelitis caused by the disease. Nothing abnormal was found in connection with the lungs, heart, or kidneys. Until the child was attacked with varicella the temperature was usually normal, but sometimes rose to 37.7° C. (100° F.), and occasionally as high as 38.3° C. (101° F.).

About one month ago the child became restless, and his temperature rose somewhat. On the following day the symptoms became more marked, and the temperature was found in the evening to be 39.4° C. (103° F.). On this day an efflorescence of varicella appeared on his skin. During the third day of his sickness his face swelled, and in the evening his temperature was found to be 41.1° C. (106° F.). The vesicular efflorescence was well developed on his trunk and face by this time. Somewhat later it became universal and assumed a purulent character, especially about the face. During the fourth, fifth, and sixth days of the disease his temperature varied in the evening from 39° C. (102.2° F.) to 39.4° C. (103° F.). On the seventh day of the disease all the symptoms increased in severity, and the temperature was found to be 41.1° C. (106° F.). On this day some of the vesicles on the face had become ulcers. Nothing abnormal was found in the lungs, and no albumin or casts in the urine. A psoas abscess developed during the progress of the varicella. The ulcers on the face, as you see, have extended to such a degree that the child has lost the sight of one of its eyes. The child is sinking rapidly. The treatment, which has been essentially with stimulants, has failed to keep up its strength, and the local treatment in connection with the eye has proved entirely unsuccessful.

This child represents a case of gangrenous varicella.

Here is the temperature chart (Chart 13) of the case.

CHART 13.



Varicella gangrænosa.

(Subsequent history of the case.) The child continued to grow weaker, and died on the thirtieth day from the time when the first symptoms of the varicella were noticed. No autopsy was obtained.

TREATMENT.—The treatment of varicella is simply symptomatic. The
ild should stay in the house, and its room should be kept at an even tem-
rature. The diet should be milk. The child should be carefully watched
prevent it from scratching, as lesions deep enough to produce scars may
en be obviated in this way. This treatment should be continued until
the constitutional symptoms have passed away and the efflorescence has
appeared. Complete isolation should, if possible, be enforced, as, although
disease is usually insignificant, we can never in the beginning determine
ether or not a rare and severe case is about to develop.

These rules for treatment are precautionary, and are based on the suppo-
ion that a child who has had a constitutional disease of this nature must
more sensitive to exposure of various kinds. As it is possible in some
es for the kidney to be affected in the later stages of the disease, just as
is in scarlet fever, it is well to guard against this complication by the
otection of the skin from changes of temperature and by the use of milk
a diet. In a considerable number of cases, especially in young children,
n anæmia of greater or less degree results from the disturbance of nutrition
hich so often accompanies the disease. In these cases the administration of
accharated carbonate of iron or of tartrate of iron and potash is indicated.

LECTURE XXV.

THE EXANTHEMATA.—(Continued.)

SCARLET FEVER.

THE third member of the group of exanthemata which I shall speak of is scarlet fever, and I have brought you to the scarlet fever ward of the Boston City Hospital to-day to show you some illustrative cases of this disease.

Scarlet fever is an acute infectious disease, characterized by a short incubation, short prodromal stage, erythematous efflorescence, pronounced desquamation, and long course. The micro-organism which produces it has not yet been determined. With the exception of variola, it is the most dangerous of the group. As it occurs so much more frequently in early life than variola, on account of its not being preventable by inoculation, it is to the physician the most important of all the exanthemata.

The complications of scarlet fever are so much more serious and its sequelæ so much more common and grave than those of varicella and measles, that its immediate diagnosis and prompt treatment are of vital necessity in every community where numbers of children are liable to be attacked by the disease. It should, therefore, receive the most careful study of every physician whose practice is among children.

Scarlet fever is the most irregular of all the exanthemata in its virulence and in the manifestations which it presents in different individuals. It is usually epidemic, returning to the same localities after a period of years. It is at times sporadic, and is commonly endemic in large cities. That the epidemics of scarlet fever vary in severity has been clearly shown a number of times, so that we cannot ascribe the virulence of the disease in certain years to individual susceptibility. The sporadic cases may be of the most malignant or of the mildest type. A mild case may give rise to a malignant case in another child, and a malignant case may give rise to a mild one. The epidemics of scarlet fever spread slowly, in contradistinction to those of measles, which spread rapidly. Scarlet fever may occur more than once in the same individual, but this is rare. Instances have occurred where a child has had scarlet fever, and, on returning after several weeks to the same room, even after it had been disinfected, has again contracted the disease in its typical form. The source and identity of the contagium have not been definitely determined, but the skin appears to be its chief vehicle. This contagium has a wonderful tenacity for clothing and other articles, and may be capable of reproducing the disease for many months.

In reference to what I have just said concerning the slow spread of fever during epidemics in comparison with the rapid spread of s, certain clinical facts are significant. The disease does not seem to y infectious in its early stages. We are thus led to believe that it ing the stage of desquamation that the contagium is most likely to be inated. Measles, on the other hand, is known to be highly infectious early stages, and for this reason to spread more quickly.

the description of actual cases aids the student to remember im- t points in a disease, I shall in a few words tell you about two en who have been under my care, in order to show you the difference n scarlet fever and measles as regards the stage in which they are likely to be infectious and the means by which their contagium is y conveyed. Notice, however, that I say usually, for the contagium h diseases may be active through their whole course.

boy (Case 233) six years old and a girl (Case 234) four years old slept in the same vith their beds touching each other. The boy was taken sick May 1, but remained ame room with his sister during that day and the following night. He was seen by y on the morning of May 3, and was then found to have scarlet fever. His sister was the country, and the boy was left in charge of a trained nurse. There was absolutely munication between the town-house and the country-house, either by people, clothes, r. I myself did not again see the boy during his sickness, having placed him under rge of another physician.

June 1 I was called to see the girl, and found that she had scarlet fever. There were r cases of scarlet fever in the vicinity of the country-house where she had remained aving the city.

e boy at this time was desquamating freely, and four days previous to the girl's being ick a letter written by him had been sent to her, and she, after having had it read to d been allowed to keep it under her pillow.

careful study of this case led to but one conclusion,—that the boy during the period lesquamation had infected his sister at a distance of twenty miles by enclosing the um of scarlet fever in an envelope. The girl, although she had been in the same ith the boy for thirty-six hours at the beginning of the disease, and although suscep- the disease, had not contracted it at that time, owing to its very slightly infectious in its early stages. On the other hand, the incubative stage of scarlet fever being few days, and many instances having proved that the disease is very infectious during od of desquamation, it was evident that the girl had been infected by means of the

the following year, on May 20, I was again called to see the same boy. He had been the morning, but in the afternoon was found to have a high pulse and temperature, ryza and lachrymation, so that it was deemed best to send the sister, who had been nursery only a few hours with her brother after he had been taken sick, to another while the boy was absolutely isolated. Three days later the boy was found to have . Ten days later the girl was attacked by measles. This case merely emphasizes w commonly accepted belief that measles, in contradistinction to scarlet fever, is infectious in the early hours of the disease.

hether the contagium of scarlet fever can be carried by the breath is, k, somewhat doubtful.

here are, however, cases which lead me to believe that scarlet fever may nsmitted at a very early stage of the disease. An instance illustrative s came to my notice not long ago:

A child (Case 235) who had contracted scarlet fever a few days previously came to a party given in a small and practically isolated community. At this time the child was beginning to feel sick and complained of a sore throat. A spoon which had been used by her was also used, before it was washed, by one of the other children. Six or seven days later this second child (Case 236) was attacked by scarlet fever.

A careful and critical investigation of the possible origin of the second case resulted in the evidence strongly pointing towards a direct transmission of the contagium from the mouth of one child to that of the other by the use of the spoon.

Scarlet fever may occur at all ages, but is rare during the first year of life. It has been met with in young infants who were nursing, and who have proved to be the focus of infection for a whole household.

It may occur in animals, and the infection may be transmitted by animals, such as dogs and cats, by milk, and by clothing.

There is no known prophylactic against scarlet fever except isolation, which for many reasons should be rigorously enforced. We must remember the fact that when the child has passed its tenth year the chances of its ever contracting the disease are very much lessened. We must also appreciate that it is especially important to protect children who are learning, or who have just learned, to talk. The commonly occurring and often intractable form of otitis which accompanies scarlet fever may not only render the child deaf, but in a case where the child has not learned to talk it may lead to deaf-mutism. We should, therefore, under all circumstances discountenance the opinion so often expressed by the laity, and sometimes even by physicians, that it is well for children to have these diseases while they are young, on the ground that otherwise they will probably contract them at a later period of life, when the type of the disease may be more severe. The assertion that the type of the disease is more severe in adults than in children is not corroborated by my experience.

PATHOLOGY.—The organs primarily affected in scarlet fever are the *skin* and the *throat*. The principal complications which arise in the course of the disease are connected with the *ear* and the *cervical glands*. The chief sequela, and the only one which is at all common, is *nephritis*. *Cardiac disease*, commonly secondary to the nephritis, may occur.

Lesions of the other organs are somewhat unusual and have no definite connection with the scarlet fever. They are generally due partly to the fever and partly to the septic processes which have arisen in the course of the disease, and are represented, as would naturally be expected, by a congested condition of the various internal organs, and by the usual changes which are found in pleuritis, pericarditis, endocarditis, and meningitis.

Skin.—Macroscopically the morbid conditions of the skin in scarlet fever, though varying in their manifestations, are usually represented by an intense general erythema covered thickly with minute macules, which are of a darker red than the accompanying hyperæmia. Minute white spots may also appear thickly scattered over the reddened surface, probably arising from areas of unaffected skin existing in the midst of the general hyperæmia. An appearance like that of milium is also at times noticed to be scattered on the areas of skin affected by the erythema. No evidence of this hyperæmic condition, which is so pronounced during life, is found after death.

According to Neumann, microscopic examinations of the skin by means of hardened sections of specimens from cases of scarlet fever and measles in the stage of desquamation explain in a measure why the former is so much more likely to be infectious during its stage of desquamation than is the latter. In contradistinction to the pathological processes which are found in the skin in measles, and which affect chiefly the blood-vessels and glands, a very different picture is presented on examination of sections of skin taken from scarlet fever. In the latter we find the pathological process represented especially by exudative cells, which are very numerous and closely packed together, reaching even up to the horny layer of the epidermis. Occasionally these exudative cells may finally take the place of the epidermal cells, appearing on the free surface of the skin, and are gathered thickly among the excretory ducts of the cutaneous follicles. You will thus readily understand why the tissue proper of the skin and its epidermis present no marked changes in measles, and why the epidermal cells are far less likely to carry the contagium than in scarlet fever, where the possibility of contagium exists until the desquamation has entirely ceased.

Throat.—The earliest lesions of scarlet fever appear on the mucous membrane of the hard and the soft palate. This appearance is very similar to the efflorescence which is seen on the skin, except that the minute white spots do not appear on the congested mucous membrane. The pathological conditions which occur in the throat in scarlet fever may either be simply catarrhal, or result in one of the more severe inflammatory conditions affecting the tonsils, the pharynx, and the larynx.

As is stated by Delafield and Prudden, one of the most marked features of scarlet fever is the predisposition which it entails to the incursion of pathogenic germs other than those which we believe to cause this disease. Thus, in addition to the inflammatory lesions produced by the scarlet fever organism an acute exudative inflammation of the mucous membrane may occur, and may be associated with them. This is apparently caused by the growth of a streptococcus which, according to Welch, in morphological and biological character seems to be identical with the *streptococcus pyogenes*. In these cases there may be much or little fibrinous exudate, and there may in the early stages, or even through the whole course of the affection, be none at all. The pellicle when formed may be more or less adherent, and sharply circumscribed, or it may tend to spread. The submucous tissue may show little change, or much congestion and œdema, or it may be the seat of suppurative inflammation. The entire process may be confined to the tonsils. While under these varying conditions the inflammatory process is usually a local one and runs its course, with or without the symptoms of septicæmia, occasionally the streptococcus finds access to the blood and may induce the lesions of pyæmia. On the other hand, it may by inhalation gain access to the lungs and induce varying phases of complicating broncho-pneumonia. The staphylococcus pyogenes is not infrequently associated with the streptococcus in these lesions, but it is not apparently of prominent significance. Simulating very closely as it does in many cases both the local and the general phenomena of diphtheria, this pseudo-membranous condition was formerly confounded with it, but it is now recognized as a distinct disease.

There have been a number of extended investigations made on what are called the pseudo-membranous inflammations of the throat in scarlet fever. Booker has reported eleven cases of pseudo-membranous angina (two fatal) complicating scarlet fever, and one case of simple angina without exanthem in a family three members of which had scarlatina. In all these cases, as well as in four scarlatinal anginas without pseudo-membranes, Booker found streptococci as the predominant organism, and in none was the Loeffler bacillus present. The staphylococcus aureus was found in eleven cases without apparent influence on the severity of the disease. No difference was observed between the early and the late pseudo-membranous anginas as regarded the bacteria present. Booker describes with much detail the morphological and bacteriological characteristics of the streptococci found, and divides them into groups.

Park, in a series of one hundred and fifty-nine cases, reports nineteen cases of pseudo-membranous inflammation of the throat complicating scarlet fever. In seventeen of these cases streptococci predominated, and in only two was the Loeffler bacillus present. Staphylococci were found in only a few cases. Williams has also reported cases of this kind, and Morse has reported ninety-nine cases of pseudo-membranous inflammation of the throat complicating scarlet fever. The Loeffler bacillus was found in twenty-three, with a mortality of forty-three per cent., and was not found in seventy-six, with a mortality of twenty-one per cent.

Finally, we may conclude that in scarlet fever the mucous membrane of the throat is rendered peculiarly vulnerable to the invasion of pathogenic germs. Where the morbid condition in the throat is represented by a pseudo-membrane it will be found that in the great majority of cases the process, as stated by Welch, is due to streptococci; but where diphtheria is prevalent and the opportunities are favorable for exposure, a large portion of the pseudo-membranous cases may be due to the Klebs-Loeffler bacillus.

In addition to the lesions of the throat just described, the micro-organism of scarlet fever may attack the naso-pharynx. In this way, also by direct extension through the Eustachian tubes, secondary aural lesions may be produced. The morbid changes in the mucous membrane of the naso-pharynx which thus take place may result in a thickening of the tissues, which in some cases lasts for many months after the scarlet fever has run its course.

Ear.—The pathological condition of the ear which is most commonly met with in scarlet fever is an acute inflammation of the middle ear. This inflammation is likely to result in destruction of tissue, the formation of adhesions, the establishment of a long-continued suppurative process, and an accompanying necrosis.

Cervical Glands.—There may be hyperplasia of the cervical lymph nodes. This condition is sometimes accompanied by inflammatory œdema of the tissues of the neck, which may go on to suppuration and even to gangrene. In these cases streptococci are found in the glands and in the areas of suppuration. The infection is supposed to originate in the throat. The enlarged glands are, as a rule, indicative of secondary or mixed infection, though it is possible that the slighter forms of enlargement may be due to reflex irritation with resulting hyperplasia from the scarlet fever contagium. In the severe form the glands are at times very much enlarged, and where a gangrenous process results the blood-vessels may be affected to such an extent as to be ruptured.

Kidney.—In scarlet fever, as in a number of other infectious diseases, there are certain poisons produced in the course of the disease which are probably soluble in character. The results of bacteriological cultures in scarlet fever have shown that in a number of cases there is a general streptococcus infection, the infection probably coming from the lesions in the pharynx. In these cases of general infection streptococci may be cultivated from most of the organs of the body, there being a general septicæmia. In a number of these cases extensive lesions may be found in the kidneys, and yet these lesions may bear no relation whatever to the presence or absence of streptococci. In like manner, streptococci may be found in the kidney without any lesion of the kidney. These lesions are diffuse, and affect both kidneys and all parts of the kidney. From the best evidence which we have it would seem that the virus, or whatever it is which produces the lesions in the kidney, is not a living organism, but is a soluble chemical poison produced by the organisms of scarlet fever, or by other organisms, located in some other part of the body. This soluble poison when produced elsewhere is taken locally into the blood and affects various parts of the economy. In post-mortem examinations of scarlet fever certain lesions will be usually found in the kidneys.

These lesions, according to Councilman, may be divided into two classes, (1) represented by simple degeneration of the epithelium, and (2) represented by marked changes in the tissues of the kidney.

In the *first class* of cases the soluble poison may only affect the integrity of the capsular epithelial cells of the glomeruli. The poison may produce certain degenerative changes in these, but need not be accompanied by any proliferation of cells, or by any condition which would be characterized as inflammatory. It is more than probable that these simple degenerative lesions are accompanied during life by evidence of albuminuria, and in case death takes place there may be no macroscopic evidence of any lesions in the kidneys. Careful microscopic examination, however, will show a condition of degeneration in the capsular epithelium of the glomeruli.

Associated with this there will usually be found cloudy swelling of various degrees of intensity in the cells of the convoluted and the smaller collecting tubules. The degeneration here is rarely of a fatty character. Clinically, in the purely degenerative changes there may be only albuminuria with the presence of faint hyaline casts, and here and there a few leucocytes.

In the *second class*, owing to a greater intensity in the action of the poison, or to some possible difference in its character, more marked changes may take place in the kidney, and may be accompanied by the degenerative lesions which are distinctive of the first class. Different forms of lesions may occur in the second class, and, according to the predominance of one form over the other, may characterize a special form of renal disease. These lesions may be divided according to their anatomical distribution into *interstitial*, where there is a marked proliferation of the interstitial tissue of the kidney, and *glomerular*, where the lesions are chiefly confined to the glomerulus and its capsule.

In the *interstitial* form there will be found in the interstitial tissue between the tubules accumulations of cells, which are probably due to a proliferation of the cells of the capsule and of the connective tissue. These cells, or most of them, are epithelioid in character, and show very few leucocytes mingled with them. This form of nephritis should be considered as purely interstitial, since its lesions are in no way related to those of the epithelial tissue. There is both a general and a focal infiltration of cells in the interstitial tissue. The focal infiltration is found principally in the cortex of the kidney and about the glomeruli, the glomerulus frequently appearing as a centre from which the infiltration extends into the interstitial tissue between it and the surrounding tubules.

This form of nephritis was first described by Wagner as the lymphoid kidney. The kidney, macroscopically, is swollen; the capsule is easily stripped from the cortex, and is moist, whitish, and opaque. Usually there is no evidence of hemorrhage, although in some cases points of punctiform hemorrhage may be found in the cortex and in the intermediate zone.

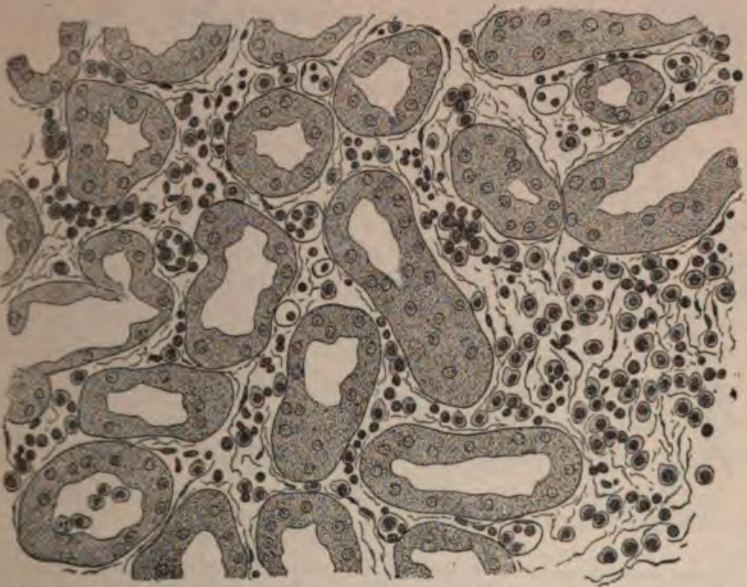
Clinically, in this form there may be little evidence of the severity of the lesions. There may be, however, albuminuria corresponding to what is seen in the purely degenerative class. The quantity of the urine may be very little diminished, and casts may be present, as well as a certain number of desquamative epithelial cells and leucocytes.

These lesions are not confined to scarlet fever, but may be found in diphtheria, in measles, and in other infectious diseases of children, but they are not common in the infectious diseases of adults.

This microscopic section, made by Councilman (Fig. 90, page 539), represents a good example of these interstitial lesions in scarlatinal nephritis.

This section was taken from a case of pure scarlet fever. There was no anuria and no dropsy. The kidneys were enlarged, whitish, and without hemorrhage. Cultures from this case gave a general infection with streptococci in all the organs except the kidney, and I wish you to notice cially that the kidneys, notwithstanding the extent of their lesions, e found to be free from streptococci. The epithelium of the tubules is ewhat swollen. The tubules themselves are slightly dilated, and the helium is more granular than normal. The interstitial tissue is much e extensive than normal. The spaces between the tubules are increased by œdema and by cellular infiltration. In the interstitial tissue you see blood-vessels filled with cells of the same character as those outside. probable that most of the cells outside come from proliferation of the of the blood-vessels. The round spaces in the interstitial tissue repre- blood-vessels.

FIG. 90.



terstitial nephritis. Section of kidney from child with scarlet fever. (Hartnack, ocular No. II., objective No. VIII. Tube closed.)

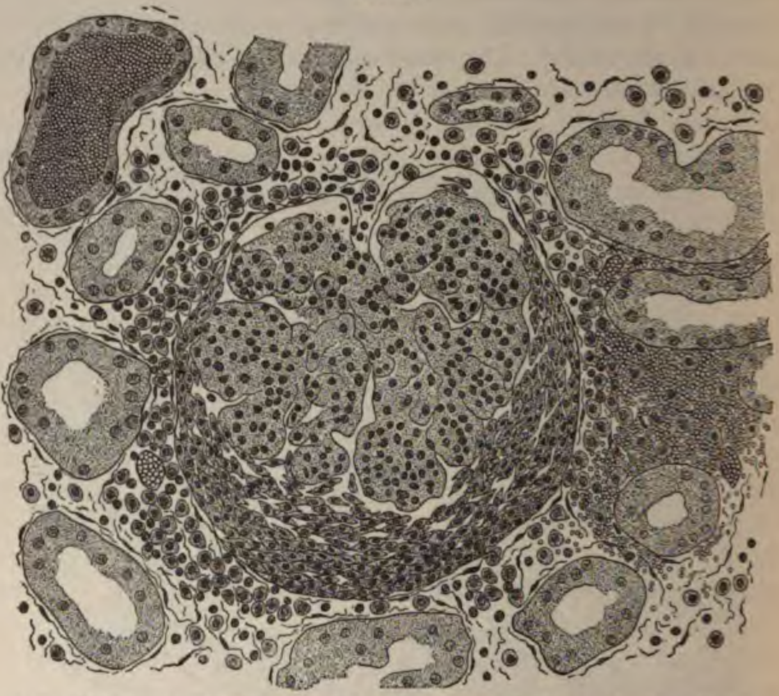
The other form of nephritis, called the *glomerular* (page 540), is much e frequently found in scarlet fever than the interstitial form, and may onsidered as almost typical of the disease. In this glomerular form chief lesion of the disease consists essentially in a proliferation of the ular epithelium combined with hyperplasia of the connective tissue.

The proliferation of the capsular epithelium leads to the formation of ses of cells within the capsule between the glomerular capillaries and capsule. These cells evidently result from the proliferation of the ular epithelium. As a result of this there may be greatly increased sure on the vessels of the glomerulus, with possibly obliteration of these els. The cellular infiltration in the interstitial tissue is not so extensive n the other form (Fig. 90). Accompanying these changes in the merulus there is almost always more or less hemorrhage both in the ules and in the interstitial tissue.

Here is a section, made by Councilman (Fig. 91), of glomerular nephritis.

This section was taken from a case of scarlet fever complicated by glomerulo-nephritis. In the centre of the field a glomerulus is seen, with an infiltration of cells in the capsular space. The capsular cells are oval and distinctly epithelioid in character. Cellular proliferation of the cells having generally the character of those in the section of interstitial nephritis (Fig. 90) is found, as you see, in the interstitial tissue. In the tubule at the left upper corner there is hemorrhage, and hemorrhage is found in the interstitial tissue on the right of the specimen. In this case the anuria and dropsy were extreme.

FIG. 91.



Capsular glomerulo-nephritis. Section of kidney from child with scarlet fever. (Hartnack, ocular No. II., objective No. VIII. Tube closed.)

This form of nephritis may be best designated as *capsular glomerulo-nephritis.* The kidney is swollen and much more hyperæmic than in the interstitial form. The markings of the cortex either are obscured or cannot be made out at all, and there are numerous areas of hemorrhage and hyperæmia, giving the kidney a mottled appearance.

It is this capsular glomerulo-nephritis which gives the most marked clinical evidence of the extent of the lesions in the kidney. In this form dropsy is almost always present, the amount of urine is greatly diminished, and in the more severe cases there may be complete anuria. Blood-casts are found more frequently in the urine than in the interstitial form. The liminution in the amount of the urine points to involvement of the glomeru-us. Even severe cases of this form may be recovered from. The process f cell-proliferation may cease, the cells formed in the capsular space may lisappear and pass out, and the kidney in after-years may show few or no vidences of the process through which it has passed. In a certain number of ases, however, from this form of nephritis a chronic nephritis is developed. 'ases of this kind have been reported, notably one by Aufrecht.

In both the interstitial and the capsular glomerulo-nephritis fatty degen-ration of the epithelium is not found to any degree. The epithelium is requently swollen and granular, and may be hyaline.

These two forms of nephritis should be separated from each other, lthough transitions between their lesions are found. Usually they can be listinguished macroscopically.

We can, therefore, recognize three pathological conditions of the kidney n scarlet fever: first, the purely degenerative; second, the acute interstitial; nd third, the capsular glomerular.

Heart.—The pathological conditions of the heart which are at times ound in scarlet fever do not differ in their macroscopic appearances from hose met with in other diseases. Cardiac disease occurring in the course of carlet fever may arise in two ways: (1) from the general septic condition xisting during the period of the height of the temperature and general fflorescence, and represented usually by an endocarditis; (2) at a much later period from a nephritis which has arisen as a complication, and following which, from the resulting increased blood-pressure, enlargement of the heart has been produced, which may be represented by hypertrophy or by dilatation, or by both.

In connection with this subject, Silbermann has found on examining a large number of cases of nephritis during attacks of scarlet fever a decided hypertrophy of the heart combined with dilatation. In some cases both sides of the heart were equally affected, but usually only the left side was involved. In only a few cases was there found a partial fatty degeneration of the muscular fibres; the endocardium, pericardium, and blood-vessels were normal. According to Silbermann's observations, the cardiac affection was related to the post-scarlatinal nephritis, and not to the scarlet fever process itself, as the hypertrophy was never found where the child died in the early weeks of the scarlet fever. He calls attention to the short period which intervened between the first appearance of the nephritis and the consecutive heart hypertrophy, in many cases the time not being much longer than a week. He also noticed that in the cases where hypertrophy and quick dilatation followed the acute nephritis of scarlet fever the ages of the children were three and a half, four, five, and six years, this post-scarlatinal cardiac enlargement thus corresponding to the physiological hypertrophy which I have referred to in an earlier lecture (Lecture IV., page 123).

Scarlet fever may be divided into (1) the benign form and (2) the malignant form.

I have already referred to the variations in type of cases of scarlet fever. The difference in the symptoms of the common, or benign, form of the disease from those of the rare, or malignant, form is very striking. They could well be classified as entirely separate diseases, were it not that the contagium has been proved to be the same in each, by the fact that one form of the disease may give rise to the other in different individuals. It seems as though it were more the susceptibility of the individual to the scarlet fever contagium than the contagium itself which produces a greater or less severity of the symptoms. I shall first speak of the benign class of cases, such as you see here in my scarlet fever ward, as it is this class which you will be more likely to meet with in your practice. These cases, as you will see, either run a simple typical course or are accompanied by variations and complications which make their course irregular. The simple typical case of the benign form of scarlet fever is such as I have already described, and is characterized by its sudden onset and long duration.

INCUBATION.—The stage of incubation of scarlet fever is uncertain and irregular, but, as a rule, it is shorter than that of any of the other exanthemata. It is usually less than seven days, and quite frequently it is only from two to four days.

SYMPTOMS.—**Prodromata.**—The invasion of the disease is usually sudden and, as a rule, active. The child feels very sick, looks dull, complains of sore throat and nausea, and in a large number of cases vomits continuously. The pulse is rapid. The temperature is high,—39.4°, 40°, 40.5° C. (103°, 104°, 105° F.). In infants and very young children if the temperature rises to 40° or 41.1° C. (104° or 106° F.) convulsions are very likely to occur. The higher the temperature at the beginning of the disease the more active the symptoms, and the shorter the prodromal period the more severe will be the case. An initial temperature of 40° C. (104° F.) points towards a severe case.

Young children seem to show a less sensitive condition of the throat than is met with in older children and in adults. The appearance of the mucous membrane of the throat, although perhaps not characteristic, as at times a simple non-infectious pharyngitis may simulate it quite closely, is, in connection with the general symptoms, at least suggestive. The mucous membrane of the hard and the soft palate and of the pharynx is much congested. On the hard and the soft palate thickly scattered over the reddened surface are minute macules the color of which is a little darker red than that of the intervening mucous membrane. This condition represents the earliest stage of the efflorescence which later appears on the skin.

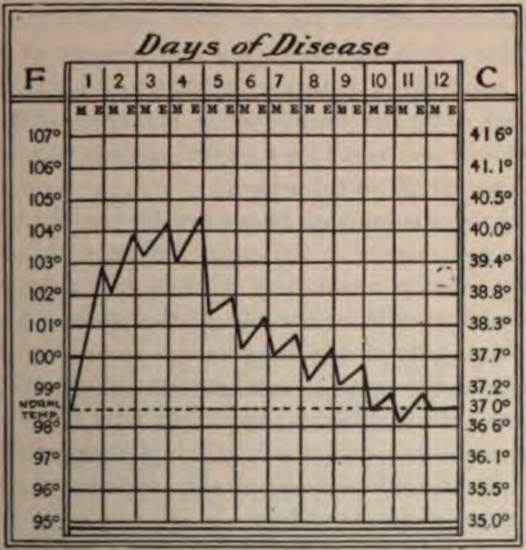
The length of the prodromal stage varies, as a rule, from twelve to thirty-six hours. During this stage the temperature continues to rise somewhat, and at its end the efflorescence appears on the skin.

Efflorescence.—The efflorescence of scarlet fever is of an erythematous and punctate character, sometimes looking as though minute macules had been sprinkled over the general redness of the skin. It starts on the front of the neck and the upper part of the chest, and rapidly extends all over the body and extremities, and upward to the face. This characteristic order of invasion of the skin aids us in distinguishing the efflorescence of scarlet fever from that of the common erythema which occurs in such diseases as pneumonia, and in cases where certain drugs, such as belladonna, have affected the skin and the efflorescence comes out everywhere at once and has an irregular distribution. It also enables us to distinguish the disease from measles, in which the efflorescence begins on the sides of the neck and on the face and extends downward. On gently drawing the finger over the efflorescence of scarlet fever the resulting white mark remains longer than is the case with a common erythema. The efflorescence of scarlet fever continues to extend over the body for two or three days after its first appearance. During this period the tongue is much reddened and its papillæ appear very prominent, constituting what has been called the "strawberry tongue." There is at times in this stage great irritation of the skin.

There may be slight delirium even in mild cases during the stage of efflorescence. This delirium may be very active and yet not be of serious import, provided the temperature remains moderate.

The temperature rises when the efflorescence appears, and reaches its maximum at the end of the outbreak, in uncomplicated cases, but there is no decided rise just before or fall after the height of the efflorescence, as is the case in measles; on the contrary, the temperature slowly diminishes until the ninth or tenth day from the beginning of the prodromal symptoms, when it becomes about normal, showing no decided crisis such as is seen in measles, but representing what is called lysis.

CHART 14.



Benign and regular form of scarlet fever.

The pulse is quickened during the period when the temperature is elevated, and corresponds to it. It varies from 120 to 160.

This chart (Chart 14, page 543) represents the temperature as it commonly occurs in cases of scarlet fever of the benign and regular form.

The vomiting usually ceases in the stage of efflorescence, and often before the prodromal stage is ended.

Desquamation.—The stage of desquamation begins at about the seventh day from the time when the efflorescence first appears, and in the parts of the skin first attacked. The desquamation, however, is not always proportionate to the intensity of the efflorescence. This desquamation is at first composed of small particles of cutis, but these soon become larger, and early in the third week from the beginning of the disease they fall from the body in large flakes. This form of desquamation is called lamellar. Here again we have an important means of distinguishing scarlet fever from measles, for in measles the desquamation is almost universally of a furfuraceous character through the whole course of the disease, while the characteristic desquamation of scarlet fever is lamellar. This lamellar form of desquamation may at times, in certain individuals, and following the more intense inflammations of the skin, be represented by large and extensive pieces of skin. This is well shown in a specimen in the Warren Museum, where large strips of skin have come from the hand of a patient with scarlet fever so as almost to form a glove.

Sometimes the desquamation lasts only ten days, but it may continue for two or three weeks. It is especially slow in disappearing from the hands and feet, and it may remain between the fingers and toes for a number of weeks. Sometimes after the desquamation has apparently ceased and the skin has been smooth and normal for several days it may begin again, and thus prolong the period of convalescence.

URINE.—The *urine* is lessened in amount during the prodromal stage, returns to the normal amount in the stage of efflorescence, increases during the stage of desquamation, amounting at times to a polyuria, and returns again to the normal amount at the end of this stage. During the stage of efflorescence, especially if the temperature is considerably heightened, there may appear in the urine a small amount of albumin, but this disappears as the temperature subsides, is probably only the result of the fever, as in many other diseases accompanied by a high temperature, and is not to be confounded with the albuminuria of the nephritis which in some cases complicates the stage of desquamation.

There is considerable reason to suppose that a mild form of nephritis accompanies almost every case of scarlet fever, although in many cases no clinical symptoms pointing towards the kidney appear and nothing abnormal is found on examination of the urine. This statement, however, rests to such a degree on the authority of the general practitioner, rather than on that of the expert in urinary analysis, that we shall probably in the future find the number of cases which show nothing abnormal in the urine greatly lessened when the number of expert examinations of the urine in mild cases of scarlet fever has increased.

PROGNOSIS.—The prognosis of the benign and regular form of scarlet fever is in almost every case favorable. It is comparatively rare for the symptoms to become sufficiently serious to cause death unless some complication has arisen in the course of the disease. The individual who succumbs to the simple uncomplicated form of scarlet fever, even when the initial temperature is high and the symptoms are severe, as a rule must have been unusually vulnerable to the toxic effects of the scarlet fever contagium, or must have had a very low degree of vitality at the beginning of the disease.

DIAGNOSIS.—The diagnosis of the benign and typical form of scarlet fever is not difficult. Its incubation is decidedly short in comparison with that of any of the other exanthemata. Its prodromal stage is short in comparison with that of variola and measles, and longer than the exceedingly brief prodromal stage of varicella. The characteristic prodromal symptoms of sore throat and a general and intense hyperæmia of the mucous membrane, accompanied by vomiting and severe constitutional symptoms, make it easy to differentiate it from measles, varicella, and variola, none of which, as a rule, show these symptoms.

The punctate erythematous lesions which appear in the stage of efflorescence of scarlet fever are rarely met with in any of the other diseases of this group. This efflorescence, beginning on the neck and chest and extending upward and downward, is distinguished by its peculiar distribution from that of the other members of the group.

The lamellar desquamation is very characteristic, and is seldom seen in any of the other exanthemata.

The complications arising in the ear, and the occurrence of nephritis as a common sequela in scarlet fever, do not to the same degree find their counterparts in varicella and measles.

TREATMENT.—As I have often told you in speaking of other diseases, so in scarlet fever, having an accurate knowledge of the chief pathological lesions which occur during the course of the disease, you can easily deduce the appropriate treatment. By treatment, you must understand, I do not mean simply the use of drugs. On the contrary, I would impress upon you that in my opinion drugs are employed to entirely too great an extent in a large proportion of the uncomplicated cases of the benign type of scarlet fever. I feel that I can speak with some authority on this point, as it has been my rule for many years to compare the results of cases treated by my colleagues with drugs with my own cases treated without drugs, and certainly nothing that I have observed in this comparison would indicate that my patients had suffered from want of treatment. We should have some definite reason for what we do, and should not be influenced by vague ideas of what drugs are supposed to be beneficial in certain diseases.

The treatment of a case of scarlet fever is that of a self-limited disease.

With our present knowledge of it, the disease cannot be cut short. We should, therefore, endeavor to keep it within its own limits by avoiding complications. To do this we must remember which tissues are affected as part of the disease and which are likely to be affected by complications.

In the first class, as I have already explained, we must consider the throat and the skin; in the second class the ear and the kidney. Remember, gentlemen, that I am now speaking of the mild cases of scarlet fever, and that the severe and complicated cases must receive their appropriate treatment as they arise.

The treatment here in my scarlet fever ward is rendered much easier than is the case in private houses by the fact that I am absolutely free from family prejudices as to how scarlet fever should be treated, and also because the patient can at once be put in a room from which all unnecessary paraphernalia have been removed. As, however, your cases in practice will be in their homes, it will be better for me to describe the general management and treatment of scarlet fever outside of hospitals.

At the onset of the disease the child, as a rule, is so profoundly affected by the scarlet fever contagium that it wishes to be put to bed at once. The symptoms which from their intensity require treatment in the prodromal stage of the disease are the vomiting, the sore throat, and the high temperature.

The vomiting, as a rule, is of such short duration, and is so symptomatic of nervous gastric disturbance caused by the toxic effect of the poison, that it should be looked upon as eliminative, and usually does not require the use of anything but pieces of cracked ice to be held in the mouth.

The treatment of the throat in scarlet fever is to be especially directed not only to allaying the temporary discomfort of the pharyngitis, but also to preventing the inflammatory process from extending through the Eustachian tubes to the membranæ tympani and producing an otitis which may result in a meningitis. This latter complication is rendered possible by the close vascular connection which exists in infancy and in childhood between the meningeal blood-vessels and the vessels of the tympanum through the open petro-squamosal suture.

Another reason for systematically treating the throat in all cases of scarlet fever is derived from the belief that the various secondary infections which take place in the disease are probably caused by the entrance of pathogenic organisms to the various tissues through the inflamed and vulnerable mucous membrane of the pharynx. This invasion is commonly of the cervical glands, the ear, the lung, the heart, and the kidney. If this belief is correct, antiseptic treatment directed to the throat is indicated as possibly preventive to the complications which may arise in the disease.

For the purpose not only of allaying the irritation of the throat, but also of preventing the spread of the morbid process to the ear, if possible, the throat and the nose may be sprayed several times during the day. Solutions of borate of sodium in water combined with a small amount of

ycerin are useful for this purpose. A four per cent. solution of boric acid
water can also be used to advantage. The local treatment, however,
ould always be of the mildest form, since any additional irritation of the
ucous membrane will render it more vulnerable to the streptococcus inva-
n. If the child knows how to gargle, the discomfort which arises usually
m the sore throat during the first day or two of the disease may often
allayed by simply gargling with cool water. This procedure answers a
uble purpose: it not only reduces somewhat the hyperæmic condition of
e mucous membrane of the upper part of the throat and cleanses the
terior fauces, but also tends to prevent the extension of the pathogenic
ganisms which would necessarily be favored by a continuous recumbent
sition of the child. If the child is unable to gargle, some pieces of ice
y be given to it to hold in its mouth, and it should occasionally be allowed
sit up, as when its nourishment is being given.

However desirable this treatment of the throat and nose may be in
rlet fever, we are but too often baffled in our attempts to treat them
ally, on account of the persistent resistance of the child.

Chlorate of potash, which is so frequently used for the treatment of
e throat in scarlet fever, is, in my opinion, a drug which in this disease it
ll be wiser not to allow the child to swallow, on account of its possible
leterious action on the kidney, which from the beginning of the disease
its end is in so sensitive a condition as to be readily affected by any
itant. Doubtless in a large number of cases we should not be likely
cause renal irritation by the small doses of chlorate of potash which
e usually given. Children, however, differ very much in their individual
sceptibility to drugs, and we can never tell beforehand whether or not
child is liable to be injured by them. We know that the vegetable
lts of potash are decomposed in the system and eliminated as alkaline
rbonates, thus causing no irritation in the kidney. Nitrate and chlo-
te of potash, on the other hand, which do not part with their oxygen in
e system, are excreted undecomposed by the kidney, and thus act as irri-
nts. Knowing that the tendency during the whole course of the disease
towards a renal hyperæmia, we should allow the child to have plenty of
ter to drink.

Unless the child shows decided signs of suffering from a heightened
mperature, I do not use antipyretics in the form of drugs by the mouth,
the cases are rare where a temperature of 38.8° to 39.4° C. (102° to
3° F.) for a few days will do harm. This is a safe rule to follow in a
ease like scarlet fever, where, if the child happens to be easily affected by
er, the unfavorable symptoms will appear at once and can be attended to.
y opinion is that mere heightening of the temperature without correspond-
gly severe symptoms causes much needless anxiety. In typical mild cases
the disease I should, knowing that a lessening of the amount of the urine
the prodromal stage as a result of the high temperature is a part of the
gular course of the disease, discountenance the administration of diuretics

beyond a plentiful supply of pure drinking-water. The temperature, although it may cause severe initial symptoms, such as convulsions, as a rule, does not have to be directly treated during the prodromal stage. If, however, convulsions occur and continue and the temperature is unusually high, such as 40.5° or 41.1° C. (105° or 106° F.), and if it remains at this height with serious general symptoms, such as delirium, you should endeavor to reduce it by sponging the body with water, the temperature of which should be varied according to the special case. To begin with, the temperature of the water should be about 32.2° C. (90° F.).

I have mentioned before that scarlet fever is rare during the first year of life. There are certain observations which seem to show that nephritis is a rare accompaniment of scarlet fever during the first year. We know that milk is the food which is least irritating to the kidney. It would, therefore, seem but rational to make milk the diet in a disease which, like scarlet fever, points out to us by its pathology that we should as far as possible avoid irritating the kidney. It may be merely a coincidence, but it seems of some significance that the first year of life should also be the one which is least likely to present cases of scarlatinal nephritis. For this reason I am in the habit of putting my patients with scarlet fever absolutely on a diet of milk from the beginning to the end of the disease, or at least for four weeks. Perhaps in this way in a certain number of cases nephritis may be warded off, and if it develops, the patient is already on a diet which is best suited to the disease.

When the nausea and vomiting are present, the child, as a rule, feels too sick to take any nourishment whatever. When the violence of the toxic invasion has somewhat abated, and the diagnosis of scarlet fever has been made, orders should at once be given that the child is to have no food but milk. The treatment of scarlet fever with a diet purely of milk has in my practice proved so eminently satisfactory that it has become my routine treatment of the disease. During the initial stage of the disease, and until the stomach has recovered its equilibrium, lime water should be added to the milk in the proportion of one part to ten. Later the alkalinity of the milk can be lessened, and after the early days of the efflorescence the milk may in most cases be given undiluted. The administration of milk alone should be continued through the stages of efflorescence and desquamation, and until you are justified in supposing that a nephritis will not develop in the special case. This in general may be estimated at from four to five weeks from the time of the height of the efflorescence and temperature.

During the stage of efflorescence there are seldom any symptoms which require special treatment, in the regular form of the disease, except a considerable irritation of the skin which at times arises. This can be allayed by the application either of some simple ointment or of a powder of oxide of zinc and starch (Prescription 56). The use of the ointment is to be recommended not only because it keeps the skin soft and in good condition, but also because this application reduces the temperature somewhat. Sponging he entire body with water at a temperature of 32.2° C. (90° F.), once or wice daily according to the comfort of the patient, is to be recommended.

During the stage of desquamation the application of a simple ointment o the whole body is desirable both for the purpose of softening the disntegrated epithelium and lessening the duration of this stage, and also o prevent the spread of the contagium by means of the loosened scales.

The child should be kept in bed until the desquamation has almost ntirely ceased. This will cover a period of from four to five weeks. By he end of the fourth week, if the desquamation has completely disappeared, he diet can gradually be increased by the addition of soup and bread. It is vell to keep the child in the house for five or six weeks, and still longer f the weather is cold or damp.

The *urine* should be frequently tested for albumin during the first three veeks, and afterwards when the child is first allowed to get up, after each hange in diet, and after going out. If any albumin is detected, the child hould be immediately put back to bed and on a diet of milk until he albumin has disappeared. Remember that the mild cases are the very nes in which a nephritis is liable to occur, and therefore we should watch hem vigilantly until they are out of danger, which is usually in the fifth or ixth week.

ISOLATION AND DISINFECTION.—The disease being eminently infectious, he patient with its nurse should be isolated to as great a degree as circumtances will permit. An upper room should be chosen preferably. It has een observed in crowded parts of large cities that scarlet fever in tenement-houses is not so likely to spread when the first cases are in the top rooms of the tenements. In a number of instances in my practice I have had one child of a numerous family strictly isolated in the upper story of the house, and the other children have remained in the house without contracting the disease.

The intensity of the lesions of the skin and the involvement of large surfaces indicate that the air of the room should be kept at an equable temperature, in order that the function of the disabled skin should be taxed as little as possible and that the internal organs should not have too great compensatory work forced upon them. The temperature should be kept at about 20° C. (68° F.).

A disease which renders necessary confinement to the room for weeks demands a room with good ventilation and plentiful sunlight. Therefore a room on the sunny side of the house, having an open fireplace, should be hosen.

The room should be free from all cotton or woollen materials except such as can be destroyed by fire at the end of the disease. The blankets, sheets, towels, and clothes can, of course, be disinfected, but it will save much ultimate trouble to remove the carpet and the curtains and replace them with pieces of old carpet and sheets. The pictures, and in fact everything worth preserving, had better be removed. The room can be made cheerful enough by means of cheap colored prints and destructible toys to amuse the child.

During the whole course of the disease the greatest care must be taken to disinfect the linen of both the patient and the nurse. This should be done by soaking it for twenty-four hours in a five per cent. solution of carbolic acid, then boiling it for half an hour in water, and finally washing it with soft soap solution, 20 grammes (⅔ ounce) to 10 litres (10½ quarts) of water.

The dejections are to be received in a vessel one-quarter full of a five per cent. solution of carbolic acid.

After the child is entirely well it is to be thoroughly washed first in a solution of corrosive sublimate 1–2000, and then immediately with water, so as to avoid irritation of the skin. The child is then to be taken to another room to be wiped and put into fresh clothes, which, of course, have not been in the scarlet fever room. The mattress is to be tied up in canvas wet with a corrosive sublimate solution 1–500, and sent out of the house to be disinfected, if possible by steam. I usually advise the family never to have it brought back again. In place of the mattress it is far better to use old blankets, which, if in sufficient number, are comfortable, and at the end of the sickness can be thoroughly boiled and thus disinfected. The useless articles which have been in the room during the sickness should be burned in the open fireplace.

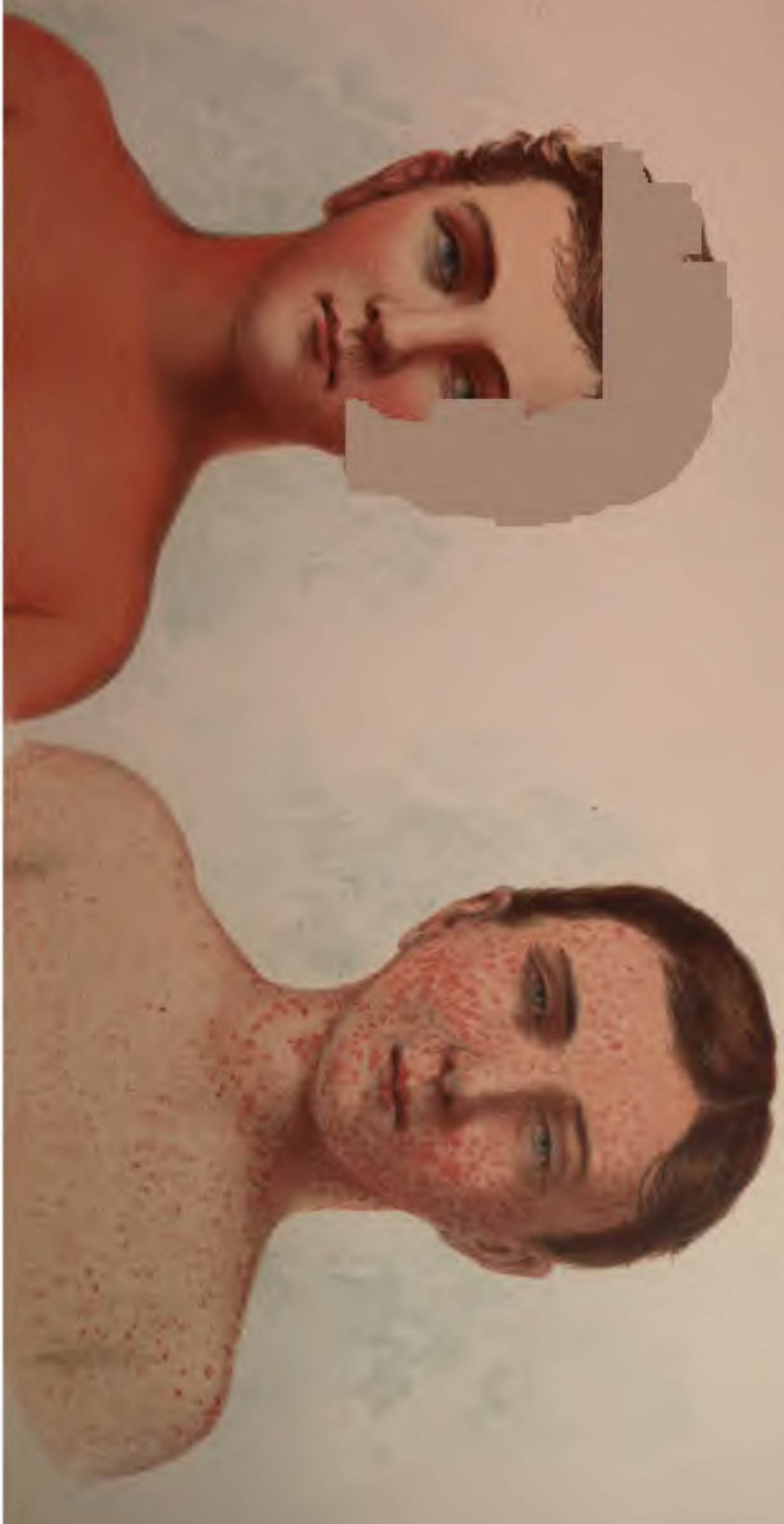
The room must next be disinfected. This is a very difficult matter to do absolutely, but there are several methods which are far better than the usually recommended disinfection by sulphur which has been so generally used for this purpose during the past century. I mention sulphur as a disinfectant merely to tell you that it was proved by Koch as long ago as 1881 to be entirely unreliable.

If there is paper on the walls, it should be scraped off and immediately burned. The floor should then be washed with a solution of corrosive sublimate 1–500, followed by soap water. The ceilings, the walls, all the woodwork, and the furniture are to be thoroughly rubbed with bread and then wiped with the corrosive sublimate solution 1–500. Esmarch has shown that bread is the best means for removing infectious material from surfaces of this kind. The micro-organisms adhere with great tenacity to the bread, which, with any crumbs that break off and fall to the floor, must be carefully collected and destroyed by fire. The room should then be aired for several days. I always advise the family, if there are other children in the house, to have the whole room, including the ceiling and the floor, painted.

You must also bear in mind that you, by means of your hair, beard and clothes, are the possible means of transmitting the contagium from one patient to another, and that it is your manifest duty to the public to change your clothing and disinfect yourselves on leaving a scarlet fever patient.

This case (Case 237), the notes of which I find in my records, will, I

PLATE VII.



rve to show you the characteristics of the benign type of scarlet
hout variation from the regular type and without complications:

four and one-half years old was noticed by me on November 6, when I was
; his sister, an infant, to be quite sick. Besides the infant the boy's two brothers,
d a half years old and the other six, were in the room with him. The mother
iat the boy had an attack of indigestion. He had been vomiting quite frequently
› appetite. His pulse was 120. His temperature was 38.3° C. (101° F.). He
dache and no sore throat, but he had the appearance somewhat characteristic of
r well marked on the hard and the soft palate. He was placed in an upper room
e and completely isolated with a trained nurse. The vomiting continued until
hen it stopped and did not return.

vember 7 he was reported to have had a restless night. His throat was found to
ch reddened and to feel a little sore. His pulse was 135. His temperature was
01° F.). He had had a natural movement of the bowels. His appetite was

necessary articles were immediately removed from the room, and he was confined
He was placed on a diet of milk and given as much water as he wished to
e efflorescence of scarlet fever very soon appeared on his chest.

vember 8 the efflorescence had spread all over his body. He was reported to
well and to have vomited his milk but once. His pulse was 125, and his tem-
is 37.7° C. (100° F.). He was sponged twice daily with water at a temperature
(90° F.), and as the skin was somewhat irritable the itching was allayed with
of vaseline. The temperature of the room was kept at 20° C. (68° F.).

vember 9 the efflorescence had spread to the limbs, and was also present to a
ee on the face. At 6 A.M. the pulse was 120, the temperature 36.6° C. (98° F.).
the temperature was 37.2° C. (99° F.), and the pulse was 120. He had a little
ite, his skin was less reddened, and his throat was not so sore.

vember 11 the efflorescence began to fade, first on the chest. On November 13
ature became normal, and desquamation began, first on the chest. On November
uamation had almost ceased, and the boy was allowed to get up and play about
r an hour. On December 1, the desquamation having almost ceased for several
gan to desquamate freely again. On December 8 the desquamation ceased. He
cted and then sent down-stairs among the rest of the children. He went out of
mber 25.

bumin was detected in his urine during the whole course of the disease. He
s usual diet on December 10.

of the other children contracted the disease, although they remained in the house
brother was sick.

ll now show you in this bed a typical case of the benign form of
ver with the distinctive efflorescence of the disease on the chest,
l face.

oy (Case 238, Plate VII., Scarlet Fever) is ten years old. He is said to have
ed to scarlet fever eight days ago. He was taken sick, with sore throat, vomit-
kened pulse, and heightened temperature, four days ago. Three days later this
e appeared, first on his neck and chest, and later it spread downward over the
extremities and upward to the face. The efflorescence is, as you see, in the form
ate erythema. You will notice that the degree of redness is much changed
is the skin is protected by the warmth of the bedclothes or is exposed for a
ess time to the temperature of the room.

is, therefore, no definite color or degree of red color which is characteristic of
r, as it is liable to vary from many causes. The vomiting ceased three days ago.
s been at times slightly delirious for the past two days, but to-day the tempera-

ture, which for the previous three days has risen to from 40° to 40.5° C. (104° to 105° F.), is beginning to fall, corresponding to the maximum of the efflorescence having been passed. His mind is now perfectly clear.

There has been until to-day a trace of albumin in his urine, but no casts have been detected, and it has been only slightly lessened in quantity.

He has no complications. Although he looks quite sick, he represents merely a pronounced and typical example of the benign and regular form of scarlet fever. On entering the hospital he was placed at once on a diet of milk. He takes and digests the milk well, and, unless some complication arises, he shall have no medicine given him, nor shall he have any food but milk for at least four weeks.

VARIATIONS IN THE BENIGN FORM.—In the benign form of scarlet fever we may have great variations from the typical manifestations of the disease which I have just described to you.

A heightened temperature in the evening sometimes continues for over a week after the efflorescence has faded, without the existence of any ascertainable cause: this occurrence should always be looked upon with suspicion. After a rapid increase of temperature at the beginning of the disease there sometimes ensues a condition of complete apyrexia, while all the other symptoms continue to develop in the usual manner. When the temperature remains heightened at the end of the period of efflorescence and continues into the period of desquamation, especially when there is no local pain anywhere, we should suspect that a nephritis may be developing. When the temperature after having become normal rises again, we should suspect such complications as otitis and suppuration of the subcutaneous tissues of the neck, or that the heart is involved.

Relapses may take place in scarlet fever. In some of these cases after the efflorescence has disappeared it may return in the second or third week, during the stage of desquamation, and even after the desquamation has ended. The symptoms of these cases are sometimes more severe than those in the first attack, but in most of the reported cases of relapse in scarlet fever the first attack has been a mild one. Such cases occur usually in older children rather than in younger, and must be sharply distinguished from the cases where a fresh infection has taken place and which are characterized as a second attack of the disease. Thomas reports a case of scarlet fever complicated by varicella, in which on the twenty-fifth day of the scarlet fever a relapse occurred, and on the twenty-sixth day a second attack of varicella developed.

Certain cases of scarlet fever have been reported in which in the latter part of the disease, and after the temperature had become normal, the temperature rose to 40°–41.1° C. (104°–106° F.), where no cause could be discovered for the hyperpyrexia, and where the patients recovered after being promptly treated with cold baths to reduce the temperature.

Scarlet fever may begin with such great cerebral excitement as to lead us to suspect meningitis, and it may not be possible to determine the diagnosis until the efflorescence has appeared, which may not be until even the eighth or ninth day.

orescence may last only twenty-four hours, or it may last fourteen
must remember that we are not to depend upon the efflorescence
our diagnosis in scarlet fever, as it may be so evanescent as to be
ognizable.

sions occurring at the onset of the disease are not, as a rule,
f a fatal issue, but when they occur later they are usually of
ort.

urrence of scarlet fever in surgical cases is of no special sig-
yond the apparently greater susceptibility of patients with open
ontract the disease. We should bear in mind the suggestion of
in the majority of these surgical cases thus far recorded the efflo-
s probably been the red rash of septicæmia, and that the reported
become rare since the gradual disappearance of septicæmia as a
n of surgical operations. Atkinson also suggests that in many
rashes may have been due to the quinine which was given to the

tion may arise from the ordinary scarlatinal inflammation of the
mbrane of the throat becoming more severe than usual and re-
xudation. The larynx in some cases may also present unusual
such as aphonia, and serious symptoms caused by a concurrent
condition of the glottis may arise and even produce a fatal issue.
in this next bed a case which represents certain variations from
symptoms which occur in the throat and nose, and which are
in their character:

(Case 239), three years old, was attacked four days ago. The invasion of the
haracterized by drowsiness, loss of appetite, malaise, slight nausea, a quick-
temperature of 39.1° C. (102.5° F.), and intense sore throat.
econd day of the disease the temperature continued to rise, and in the latter
y a punctiform erythema appeared on the neck, and later on the face and hands.
ext night he was very restless, sleeping only five or ten minutes at a time, and
of his throat, of headache, and of being very thirsty. His breathing was rather
he morning of the third day the record showed that in the past twenty-four
taken only 120 c.c. (4 ounces) of milk; he had had no movement of the bowels
ed 300 c.c. (10 ounces) of urine. His pulse was 134, his temperature 39.4° C.
d his respirations 34. In the evening the pulse was 134, temperature 39.6°
), and respirations 30. His throat continued to be painful. The whole throat
, and the tonsils were enlarged. There was a thick muco-purulent discharge
. The glands of the neck on each side were enlarged. There was consider-
n of the skin during the day, which was relieved by the occasional use of a
ing carbolic acid 4 c.c. (1 drachm) to water 473 c.c. (1 pint).
ning he was reported to have had a very restless night, to have taken 420 c.c.
f milk in the twenty-four hours, to have had one movement of the bowels,
assed 600 c.c. (20 ounces) of urine in the twenty-four hours; the temperature
101.5° F.), pulse 128, and respirations 28. The child is very irritable and rest-
e that there is a constant copious muco-purulent discharge from the nose, and
s quite frequently. The glands on each side of the neck are still considerably
e scarlatinal efflorescence has invaded the entire body, has extended over the
accompanied by considerable irritation. On examining the throat you will see
mucous membrane is very much reddened and that the tonsils are swollen.

On both tonsils, especially on the left, are some small yellowish-white spots apparently connected with the crypts. In one place these spots have coalesced. There is also considerable exudation, though apparently not of a membranous character, in the pharynx. I shall have a bacteriological examination made from the exudation in various parts of the throat. Material for this examination can be procured by means of a sterilized platinum wire, which you see can be easily used, as the child does not object to opening his mouth and allowing me to use the wire.

(Subsequent history of the case.) On the evening of the fourth day the temperature rose to 39.6° C. (103.5° F.), the pulse was 124, and the respirations were 28.

On the fifth day the report was that during the previous twenty-four hours the child had taken 540 c.c. (18 ounces) of milk and had passed 660 c.c. (22 ounces) of urine. He had slept better, but, owing to the extreme restlessness, he had been given 0.6 gramme (10 grains) of bromide of soda during the night. The efflorescence was beginning to fade. The throat was not so sore, and there was no appearance of any newly-developed morbid conditions in it.

On the sixth day of the disease the temperature in the morning was 37.7° C. (100° F.) and in the evening 39.6° C. (101.8° F.); 300 c.c. (10 ounces) of milk had been taken in the previous twenty-four hours and 915 c.c. (30½ ounces) of urine had been passed. There was decided improvement in the throat and nose.

The bacteriological report stated that in the culture made from the exudation which had been taken from the throat the Klebs-Loeffler bacillus could not be found.

On the seventh day of the disease the temperature in the morning was 37.4° C. (99.4° F.) and in the evening 38.5° C. (101.5° F.). Although the appearance of the throat had improved, the patient was very fretful, and the voice was quite hoarse.

On the eighth day the child had become much more hoarse, and was unable to speak except in a whisper. The discharge from the nose had ceased. The temperature in the morning was 37.4° C. (99.5° F.) and in the evening 38.2° C. (100.8° F.).

On the following day, the ninth from the invasion of the disease, the child was much brighter; his appetite returned, so that he took 1200 c.c. (40 ounces) of milk in the twenty-four hours, and he passed 840 c.c. (28 ounces) of urine. The swelling of the glands in the neck had almost disappeared, and the throat showed no evidence of irritation.

From this time the temperature continued to vary from 37.5° C. (99.5° F.) in the morning to 37.7° C. (100° F.) in the evening until the thirteenth day, when it became normal. Desquamation began on the eighth day and continued until the twentieth day. No other symptoms arose, and there was no disturbance in connection with the kidney. He recovered his voice on the nineteenth day.

In the benign form of scarlet fever certain cases are at times met with in which the high temperature, or the especial vulnerability of the child to the scarlet fever contagium, causes the symptoms to vary considerably from the typical form and to be unusually grave. As an instance of this class of cases I will report to you one which was seen by me in consultation with Dr. Robert P. Loring, of Newton Centre.

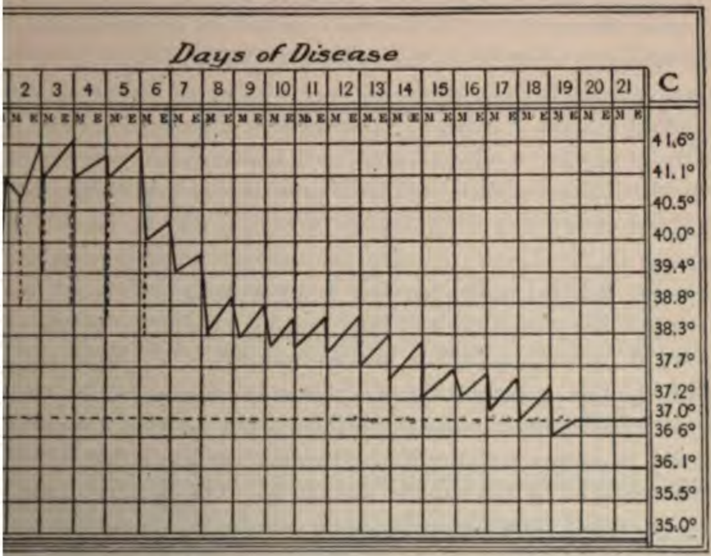
The child (Case 240) was a girl, six years old. The point of variation from the typical cases of scarlet fever was in this case an unusually high temperature. The invasion of the disease was characterized by restlessness and sore throat, which were soon followed by vomiting and delirium. The temperature on the first day rose to 41.1° C. (106° F.). The highest temperature was on the second and third days, when it reached 41.6° C. (107° F.). During the first three days the pulse could not be counted. The high temperature continued until the sixth day from the beginning of the prodromal symptoms. There was great gastro-enteric disturbance, and during the first forty-eight hours there was almost continuous vomiting. This was succeeded on the third day by frequent profuse, and often involuntary, serous discharges from the bowels. These discharges continued until the fifth day. On the fourth day a slight erythematous efflorescence appeared on the neck and chest, and on the tended all over the body and was of an intense character. On the sixth day n of pain in the wrists began, but it disappeared in twenty-four hours under ation of salicylic acid. At this time also there was considerable swelling on of the neck, which gradually disappeared in four or five days. When the its height there was considerable cyanosis, with quickened respiration. The ime was weak and difficult to count. From time to time during the attack given for the restlessness, and bromide of soda was occasionally used. Tinc-lis was given when the pulse was quick and weak and cyanosis was present, nent which was most depended upon was by bathing.

hod of bathing for the purpose of reducing the temperature was that of hild in a tub of water. Whenever the temperature reached 40.5° C. (105° F.) placed on a pillow in the bath, and was kept there until the temperature was or four degrees. The time required to accomplish this was usually from d a half hours. While the child was in the bath stimulants and milk were The temperature of the water was about that of the child, and was gradu-o about 32.4° C. (90.5° F.). During the first four days the child was either a comatose condition, and when in the bath would pass its urine and fæcal voluntarily.

temperature continued until the sixth day from the beginning of the prodromal hen it fell decidedly, from which time the baths were omitted, and the tem-inued to fall by lysis until it reached the normal degree on the sixteenth invasion of the disease. After this the child had no unusual symptoms, rapid recovery. There were no complications. The desquamation took the

he chart (Chart 15) of this case. The broken lines show the degree to which ire was reduced by the baths.

CHART 15.



Toxic symptoms and high temperature in scarlet fever treated by baths.

CATIONS AND THEIR TREATMENT.—Most of the complications in scarlet fever are due probably to the action of streptococci, ted or associated with other micro-organisms. These micro-produce serious symptoms, which are often followed by death, either directly by giving rise to septicæmic processes or indirectly by nephritis.

It is supposed that the infection which complicates scarlet fever enters the system commonly through the pharynx either by direct absorption or by inhalation of these organisms.

Throat.—In addition to the milder forms of inflammation in the throat which occur in the course of scarlet fever, this simple inflammation may be complicated by more severe lesions. In these cases there may be an exudation affecting the mucous membrane of the entire buccal cavity and throat, evidently produced by streptococci. This complication adds greatly to the severity of the scarlet fever, and is a common source of invasion of the ear and of infection of the cervical glands. In addition to lesions of this class you will at times meet with a membranous condition of the mucous membrane of the throat, the pathological lesions of which cannot be differentiated from those of diphtheria. This membranous condition is caused by the action of streptococci, and the diagnosis between these membranes and those which are produced by the Klebs-Loeffler bacillus cannot be made except by means of bacteriological examination. These more severe inflammatory conditions of the throat are not common in my experience outside of hospitals, but have been observed a number of times in our scarlet fever and diphtheria wards at the City Hospital. In these cases of streptococcus invasion the entire throat may be very much swollen, the tonsils enlarged, and the naso-pharynx affected to such a degree as almost to occlude the nares. It is necessary to make a bacteriological examination of these lesions which have been called pseudo-membranes, if we wish to determine in the early days of the disease whether or not we are dealing with a case of diphtheria. After the first three or four days in most cases there is usually so marked a clinical difference between the progress of the disease where the Klebs-Loeffler bacillus is present and that where the exudation is simply secondary to a streptococcus invasion, that we are not long in doubt as to our diagnosis, even without the decisive proof by culture. As a rule, where the Klebs-Loeffler bacillus is present the continued increase in the severity of the symptoms and the resulting exhaustion of the child show us that we are dealing with this micro-organism. We must not, however, be misled by this general rule of differential diagnosis, for there are many cases in which it is impossible to differentiate between a streptococcus invasion and an invasion of the Klebs-Loeffler bacillus either by the appearance of the throat or by the clinical symptoms. On the one hand, the streptococcus invasion may be quite as severe in its symptoms as that of the Klebs-Loeffler bacillus, while, on the other hand, true diphtheria may occur where the symptoms are as mild as any that are produced by the other micro-organisms.

The treatment of the throat in these severe secondary conditions is the same as I have already spoken of in the treatment of the benign forms, except that, if possible, it should be carried out more rigorously. As the disease

mparatively short course, there is not such a need for stimulants
icated where diphtheria is present. In young children it is often
le to treat the throat locally, and I have usually found that my
ance in tiding over the severe stage of the disease is the admin-
of sufficient food, and of stimulants when they are indicated by the
ondition of the child. It is to be remembered, of course, that the
scarlet fever may be attacked by the Klebs-Loeffler bacillus and
se brought to a fatal issue by a complicating diphtheria. When
a is present, the treatment should be the same as for a primary case
ieria; and this I shall refer to in a later lecture (Lecture XLII.,
).

e more severe forms of inflammation in the throat the inflammatory
ay go on to abscess, as in the pharynx, but the most common place
tonsil or in its neighborhood. These abscesses must be carefully
or, and when detected opened with antiseptic precautions as soon as
We shall by this treatment often shorten the course of the disease,
save loss of strength and vitality on the part of the patient.

example of one of the more severe lesions occurring as a com-
in the throat in scarlet fever, I shall report to you a case which
in my practice.

(Case 241), two and a half years old, was seized on December 4 with diarrhœa,
nd sore throat. On December 5 the tonsils were found to be enlarged and the
at much reddened. The efflorescence of scarlet fever appeared on the chest, and
emed dull and sick. The temperature was 40° C. (104° F.) and the pulse 150.
cember 6 both sides of the neck were much swollen, and the tonsils were much

cember 7 there was considerable muco-purulent discharge from the nose, and the
e was 39.7° C. (103.5° F.). The child was not so dull, and the efflorescence was
d over the whole body.

cember 8 the diarrhœa continued, and the temperature and pulse remained about
The child took milk regularly, but refused to have any applications made to its
se.

cember 10 there was no especial change in the general symptoms, except that
ea was less and the throat and nose were rather sore. There was a peculiar
ite exudation around the mouth and throat which could be easily removed. The
th sides was swollen and hard. The pulse was rather weak, 140, and stimulants
every two hours.

cember 11 there were less hardness and swelling of the neck, and less discharge
ose, the pulse was 150, the temperature was 39.4° C. (103° F.), and the efflo-
s fading.

cember 12 there was much exudation from the mouth, but the child took more
lesquamation had begun.

cember 13 the temperature was 38.8° C. (102° F.) and the pulse 150. The child
t deal, was very restless, and complained of pain in the joints, but the neck was
.

cember 14 the child vomited twice during the night. On December 15 the
35 and the temperature 38.3° C. (101° F.). On December 18 the temperature
C. (100° F.), and there was a profuse flow of saliva: the breathing sounded as
throat and posterior nares were considerably occluded.

cember 20 the pulse, which had been decidedly weak, became stronger; its rate was about 150, and the temperature was 37.2° C. (99° F.). The child seemed much brighter, and the throat was less troublesome. The pains in the legs, however, were quite severe.

On December 21 the pulse was 148 and the temperature 36.6° C. (98° F.). There was considerable discharge from the nose, and there was an efflorescence of herpes on the lips and face.

On December 23 both tonsils were found to be much enlarged and of a deep red color. The temperature from this time remained normal, and the child rapidly improved until December 28, when it complained of pain in the left ear: some hours later, perforation of the membrana tympani took place and there was a slight muco-purulent discharge.

On December 29 both ears were gently irrigated with lukewarm water. Up to this time the urine had shown no abnormal condition, but on this day it was found to contain a faint trace of albumin, and the specific gravity was 1013.5. The sediment was small, and consisted of small round renal epithelium, mucous casts, and an occasional hyaline cast, representing a condition of hyperæmia. From this time the child rapidly recovered, the temperature remained normal, the swelling and hardness of the neck entirely disappeared, the albumin and casts disappeared from the urine, and the ears recovered without resulting deafness, but for over a year there was evidence of decided thickening of the tissues of the naso-pharynx. There was no subsequent paralysis.

In this case the child resisted all attempts at treatment so strenuously that little was done beyond the administration of milk and brandy. A bacteriological examination of the exudate in the throat was not obtained, so that the Klebs-Loeffler bacillus could not be definitely excluded as a cause, but the subsequent course of the disease showed that in all probability diphtheria had not been present. It therefore represents very well the typical course, uninfluenced by drugs and special treatment, of one of the more severe forms of scarlet fever with a complication in the throat.

Cervical Glands.—The glands of the neck are more or less enlarged, according to the severity of the infection. This enlargement may in some cases be so great as to cause much swelling and distortion of the face and neck. The swelling extends at times under the chin from one ear to the other as a mass of cellulitis. The tissues of the neck under these conditions may, as I have already described to you in speaking of the pathology of the disease, suppurate, and this condition, even if it does not produce a fatal result from gangrene, may greatly prolong the period of convalescence.

While the glands are enlarged and tender, the application of hot fomentations usually gives much relief, as does also in some cases an ice poultice. Beyond this I am not in the habit of making any external application.

Ear.—The middle ear is so closely connected by means of the Eustachian tubes with the naso-pharynx that aural complications are exceedingly common where naso-pharyngeal irritation exists. I shall, therefore, next speak of the complications which arise in the ear during the course of scarlet fever.

The symptoms which indicate that a secondary infection of the ear is taking place are not always clear, as they may differ much in their manifestations. We should therefore watch with the greatest solicitude and examine with the greatest care the ear during the course of scarlet fever. The symptoms may be active and represented by aural pain and great restlessness. On the other hand, there may be no apparent pain, especially in infants and young children, who are often unable to indicate the location of the pains by y means of a Politzer bag. Pain should be combated by the a of a solution of atropine in glycerin and water into the ear (Pre- 70) and by the application of dry warmth. In addition to this, an uld, if required, be given internally.

PRESCRIPTION 70.

Metric.	Gramma.		*Apothecary.*	
e sulphatis	0 | 06	℞	Atropinæ sulphatis	gr. i;
i,			Glycerini,	
il.	āā 3 | 75		Aq. destil.	āā ℥i.
			M.	

Three or four drops to be warmed and dropped into the ear once every three

ongestion should be controlled as far as possible by the internal ation of bromide of potassium in small and frequently repeated these measures fail to give relief, and if there is an increase of ion in the middle ear, as shown by marked swelling and conges- ially of the superior posterior portion of the membrana tympani, lging of the membrane, which is seen to be pressed outward by n the tympanum, paracentesis with the knife should be performed, ith antiseptic precautions and under good illumination. In the es of congestion a crescentic incision carried along the superior border of the membrana tympani through the congested region, ting in free hemorrhage, will often cut short an acute process. cision in the most prominent portion of a bulging membrana by giving a vent to the contained pus, may result in speedy relief pain and fever, and justifiably forestall the effort which nature to obtain this relief. In the acute congestive stage, after incision embrana tympani drainage-wicks made of dry absorbent cotton applied, and covered at their outer end with a pad of absorbent ing the concha. These wicks should be renewed as often as both means of the air-douche used in accordance with the evidence afforded by hearing-tests and by the objective examinations. In cases where there is perforation of the membrana tympani with continued suppurative discharge, thorough cleansing should be employed. If under this treatment improvement does not soon take place, the patient should be referred to an aurist.

I have already referred to the importance of detecting at once a complication of the ear during the progress of a case of scarlet fever and immediately treating it. Children are so often rendered deaf by the morbid processes resulting from the scarlet fever contagium that it becomes a positive duty for the attending physician to watch the ear as carefully in these cases as he would watch the heart in a case of rheumatism. In addition to the danger arising from a chronic disturbance of the tissues of the ear, you must carefully look for any evidence of the rapid extension of secondary infection from the naso-pharynx to the middle ear, and thence through the petro-squamosal suture to the cerebral meninges, a series of complications which usually proves fatal.

I recently saw a case in consultation with Dr. Forster which illustrates the danger of not treating promptly and thoroughly the complication of otitis in cases of scarlet fever.

A child (Case 242) two and a half years old had been attacked with scarlet fever and later with a complicating purulent otitis. When I saw the child it was lying in a state of stupor, apparently induced by pressure on the cerebral blood-vessels of an unusually large collection of pus in the middle ear through the petro-squamosal suture. In this case rupture had taken place in both membranæ tympani, and the pus was flowing in large quantities from the external meatus. A careful examination by Professor J. O. Green showed, however, that the perforations of the membranæ tympani were very minute, and the cerebral stupor was not relieved until a free opening was made in each tympanum and the entire middle ear thoroughly syringed out. Although the symptoms of pressure were relieved by these procedures, secondary infection of the cerebral meninges had already taken place, and the boy subsequently died of an acute purulent meningitis.

This case warns us that we should not be misled by the idea that a simple flow of pus from the auricle is necessarily sufficient to provide a proper exit for collections of pus in the middle ear, and that, unless the case is in the hands of an expert aurist, cerebral pressure or purulent meningitis is likely to occur at any time. It also represents a class of cases to which I shall refer again when speaking of meningitis, and illustrates one of the secondary forms of that disease.

Kidney.—I have spoken somewhat at length in the earlier part of this lecture concerning the albuminuria which is present in the different stages of scarlet fever, and also of the different forms of nephritis which may occur. What I hope I have impressed upon you is the great importance of detecting by means of frequent analyses of the urine the beginning of either the milder forms of renal disturbance or the more severe forms of nephritis, usually represented by that which is called capsular glomerulo-nephritis. If carefully watched for, the appearance of albumin will almost always precede the clinical symptoms, and by a still more rigid enforcement of the rules which I have laid down as practically governing the treatment the further development of a nephritis may be prevented or at least rendered nuch less pronounced. It is quite frequently the case that a suspicion is irst aroused of the presence of a nephritis either by vomiting or by œdema f the face, especially about the eyes, and commonly occurring during the eriod of desquamation, from the eighteenth to the twenty-fourth day. Jnder these circumstances the urine will be found to be diminished in uantity and to contain albumin. The daily amount of the urine may be educed as low as 100 c.c. (3⅓ ounces), or even lower. The microscopic xamination of the urine does not differ materially from that which results rom the other forms of nephritis in their early stages, but later you may ossibly find that fatty casts are less numerous in the nephritis of scarlet ever, because there is less fatty degeneration in the renal epithelium. The arlier in the course of the disease the symptoms of nephritis appear, the nore severe, as a rule, will be its type. The extent of the albuminuria s of less consequence than the total quantity of the urine. A rapid and xtensive diminution of the urine is ominous, as it indicates the accumuation of nitrogenous waste in the blood and the danger of a resulting ræmia. The albumin occurring early in the disease is more apt to be n large quantities than when it appears first in the third or fourth week. Iæmaturia is frequently present in this form of nephritis, but ordinarily f itself adds little to the gravity of the disease. The œdema of the face nay be followed by a rapid involvement of the ankles and legs and at times nay become general. During the course of a general œdema the desquamaion is apt to cease, returning on its disappearance. The œdema may last or months or may pass away quickly; it may be entirely absent, but in uch cases the nephritis is almost invariably of a light grade.

At times during the presence of a general œdema serous effusions into he pleura may occur. Œdema of the lungs and brain, though rather rare, may also take place. Instead of a slow development beginning with œdema of the face we may have an acute attack, ushered in by fever, vomiting, headache, œdema, amblyopia, coma, and convulsions.

Relapses may occur many weeks after an attack of scarlatinal nephritis, and we should watch the case with the greatest care for several months. The nephritis of scarlet fever, although it may last for months, has a tendency in children ultimately to recover, on account of their wonderful recuperative powers. It is also rare for the renal disease following scarlet fever to become chronic.

Retinitis and amaurosis at times occur during the progress of the ephritis in scarlet fever. In these cases of amaurosis it has been noticed hat, although the loss of sight may be complete, almost always where ræmia and amaurosis are coincident there are found no perceptible change n the retina, no congestion of the papillæ, no increase of intra-cranial ressure, and no intense œdema of the brain. The sight, under these cirumstances, may be recovered completely.

The alterations in the glomeruli already spoken of not only cause the anuria and the uræmia, but also obstruct the renal arteries, as very nearly all the renal blood has to pass through the glomeruli. We find in quite a large number of cases of capsular glomerulo-nephritis a rapid hypertrophy of the left ventricle. This cardiac complication is not to be confounded with the endocarditis which I have already spoken of as secondary to the scarlet fever, and which is supposed to be caused by its special poison or by the streptococci which I have already described as being present in the disease. It is, in fact, not the direct result of the scarlet fever, but is secondary to the nephritis, and is, in this sense, tertiary to the scarlet fever. We therefore do not find this acute cardiac hypertrophy in the earlier stages of scarlet fever, but when a capsular glomerulo-nephritis is once established it may take place in so short a period as a week. This rapid hypertrophy has usually been observed in children between the ages of three and six years, which is of some significance in explaining why this hypertrophy should take place so easily. If you will recall what I have already told you in my lecture on development (Lecture IV., page 122), you will understand that between the ages of three and eight years a physiological hypertrophy of the heart exists, possibly caused by a continuance of the aortic narrowing in the neighborhood of the ductus arteriosus, and that the heart will be more readily affected by increased blood-pressure at that age. This tendency to change in the cardiac muscles is also accentuated by the rapid growth of the organ at this period of life. Besides the cardiac hypertrophy we may, at times, have an acute dilatation of the heart in these cases. This is a serious complication, which must be guarded against, and when it occurs must be recognized at once. These cardiac complications very frequently recover completely, as it is seldom that any extensive changes in the muscles of the heart take place.

Although the occurrence of sugar in the urine during the course of scarlet fever is very rare, yet it is well to examine the urine for this element in cases of scarlet fever. By taking this precaution it will sometimes be possible to explain some otherwise obscure symptoms which may arise.

Dr. Zinn, of Bamberg, reports the case (Case 243) of a boy, four years old, previously strong and healthy, who was attacked with scarlet fever and diphtheria on January 27. The diphtheria was light in form and gradually subsided, but on the thirteenth day from the time when the child was seized with scarlet fever an otitis externa appeared, accompanied by excessive vomiting and by the rapid development of œdema and ascites. The urine showed evidence of nephritis by being lessened in quantity and by containing a large amount of albumin and numerous casts and blood-corpuscles. After a few days the more dangerous symptoms passed off, and the patient was treated with hot baths and injections of pilocarpine. Although the appetite improved considerably, the child's strength did not return, and he remained in bed during the whole of March. Early in April, on attempting to walk he was found to have paralysis of the right leg, which soon passed off. At this time there was a slight trace of albumin in the urine. He then began to show an increased action of the heart, and an examination of the urine on the 10th of April showed that the specific gravity was 1030 and that it contained a considerable amount of sugar. The total amount of urine passed in twenty-four hours was somewhat decreased. The appetite at this time was good, the thirst was not noticeably increased, and nothing else abnormal was discovered. The child was placed on a diet of meat, milk, eggs, and red wine, and

April there was only one per cent. of sugar in the urine, and by the middle
ne-fourth of one per cent. From this time the child improved in strength
ed to have a mixed diet. By the middle of June the urine was found to
ugar and albumin, and the child became as strong and as well as ever.

already told you that very little treatment beyond hygienic
needed for the mild uncomplicated cases of scarlet fever.
ardly be said of the cases that are complicated with severe
ephritis, for in these we must act promptly and with great

uld be careful about using diuretics which might irritate the
etate of potash is one of the safer diuretics in this complication.
er cases a lemonade made with bitartrate of potash will be taken
ill often quickly increase the quantity of the urine, reduce the
inish the albumin, and cause a radical change for the better.
ade may be made by using 4 c.c. (1 drachm) of bitartrate of
73 c.c. (1 pint) of boiling water into which a lemon cut in thin
en dropped. This quantity a little sweetened may be drunk in
hours by a child five years old.
e cases with general œdema and threatening uræmia cathartics
ore certain in their action than diaphoretics and diuretics, and
y indicated where, as is usually the case, constipation is present.
in doses of 0.006 gramme ($\frac{1}{10}$ grain) may be given to a child
ld, and repeated a number of times. It usually acts quickly.
nd jalap powder in doses of 0.3–0.6 gramme (5–10 grains) may
en where a rapid and decided derivation by the intestine is

provided for the proper movement of the bowels, if the skin is
y, and uræmic symptoms, usually represented by anuria, som-
blyopia, and headache, are present, the hot pack, either wet
uld be resorted to. I prefer in these cases to have the child
a blanket and placed directly in a tub containing water at a
of 40.5°–43.3° C. (105°–110° F.). The child should be kept
r fifteen or twenty minutes, and even longer if necessary, and
be taken from the wet blanket, enveloped in hot, dry blankets,
them until the skin has become moist and reaction has taken
ile the child is in the bath, milk can be given to it, and stimulants
indicated by a weak or an intermittent pulse.
tion to this treatment, muriate of pilocarpine in doses of 0.003
grain) should be given by the mouth to a child of two years,
aneously, if desired, to a child five years of age. In these
eatening uræmia, convulsions sometimes appear quite suddenly.
circumstances enemata of hydrate of chloral, 0.3–0.6 gramme
s) dissolved in water, are of value in controlling these nervous
I myself prefer to use a combination of bromide of potash
of chloral, such as you see in this prescription (Prescription 71):

PRESCRIPTION 71.

Metric.	Gramma.	Apothecary.	
℞ Chloral. hydrat.	7 | 5	℞ Chloral hydrat.	[illegible]
Potassii brom.	15 | 0	Potassii brom.	ʒ [illegible]
Aq. destil.	90 | 0	Aq. destil.	[illegible]
M.		M.	

Sig.—3.75 c.c. (1 drachm) in 30 c.c. (1 ounce) of warm water: to be given by ene[illegible] and repeated in half an hour if needed.

Where the ascites is extreme, paracentesis abdominis is often of great value, not only in relieving the pressure, but also in increasing the action of the diuretic, which, perhaps, before was not acting freely. Digitalis is a valuable remedy especially adapted to the treatment of the nephritis of scarlet fever and to that of the cardiac changes which result from it. By the administration of this drug the flow of urine is increased. It is best given in the form of a freshly prepared infusion, in teaspoonful doses every four hours to a child five years old. Diuretin, 0.3 gramme (5 grains), dissolved in water and given two or three times in the twenty-four hours, has proved of considerable value in my cases, and is apparently harmless.

I speak of special ages, such as five years or two years, merely as a guide by which you can judge what the proper doses should be at the other ages.

In addition to these more common complications of scarlet fever a number of secondary infections are at times met with. Thus, cases of purpura following or complicating scarlet fever have been reported, and are usually fatal.

An acute inflammation of the joints, usually the larger ones, is not infrequently met with during the course of scarlet fever. This acute synovitis is at times apparently either due to or closely connected with rheumatism, and may be accompanied by endocarditis and pericarditis. The latter disease is, however, rarely met with unless in the later stages of scarlet fever in cases where nephritis has developed. These rheumatic cases are usually controlled by the administration of salicylic acid. As a rule, they are not of long duration, and if effusion takes place in the joints it is serous, does not become purulent, and does not give an especially serious prognosis.

In connection with these cases, either uncomplicated or where the heart is also affected, chorea has sometimes arisen as a complication.

A more severe form of synovitis, apparently caused by sepsis, may also occur during the course of scarlet fever. The effusion in the joints in these cases may become purulent and lead to serious and permanent disorganization of the tissues and often to death from general septic infection.

Besides these acute inflammations of the joints a chronic process at times arises, appearing, as a rule, very late in the disease or subsequent to it by many months. This inflammation is tubercular in character, and affects with especial frequency the hip and knee. Although tubercular, it seems to be a late result of the original toxic effect of the micro-organisms of or secondarily connected with the scarlet fever contagium.

A case which I saw in consultation with Dr. Miller, of Providence, represents so well one of the milder forms of what was probably capsular glomerulo-nephritis, and the effect of rest in the treatment of the disease, that I shall report it to you.

A girl (Case 244), five years old, was attacked by scarlet fever of the benign form and very mild in its character. After the usual prodromal symptoms the efflorescence appeared and ran its course, and desquamation became established. At the end of the second week, and while the desquamation was still present, the child seemed so well that it was allowed to be dressed and about its room. It was also allowed to have its usual food, which included a considerable amount of meat.

On January 4 the child was very irritable during the day, and passed her urine involuntarily in the forenoon. During the afternoon she was feverish, and passed frequently small amounts of urine. That night she slept well, but on awaking on the morning of January 5 she seemed dull, and was said to be feverish and to have little appetite.

On January 6 the record stated that she had passed only 90 c.c. (3 ounces) of urine in the twenty-four hours. She seemed tired and languid, and there was an œdematous condition of the eyes and upper part of the face. She had one normal movement of the bowels.

On January 7 the total amount of urine passed in the twenty-four hours was 480 c.c. (16 ounces). She was given infusion of digitalis and cream of tartar water on this day, and placed on a diet of milk.

On January 8 she seemed better, and passed 480 c.c. (16 ounces) of urine in the twenty-four hours. She was then allowed to have an increase in her diet, consisting of broth and various kinds of soups. An examination of the urine (Analysis 61) by Professor E. S. Wood on this day gave the following result:

ANALYSIS 61.

Color	Rather pale.
Reaction	Acid.
Urophæin	Diminished.
Indoxyl	Increased.
Urea	Diminished.
Uric acid	Increased.
Albumin	Considerable trace.
Sugar	Absent.
Bile-pigments	Absent.
Specific gravity	1009.
Chlorides	Almost absent.
Earthy phosphates	Diminished.
Alkaline phosphates	Diminished.
Sediment	Slight in amount; consisted chiefly of normal blood-globules, a few renal cells, and a few hyaline, fibrinous, blood, and epithelial casts. The blood-globules and the casts were normal in appearance.

In regard to this examination Professor Wood remarks that the important features of the urine were its dilution, the great diminution in the normal salts, especially in the chlorides, the considerable trace of albumin, and the blood and casts. The normal character of the blood-globules and the comparatively small number of the casts seemed to show that only a small portion of the kidney was affected. At the time of the great diminution in the quantity of the urine the tubules were probably nearly completely blocked up. The low specific gravity and the great diminution of the urea and chlorides seem to indicate that it would need but little additional irritation to produce a marked nephritis. The present condition seems to be one of a mild nephritis.

The general symptoms presented by the child and the disturbance of the kidney shown by the examination of the urine made me advise that she should be kept in bed in a warm room and placed on a diet exclusively of milk. A warm bath was to be given once or twice daily until a larger amount of urine was passed, and 4 c.c. (1 drachm) of infusion of digitalis administered four times in the twenty-four hours.

On January 9 the total amount of urine passed in the twenty-four hours was reduced to 90 c.c. (3 ounces), and the child was nauseated and vomited a number of times during the day.

On January 10 she was reported to have had a very restless night and to have been very much excited on waking. She had no pain anywhere. Her face continued to be œdematous. The total amount of urine passed in the twenty-four hours was 240 c.c. (8 ounces). She perspired slightly, and had one large, loose dejection. She so absolutely refused to take milk that she was given 103 c.c. (3½ ounces) of beef juice, which was all the nourishment that she took on this day.

On January 11 the face was more œdematous, and she was languid. She had two large, loose, offensive dejections from the bowels, and complained of a burning sensation in the rectum at the time of the movements. The total quantity of urine was 300 c.c. (10 ounces). On this day she was finally persuaded to take milk, and no other food was given to her.

On January 12 the child seemed brighter and the face was not so much swollen. The total amount of urine in the twenty-four hours increased to 540 c.c. (18 ounces), and an analysis (Analysis 62) made by Professor Wood gave the following results:

ANALYSIS 62.

Color	Normal.
Reaction	Acid.
Urophæin	Diminished.
Indoxyl	Increased.
Urea	Slightly diminished.
Uric acid	Increased.
Albumin	A slight trace, and less than on January 8.
Sugar	Absent.
Bile-pigments	Absent.
Specific gravity . . .	1014.
Chlorides	Almost absent.
Earthy phosphates. .	Diminished.
Alkaline phosphates .	Diminished.
Sediment	Considerable in amount, and consisting chiefly of numerous blood-globules, a few renal cells, an occasional hyaline and blood cast, and an occasional small epithelial cast.

This specimen showed that improvement had taken place in the condition of the kidney since the previous examination, as the albumin had lessened in quantity and the urea had increased.

On January 13 the total quantity of urine had increased to 1410 c.c. (47 ounces). The child seemed very well, and was reported to have slept quietly all night. An analysis of the urine showed the specific gravity to be 1011. The chlorides, though still much diminished, were beginning to reappear, which was a very favorable symptom.

On January 14 the total amount of urine was 1545 c.c. (51½ ounces). The child continued to improve in appearance, and seemed bright and well.

On January 15 the total quantity of urine was 1440 c.c. (48 ounces), and on January 16 it was 1035 c.c. (34½ ounces).

During the rest of the attack there was no notable change in the total amount of urine passed in twenty-four hours. The results of the analyses of the urine which were made from time to time showed that there was some process going on beyond a simple hyperæmia existing in the kidney. The urea remained diminished until the 12th of March, when it was found to be increased, and on the 22d of March it was normal. The chlorides continued to be diminished until April 7, when they were reported to be normal. The specific gravity remained below 1020 until April 18, when it became 1024. A slight trace of albumin continued to be found until the following autumn. An analysis (Analysis 63) of the urine made September 25 by Professor Wood gave the following results:

ANALYSIS 68.

	Normal.
on	Acid.
ein	Normal.
yl	Normal.
	Normal.
cid	Normal.
in	Very slightest possible trace.
des	Normal.
phosphates	Normal.
ne phosphates	Normal.
c gravity	1017.
ent	Slight, and consisting of a very few normal blood-globules. Slight excess of small round cells and of cells like those from the neck of the bladder.

gh a very prolonged search was made for casts, none were found. Professor idered that at this date the kidneys had practically recovered, as they were doing ormal work. The blood probably came from the neighborhood of the urethra, s irritation in that locality.

il the child was allowed to have, besides her diet of milk, some broth and bread and in May she was given meat. She was kept in bed until the latter part of March. the course of her sickness various attempts were made to increase her diet more l to allow her to be dressed and about the room, but each time when this was owed symptoms which pointed towards the presence of a renal complication, welling of the eyes and face and a rise of temperature, with resulting nausea appetite.

ase shows how careful we must be for many weeks and even months to control ture of the room, the amount of exercise, and the kind of food, where a nephritis ated a case of scarlet fever. It also shows how entire recovery may take place the renal irritation is pronounced and unusually prolonged.

table (Table 94) gives the record of the total amount of urine each twenty-four hours for ninety-two days.

TABLE 94.

C.c.	Ounces.	Days.	C.c.	Ounces.
90	3	21	855	28½
480	16	22	900	30
480	16	23	1020	34
90	3	24	1125	37½
240	8	25	1020	34
300	10	26	1185	39½
540	18	27	975	32½
1410	47	28	1260	42
1545	51½	29	990	33
1440	48	30	1155	38½
1035	34½	31	1230	41
930	31	32	1230	41
915	30½	33	1125	37½
930	31	34	1185	39½
900	30	35	1185	39½
1065	35½	36	1050	35
1095	36½	37	1005	33½
1065	35½	38	990	33
1140	38	39	1020	34
1020	34	40	1290	43

TABLE 94.—*Continued.*

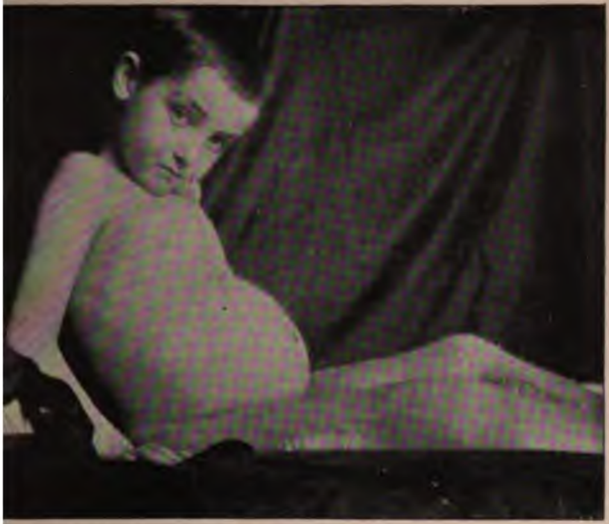
Days.	C.c.	Ounces.	Days.	C.c.	Ounces.
41	1170	39	67	900	30
42	1215	40½	68	1275	42½
43	1020	34	69	1230	41
44	1110	37	70	1140	38
45	1095	36½	71	1275	42½
46	1425	47½	72	1185	39½
47	1305	43½	73	1230	41
48	1125	37½	74	1380	46
49	1230	41	75	1275	42½
50	1125	37½	76	1260	42
51	1155	38½	77	1230	41
52	1080	36½	78	1215	40½
53	1005	33½	79	1230	41
54	1080	36½	80	1140	38
55	1200	40	81	1230	41
56	915	30½	82	1305	43½
57	1215	40½	83	1230	41
58	1335	44½	84	1170	39
59	1245	41½	85	1200	40
60	1095	36½	86	970	29
61	1040	35	87	735	24½
62	1050	35	88	1235	34½
63	975	32½	89	930	31
64	935	31½	90	885	29½
65	990	33	91	885	29½
66	1050	35	92	1065	35½

This table (Table 95) shows the record of the total amount of milk taken by the child in each twenty-four hours during thirty-one days. Milk was her exclusive diet during these days, and although, as I have already told you, in the beginning of her sickness she disliked and refused to take milk, she was, nevertheless, persuaded to take it, and finally did so without resistance. The table is instructive as showing the amount of milk which is sufficient for nourishment for a child of this age.

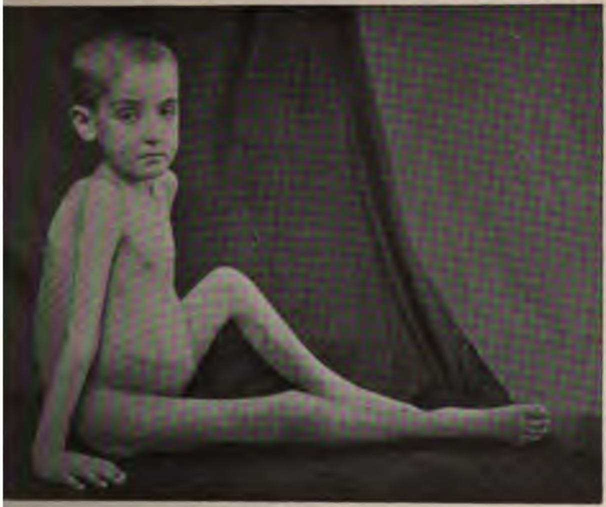
TABLE 95.

Days.	C.c.	Ounces.	Days.	C.c.	Ounces.
1	630	21	17	1260	42
2	1440	48	18	1620	54
3	1530	51	19	1530	51
4	1440	48	20	1440	48
5	1440	48	21	1530	51
6	1440	48	22	1530	51
7	1440	48	23	1530	51
8	1260	42	24	1530	51
9	1260	42	25	1620	54
10	1260	42	26	1620	54
11	1260	42	27	1620	54
12	1260	42	28	1620	54
13	1440	48	29	1620	54
14	1350	45	30	1620	54
15	1350	45	31	1620	54
16	1350	45			

CASE 245.



I.—Before treatment.



II.—After treatment.

Scarlet fever. Nephritis; enlargement of the heart.

his boy (Case 245, I.) whom you see here in the convalescent ward illustrative case of scarlet fever complicated by a probable capsular erulo-nephritis and a resulting cardiac enlargement.

e is seven years old, and entered the hospital on July 28. His mother is living and and states that his father died of Bright's disease. The child is said to have been well eighteen months ago, when he had an attack of scarlet fever, mild in form and not panied by any severe symptoms. In the latter part of the attack his temperature rose, e began to have dyspnœa and dropsy. Since that time he has been slowly but steadily ng worse. As you see, he has extensive œdema of the face, chest, arms, abdomen, egs. He is somewhat cyanotic, and his breathing is so much affected that he is unable down, the orthopnœa compelling him to be supported in a semi-recumbent position. oser examination you see that there is a slight puffiness about both eyes, that there is ow tinge of the conjunctivæ, and that the lips and tongue are cyanotic. The extremi-re cold to the touch, and their skin pits readily on pressure. The skin of the whole is dry and harsh and in certain portions is covered with fine scales. On the inner side e left leg and on the outer side of the right leg are some old scars, apparently resulting a previous scarification performed for the reduction of the anasarca. In addition to dematous condition of the walls of the abdomen, a distinct fluctuation is found on pal-n, showing that there is fluid in the abdominal cavity. An examination of the lungs s that there is dulness over both bases behind, and over these areas of dulness, as well er the whole front of the chest, fine moist râles can be heard, indicating an œdematous ition of the lungs. On examining the heart, I find that its impulse is most distinct e sixth interspace a little outside of the mammary line. The area of cardiac dulness ds from the second rib on the left to 2.5 cm. (1 inch) to the right of the sternum, in ea corresponding to the third interspace and fourth rib. The dulness then extends to eft across the sternum to a point 2.5 cm. (1 inch) outside of the mammary line and as s the sixth interspace, corresponding to the cardiac impulse. A loud systolic murmur e heard over the region of the cardiac impulse, and is transmitted so that it can be l in every part of the thorax. The total amount of urine in twenty-four hours has d from 900 to 1050 c.c. (30–35 ounces). An analysis (Analysis 64) of the urine gives ollowing results:

ANALYSIS 64.

olor	Darker than normal.
pecific gravity	1013.
eaction	Acid.
rophæin	Diminished.
ndican	Increased.
hlorides	Diminished.
lbumin	$\frac{4}{10}$ per cent.
ugar	Absent.
ediment	Very slight and flocculent. Microscopic examination shows numerous short hyaline and granular casts of medium diameter and occasionally of small diameter; an excess of renal epithelium; considerable abnormal blood; an occasional white corpuscle; one or two blood-casts, many hyaline and granular casts, with one or more renal cells adherent; occasional fatty renal cells and casts with a few fat-drops adherent.

On entering the hospital yesterday he had a slight diarrhœa. You see that to-day he is ble to lie down with comfort, on account of the dypsnœa arising from an accumulation luid in the abdomen. The legs are also very much swollen and œdematous. His face is ewhat puffy. The cyanosis is so marked and the child is in so much distress that it is lent that immediate relief should be given not only to the general symptoms, but also to the great tax which is being imposed upon the already disabled heart. Unless some relief to these symptoms is given, it is very likely that he will die suddenly from heart-failure. I shall, therefore, withdraw a certain amount of fluid from the abdominal cavity, which will, I think, be followed by considerable relief to the dypsnœa. You see that I have first assured myself that the bladder is empty, have then had the child supported on the side of the bed with the legs apart, have introduced a trocar into the median line of the abdomen just below the umbilicus, and have withdrawn 480 c.c. (16 ounces) of clear, yellowish fluid. You will notice that the child already breathes with much greater freedom and that the cyanosis is decidedly diminished.

This case illustrates some of the points in the pathology and clinical symptoms of scarlet fever to which I have already referred. Of course at as late a stage of the disease as that when the boy entered the hospital it would be impossible to make a definite diagnosis as to the condition of the kidney and heart which may have existed at an earlier stage. It is possible that during the stage of efflorescence an endocarditis such as might complicate the earlier symptoms of scarlet fever may have been present and may have been followed by a pathological lesion of the valves. The history of the case, however, states that the course of the scarlet fever was a mild one in its early stages, and that whatever complications followed arose at a later stage of the disease, during desquamation. It would seem probable, therefore, that the symptoms of œdema and cyanosis which appear in this later stage of the disease were caused by a disturbance of the kidney. As I have already told you, a renal complication is most common in the later stages of scarlet fever. The physical examination made when the child entered the hospital showed that there was a complication of the heart, represented by cardiac enlargement and a mitral systolic murmur. The examination of the urine shows us that it is probable that there are organic changes in the kidney as well as in the heart, although we cannot say definitely that such conditions as we find in the urine have not been produced by a cardiac lesion followed by passive congestion of the kidney. Therefore, although we cannot decide without a post-mortem examination whether both kidney and heart are affected, we can at least suppose that the following sequence of complications has resulted and has produced the present clinical symptoms.

The child had scarlet fever in a mild and apparently uncomplicated form until he reached the stage of desquamation. During the latter part of this stage a lesion of the kidney, presumably of the capsular glomerular form, occurred, and, owing to the increased blood-pressure which finally resulted from the changes in the kidney, hypertrophy of the heart, presumably followed by dilatation, appeared. If I have correctly read this sequence of lesions, we have, then, cardiac enlargement secondary to a renal disease and tertiary to the original scarlet fever contagium.

The prognosis in this case is very unfavorable. Although we know that in children dilatation of the heart may be entirely recovered from, yet as long as this condition exists there is danger of sudden death from cardiac failure. Where the cardiac dilatation results from extensive disease of the kidney, especially in the form which we most commonly meet with in scarlet fever, capsular glomerulo-nephritis, the chances are that this failure will take place before the nephritis has been recovered from when a patient has been reduced to such a degree as is the case with this boy.

The treatment should be absolute rest, so as not to tax the muscles of the heart mo than can possibly be avoided. To relieve the intra-abdominal pressure, which augments t œdema of the lungs and interferes with the action of the heart, paracentesis of the abdom should be performed, as I have just shown you. Hot baths should be given to incre the action of the skin, laxatives to relieve the congested condition of the kidneys, and n irritating diuretics, such as acetate of potash and digitalis, are indicated. Nitro-glyce is valuable where the action of the heart at any time becomes suddenly feeble a irregular.

(Subsequent history of the case.) For the next few days after paracentesis of t abdomen the child improved greatly, the dyspnœa ceased, the urine became of a better co and increased in amount, the cyanosis grew less, and, although the pulse was still small a feeble, the child showed great general improvement. In the course of a month the œdema so much reduced that the child looked like a different person (Case 245, II., facing page 5

was able to lie down with comfort, slept well, his appetite returned, and at one time he
d even be moved about the ward in a wheel-chair. Some weeks later the cardiac symp-
returned, and he again began to have œdema and ascites, cyanosis and orthopnœa. The
e, as you see in the table (Table 96), varied considerably, but at no time did it show the
t lessening which is found in cases of threatening uræmia. The symptoms were, indeed,
ly those of a crippled heart. At one time the temperature, without any assignable
e, rose to 41.1° C. (106° F.), and somewhat later it became subnormal. On September
ascites had increased to such a degree that paracentesis of the abdomen had to be again
rmed.

On September 8 the œdema increased, and the urine was reduced to 450 c.c. (15 ounces).
etin was given in doses of 0.6 gramme (10 grains), which increased the flow of urine
230 c.c. (41 ounces). The diuretin given in these doses once or twice a day for some
continued to act successfully.

In October the action of the heart grew still weaker, the œdema of the lungs increased,
although there had been a general improvement, the child grew progressively weaker
ng November. Early in December he was attacked with vomiting, had a weak and
d pulse, gradually failed in strength, and on the 21st of December died suddenly. No
psy was obtained.

The total amount of urine in this case, measured daily from July 29 to September 15,
as follows:

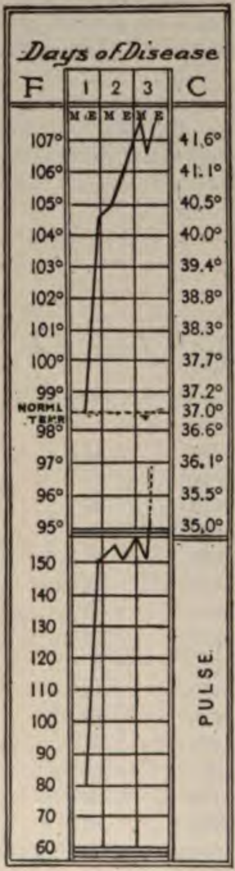
TABLE 96.

tal amount of urine passed in twenty-four hours during thirty-one days in a case of scarlet fever complicated by nephritis and resulting in cardiac dilatation.)

Date.	C.c.	Ounces.
July 29	1440	48
July 30	1230	41
July 31	810	27
August 1	810	27
August 2	900	30
August 3	600	20
August 4	1320	44
August 5	990	33
August 6	630	21
August 7	510	17
August 8	510	17
August 9	840	28
August 10	1020	34
August 11	1020	34
August 12	720	24
August 13	1020	34
August 14	690	23
August 22	840	28
August 23	990	33
August 28	600	20
August 29	750	25
August 30	480	16
August 31	600	20
September 1	840	28
September 2	480	16
September 3	900	30
September 4	540	18
September 7	450	15
September 10	1230	41
September 14	660	22
September 15	750	25

Malignant Form.—I have told you in the beginning of this lecture that there are two distinct forms of scarlet fever, and I have spoken at length of the benign form, with its variations and complications, and its, as a rule, favorable prognosis. I shall have but a few words to say of the malignant form of scarlet fever, for it is almost without exception fatal, and is very rare in comparison with the benign form. Malignant scarlet fever appears to attack those individuals who have a predisposition to be profoundly affected by the scarlet fever contagium. In these cases we see healthy children attacked with intense headache, high fever, delirium, sometimes coma, and death follows usually in two or three days. A case of this kind was seen by me in consultation with Dr. Emerson, of Concord, and represents so well the conditions which are present in these cases of scarlet fever that I shall report it to you.

CHART 16.



Malignant form of scarlet fever. Girl, 11 years old.

A girl (Case 246), eleven years old, was perfectly well and strong and had no other diseases up to January 10. In the middle of the day she felt very ill and vomited. Her pulse was 150, temperature 40.2° C. (104.5° F.). The pharynx and tonsils were much reddened, but there was no exudation or membrane to be seen. An efflorescence of a scarlatinal type appeared on the chest in the afternoon. The vomiting continued through the night and up to the morning of January 11. The child was conscious, but dull. The pulse was 150, and the temperature was 40.5° C. (105° F.). At 4 P.M. the face became puffy, and the efflorescence was well marked on the body and extended to the extremities. The child was wandering and stupid, and the temperature rose to 42.2° C. (108° F.). The extremities became livid, and the vomiting began again. At 6.30 P.M. the temperature, after the internal administration of various remedies, was found to be 41.1° C. (107° F.), and at 10 P.M. 41.1° C. (106° F.), and the pulse 160, weak and difficult to count. At 6 A.M. on the 12th, forty-eight hours from the appearance of the first symptoms, the child died.

The case was a perfectly hopeless one from the beginning, as every method of treatment which could be thought of was tried and proved absolutely fruitless. Tub bathing with water at different temperatures, and finally sponging with ice-water, had no effect whatever on the temperature or the general symptoms.

This chart (Chart 16) shows the temperature from the time of the attack to within a few hours before death.

LECTURE XXVI.

THE EXANTHEMATA.—(Concluded.)

MEASLES.—RUBELLA.

MEASLES (Rubeola).—Measles is one of the most common diseases of ldhood, and has been known for many centuries. It is an acute infectious ease, evidently caused by a specific micro-organism. It is characterized lachrymation, photophobia, coryza, cough, a papular efflorescence, and light desquamation. The micro-organism which produces measles has t yet been determined. It is supposed to find its vehicle in the tears, and the secretion of the throat and nose, and possibly to exist in the blood. tenacity for clothing, thus continuing as a fresh source of infection, is ld in comparison with that of scarlet fever. It is very infectious, and some communities is at times exceedingly fatal. This was the case in epidemic of 1873 in the Fiji Islands, where it had not occurred for a g time; it spread rapidly, and caused two thousand deaths, of which ty-seven per cent. were in children under five years of age. The high rtality in measles is, as a rule, not caused by the measles itself, but by its mplications. The epidemics of measles, as I have already told you in mparing the disease with scarlet fever, spread rapidly and appear to have element of periodicity. This has been well exemplified here in Boston, the crowded districts at the North End, where in certain years large mbers of children are affected, and where in the succeeding years the ease appears only sporadically. Measles can occur three or four times in e same individual: this recurrence was one of the peculiar features of the idemic in Boston in 1880. It may attack young infants, but is rare der six months. After the sixth month, and especially during the first ar, the susceptibility to the disease is increased, and we meet with the atest number of cases between the first and the fifth year. The suscep- ility to measles appears to lessen as puberty is approached. It is some- at rare in adult life, though the fact of its attacking large numbers of lts was also a peculiarity of the epidemic of 1880 in Boston.

Measles is an extremely infectious disease, the contagium apparently sing from one individual to another after a very short exposure, and often hout any direct contact, as by transmission through clothing or by the ds. It is most infectious in the beginning of the attack, and the infec- may be transmitted three or four days before the efflorescence appears the skin. There seems to be much less liability for the transmission the disease during the stage of desquamation than is the case in some the other exanthemata, such as scarlet fever and variola, the means of transmission corresponding more to that of varicella. In speaking of scarlet fever I have already referred to the case (Case 234, page 533) of the little girl who, although exposed to the scarlet fever infection in the beginning of the disease, did not contract it, but in the following year, when exposed for a shorter time to the contagium of measles, was immediately infected by that disease. The following cases which I shall report to you will be interesting and instructive as examples of how the transmission of the contagium of scarlet fever can be prevented by treatment, and will also illustrate the high degree of the infection in the early stages of measles.

A boy (Case 247) who was in my ward at the Children's Hospital was attacked with scarlet fever.

I had him removed to the contagious ward and placed under the care of a special nurse, who had orders to carry out the most precise antiseptic treatment. The directions to the nurse were that she should apply an ointment to the child, rubbing it into the skin thoroughly from the head to the feet twice daily. The child was also to be bathed twice daily with a solution of corrosive sublimate, 1 to 10,000. The nurse was cautioned not to allow her clothes to touch the boy's bed.

During the early stage of this boy's desquamation a second boy (Case 248), who occupied the bed in the general ward next to the bed from which the first boy had been removed, was attacked with sore throat, vomiting, and fever. I had already paid my visit for the day, and my house officer, thinking the case was probably one of scarlet fever contracted from having been in such close proximity to the bed from which the first boy was taken, had the second boy removed to the contagious ward and placed in the same room with the first boy. On the following morning I found that the second boy did not have scarlet fever, but had measles. I immediately had the second boy removed to another room, and he was carefully watched for a week, supposing that having passed the night with the first boy, who was in the most infectious stage of scarlet fever, he might have contracted scarlet fever. A week passed, and he evidently had escaped infection by the scarlet fever contagium.

Ten days later the boy who had scarlet fever was attacked with measles, presumably contracted during the night from the boy who was his room-mate in the early stage of his attack of measles.

These two cases apparently show—first, that scarlet fever, even during its most infectious stage, can be prevented from spreading by thorough and constant disinfection; second, that measles is highly contagious in its early stages.

PATHOLOGY.—Beyond the morbid conditions which appear on the skin and on the mucous membrane of the *throat*, there is no especially characteristic pathology of measles.

Neumann has studied the pathology of the skin in measles by means of specimens which were hardened in a dilute solution of chromic acid and colored with carmine, hæmatoxylin, and picro-carmine. The pathological changes were found to be almost entirely confined to the glands of the skin and to the blood-vessels. About the walls of the blood-vessels, principally in the upper layers of the cutis, were found collections of round cells which in crowded masses surrounded the loops of the blood-vessels even in the papillæ. The blood-vessels themselves were dilated and full of blood. The coils of the sweat-glands, as well as the excretory ducts, were enveloped in accumulations of round cells, while the neighboring tissues were filled h these cells. These collections of cells were always situated outside the walls of the glands. The sebaceous glands presented like changes. e hair-follicles showed rounded protuberances which corresponded to the nts of insertion of the erectores pilorum, and which were probably caused contraction of these muscles. In the muscles themselves there were to found, between the cells proper of the muscular tissue, scattered round ls, which showed the participation of the muscular tissue in the inflamma-y process. The hair-follicles, in the same manner as the sweat-glands, re seen to be surrounded in their entire length by collections of round ls, which were more numerous in the lower than in the upper part of the n. We therefore see that in measles the pathological process in the skin cts chiefly the blood-vessels and glands, while the tissue proper of the n, as well as of the epithelium, presents no marked changes.

From the fact that in measles the pathological processes of the disease situated more particularly around the blood-vessels and cutaneous glands, nay be assumed that the infectious material of the malady, whatever its ure, is eliminated from the system through these channels.

In addition to the pathological lesions which occur in the uncompli-ed cases of measles, there is almost always associated with the catarrhal dition of the mucous membrane of the upper air-passages a catarrh of larger bronchi. One of the most common complications of measles is umonia; this is usually a broncho-pneumonia, lobar pneumonia being nparatively rare.

In some cases an inflammation of the smaller bronchi accompanied by monary collapse occurs. The bronchial glands are apt to be swollen if secondary infection is a severe one. According to Osler, a swelling of er's glands is not uncommon, and may be accompanied by a hyperæmic dition of the mucous membrane of the gastro-enteric tract.

Although a secondary infection of the ear has been considered rather tinctive of scarlet fever, this complication has in my experience arisen quite frequently in measles. When the ear is affected in measles there congestion of the middle ear. When the onset of the preliminary gestion occurs in connection with the inflammation of the nasal and o-pharyngeal mucous membrane, it consists of a simple, general, acute gestion of the middle ear, accompanied in the beginning with serous dation, and later with a rapid thickening of the membrana tympani in nection with the inception of the suppurative process. When, on the er hand, the preliminary congestion is coincident with or follows the orescence on the face, the congestion is primarily in the upper portions of membrana tympani as the result of a suspension of vaso-motor inhibi-n. Under these conditions there is a congestion of the manubrial plexus, the superior and posterior portions of the membrana tympani, and of the rresponding portions of the inner end of the external auditory canals.

In addition to this more common condition, a general congestion of the embrana tympani is found during the stage of efflorescence, and is likely to be more severe in its type than that which occurs during the prodromal stage of measles.

The inflammation of the middle ear accompanying measles is more likely to leave behind such trophic changes as thickening of the tympanic mucous membrane with the formation of adhesions than is scarlet fever.

During an attack of measles, and subsequent to it, the tissues show an especial vulnerability to infection by the bacillus of tubercle. The tubercular infection may be represented by the lesions of a general miliary tuberculosis or by those of especial tissues, such as of the cervical and bronchial glands, the joints, the ear, and, most commonly of all, the lung. In the latter instance the lesions are usually those of a tuberculous broncho-pneumonia.

INCUBATION.—The time of the incubation of measles may vary very much, and may cover a period of two or three weeks; the usual time, however, is ten days.

SYMPTOMS.—**Prodromata.**—The prodromal stage varies in length, but, reckoning ten days as the usual time for the stage of incubation, the prodromal stage may be considered to last from two to three days, and in some cases four days. In this stage we have in typical cases of the disease symptoms distinctive of measles. The invasion is characterized by severe catarrhal conditions affecting the nose (coryza), the eye (lachrymation), and the throat and upper air-passages (cough). In the first twenty-four hours the temperature rises to 38° or 39° C. (100.4° or 102.2° F.), and often to 40° C. (104° F.). The height of the temperature on the first evening is a fair indication as to the severity of the coming disease. Thus, a temperature of 40.5° C. (105° F.) indicates a severe case. An important point to be noticed regarding the prodromal symptoms is that after the first twenty-four hours there is in a large number of cases a remission in the temperature, which goes down, perhaps, to 37.5° or 37° C. (99.5° or 98.6° F.), and remains down for about twenty-four hours, when it again rises. The cough, coryza, and lachrymation, which appear early in the prodromal stage, do not abate, but rather increase, during this remission of the temperature. This is an important point to remember, as the child who seems quite sick and loses its appetite while the temperature is high during the invasion of the disease, seems brighter and has a return of appetite on the second day when the temperature is lower. This peculiarity of the prodromal stage is often misleading both to the parents and to the physician, who, because the child appears so much better, are led to believe that one of the infectious diseases is not developing. In infants and young children the prodromal stage may begin with a convulsion, but this is unusual, and if it occurs it is not, as a rule, particularly severe, and does not necessarily make the prognosis more grave. Headache in the prodromal stage is quite frequent; vomiting is rather rare. The tongue is usually furred, and the mucous membrane of the throat towards the end of the second day, and before the efflorescence has appeared on the skin, shows a condition which is very

that which is about to appear on the skin. These lesions, which
ially pronounced on the soft and the hard palate, are represented by
r macules of a dark-red and later purplish-red color, of different
l considerably larger than the punctate macules which I have
in speaking of the throat in scarlet fever. These papules may
s be found to have coalesced in some parts of the fauces. The
nembrane between the lesions is comparatively normal in color,
here may be a slight hyperæmia of the entire throat. This hyper-
wever, is not nearly so intense as is seen in the throat in scarlet
After the remission of the temperature, which I have already
as taking place on the second day, the temperature on the third or
y again rises.

ORESCENCE.—At the end of the third day or at the beginning
urth day—that is, the thirteenth or fourteenth day from the time
ection took place—an efflorescence appears on the skin. The efflo-
usually reaches its maximum in about thirty-six hours, this being
onstant number than the other figures which I have given you;
is about the fifteenth day from the date of infection. The stage
ation is rather more constant than the stages of prodrome and efflo-
the latter two varying as to their length, but together amounting
six days.

the efflorescence appears on the skin it consists commonly of small
or papules on a slightly reddened base, which first appear on the
the disease progresses, these lesions extend to the neck and chest,
e latter locality are, especially in the beginning, of a delicate pink
form of distribution in some cases being crescentic. The efflores-
n rapidly extends to the rest of the body and to the extremities.
ally more pronounced on the face, where the papules are apt to
and where an œdematous condition of the tissues, especially around
and nose, usually occurs. The eyes are swollen and partially
d the conjunctivæ are reddened. Photophobia at this time is pro-
The efflorescence may also appear on the scalp. The efflorescence
well marked for from one to two days, and while it is at its height
erature reaches its maximum, and remains high for two or three
responding to the intensity of the efflorescence. It then rapidly
reaches the normal point in about two days more,—that is, there
to be often a distinct crisis in the disease. During the period
scence, when the temperature is still raised and the efflorescence is
ximum, it is usual to have, in addition to the symptoms of cough,
nd lachrymation, a slight disturbance of the intestines, represented
, frequent, loose discharges, apparently arising from irritation of
m and descending colon. This condition is seldom a serious one,
special attention need be paid to it unless it should continue for
s, or after the maximum of the temperature and efflorescence has
sed for a day or two.

Desquamation.—The desquamation is usually furfuraceous in character,—that is, the epithelium is cast off in fine flakes, and is thus distinguished from the large lamellar flakes occurring during the period of desquamation in scarlet fever. The desquamation begins in the order in which the efflorescence came out,—namely, first on the face and later on the chest. The furfuraceous character of the desquamation is especially noticeable on the sides of the nose. The disease usually runs its entire course in three weeks.

Prognosis.—The prognosis of measles, as a rule, is good, but this depends almost entirely upon whether the disease is free from or accompanied by complications.

Diagnosis.—In order that you should understand how difficult it sometimes is to diagnosticate measles, you must recognize that it is one of the most variable diseases with which we have to deal. During epidemics of undoubted measles cases arise which differ materially from the disease as it appears in its typical form, yet these cases, by producing the typical form in other individuals, prove that they are all caused by the same contagium. In like manner certain epidemics may be characterized by irregular forms of the disease, and, as true measles can occur a number of times in the same individual, the recognition of a sporadic case is often impossible. As in other diseases of the skin, we should recognize measles not by any particular dermal lesion, but by the peculiarities of the prodromal symptoms, the general course and location of the efflorescence, the time of the maximum of the efflorescence and temperature, and the character of the desquamation. Thus, a prodromal stage of three or four days, characterized by catarrhal symptoms of the eyes, nose, and upper air-passages, and a papular efflorescence appearing first on the face, differentiate the disease at once from variola, varicella, and scarlet fever.

Treatment.—The treatment of measles is essentially symptomatic. There is no known means of producing immunity from the disease or of shortening its course. It is a self-limited disease, and the treatment should be directed to protecting the organs which are most likely to be attacked by complications. Bearing in mind that the eye, the nose, and the throat are affected in the prodromal stage, that later the skin is in a very sensitive condition, and that the lung is frequently the seat of some complication, we should direct our treatment especially to the protection of these organs.

The child should be placed in a room kept at an equable temperature, 20°–21.1° C. (68°–70° F.), and well ventilated. The room should be darkened, and the eyes should be protected from light during the whole course of the disease. Unless this precaution is taken, the eyes are often seriously affected for many months after the measles itself has disappeared. The child should be kept in bed until the temperature has been normal for a few days, the efflorescence has faded entirely, and the desquamation has almost ceased.

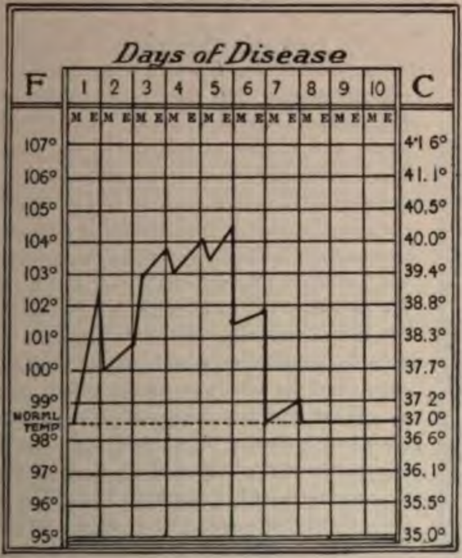
The diet during the period of the height of the temperature should be ilk, and bread. Later, when the temperature is normal and desqua- has begun, the child can gradually have its diet increased, until hird week from the beginning of the attack it is having its usual

cough, which is very troublesome at times, does not, as a rule, require ial treatment, as it will of itself in most cases pass off in a few While it continues it can be treated with some simple mixture, such horated tincture of opium in cold water in doses of 0.3–0.6 c.c. inims), to allay the irritation in the throat.

the irritation of the nose I have found that atomizing the nares me simple refined oil, such as oleum petrolatum album, is useful. the invasion of the disease, however, these catarrhal symptoms edingly difficult to control by any treatment whatever.

t times there is great irritation of the skin during the period of ence, this powder (Prescription 56, page 466) should be applied to the entire body and limbs. In place of the powder some simple t, such as petrolatum, may prove to be more soothing.

CHART 17.



Typical measles.

rule, the child should be kept in an equable temperature for at least eeks, and at the end of that time, if the desquamation has ceased, it allowed to go out of its room, and in pleasant weather out of the few days later. For several months, however, it should be carefully d from sudden changes of atmosphere, as the catarrh of the air- s is so likely to leave them in an extremely sensitive condition that slight irritation may cause its recurrence.

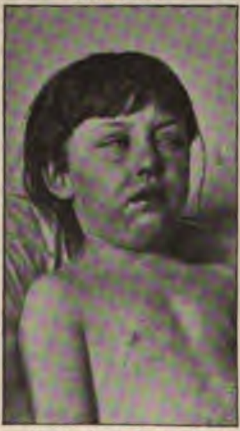
Before the child is allowed to leave its room it should be thoroughly bathed from head to foot in hot water. Although the contagium of measles has not the same tenacity for clothing as the contagia of variola and scarlet fever, yet the room should be thoroughly disinfected after the child has left it. This can be done in the same way that I have described to you in speaking of scarlet fever; but the extreme precautions taken in the latter disease are not considered necessary for the prevention of the extension of measles. If the carpet had not been removed when the child was put into the room, it can be taken from the house and thoroughly cleansed before it is brought back. The bedclothes and everything that can be washed should be thoroughly boiled. The room should be cleansed and the windows should be allowed to remain open for several days, as fresh air is one of the best means of eradicating the micro-organisms connected with the exanthemata.

This chart (Chart 17, page 579) shows the temperature as it usually occurs in the typical and regular form of measles.

Before speaking any further of measles I will show you here in the isolating ward at the Children's Hospital a case which illustrates so fully a typical picture of the regular form of measles that it will be very instructive for you to examine it.

This little girl (Case 249), six years old, after exposure to measles fourteen days ago, was attacked with lachrymation, coryza, cough, and a temperature of 39.4° C. (103° F.). On the second day from the beginning of the invasion the temperature fell to 37.7° C. (100° F.), but yesterday it rose again, and to-day, as you see by the chart, is 40° C. (104° F.). Later yesterday afternoon an efflorescence, papular in character, appeared on the face, and, as you see, has now extended to the neck and thorax. The disease is now at its height. You see the swollen condition of the eyes, nose, and entire face; also the extreme photophobia from which the child is suffering, the presence of considerable lachrymation, a continual, short, dry cough, and the extensive coryza. You will also observe how the papules have coalesced on the face, and are of a darker color than the widely separated lesions on the chest.

CASE 249.



Typical condition of the face in measles. Female, 6 years old.

When you have once seen a case of this kind you will never have any difficulty in making your diagnosis in a typical case of measles at the height of the stage of efflorescence.

In this next bed is a boy (Plate VII., Case 250, facing page 551), eight years old, who is at the height of the efflorescence of an attack of measles.

He was seized with the usual prodromal symptoms of cough, coryza, and lachrymation five days ago, and to-day has the different stages of the typical lesions of measles represented on his face and chest. You will notice how the conjunctivæ are reddened, and how the eyes, nose, and lips are swollen, although this swelling is not so intense as in the case of the little girl (Case 249) whom I have just shown you. The efflorescence in this case has run a very rapid course, beginning on the face in so intense a form that the desquamation has already appeared, although the efflorescence on the chest is in a much earlier stage of development. The papules and macules have, as you see, coalesced on the cheeks and chin, while they still appear as large, deeply reddened lesions on the forehead. On the chin and neck you will notice the areas of normal skin appearing like white blotches, their boundaries ermined by the clusters of papules. On the side of the nose you see a slight desquama- n, which has the furfuraceous character that I have already described to you as typical measles. You will notice that on the chest the papules and macules are much smaller ize, are of a much lighter color, and in some places have assumed a crescentic shape.

This case represents the typical efflorescence of measles, and up to this time has not wn evidence of any complication. Both this boy and the girl (Case 249) have received drugs directly for the measles, but have been kept in a dark room to protect the eyes, l have been surrounded by an equable temperature. Their food has been milk, broth, l bread.

In this next bed is a boy (Case 251), three and a half years old, who is convalescent m an attack of measles. He was exposed to measles on the 2d of the month, and l his first prodromal symptoms on the 12th. These prodromal symptoms continued on 12th, 13th, 14th, and 15th, making the prodromal stage four days. On the 16th a pular efflorescence appeared on his face, and desquamation began on the 21st of the nth.

I merely show him to you as representing the usual time, ten days, in the incubation measles, the rather prolonged prodromal period of four days, the appearance of the lorescence on the face about the fifteenth day from the time of infection and lasting four ys, and the desquamation beginning five days from the first appearance of the efflo- scence.

VARIATIONS IN TYPE.—I have already referred to the important fact garding the diagnosis of measles, that during epidemics and in sporadic ses the disease varies much in its type, and presents great variations in its rodromal stage, in its dermal lesions, in its desquamation, and in its entire ourse. I wish especially to impress this upon you, as it is through a lack f appreciation of this fact that the diagnosis of other diseases, such as ubella and various forms of erythema, is continually being made where, in act, the disease represents one of the more unusual forms of measles. If hese variations in measles were better understood, we should not find the isease rubella so often diagnosticated.

At times the stage of incubation of measles varies considerably. It may ven be extended from the usual ten days to twenty-one days.

Instead of the usual prodromal stage, certain cases during epidemics of ndoubted measles show few, if any, prodromal symptoms.

In this next bed is a boy (Case 252), seven and one-quarter years old, who was attacked ith the prodromal symptoms of measles on the 9th of the month. These symptoms were heightened temperature of about 38.8° C. (102° F.), a quickened pulse, cough, and coryza. n the 10th, 11th, and 12th the child felt perfectly well, had a good appetite and an almost ormal temperature. On the following day, the 13th, he was found to have the papular florescence of measles on his face, and a temperature of 38° C. (100.5° F.) in the morning nd 38.8° C. (102° F.) in the evening. It has been a very mild case, and, as you see, is ow desquamating slightly.

I show him to you as representing one of the many variations which arise in measles, the variation in this case consisting in the child being perfectly well during the last three days of the prodromal stage, and thus showing prodromal symptoms only during the first twenty-four hours of the invasion.

In addition to the usual catarrhal symptoms which I have described, in some cases there are vomiting and sore throat. Again, instead of a considerable elevation of the temperature, it may be scarcely above the normal degree. In addition to the other variations in the course of the prodromal stage of measles, cases have been noticed during epidemics of this disease where the catarrhal symptoms were absent. Epistaxis of a mild form, and not apparently connected with the more severe types of hemorrhage, is sometimes met with. I have seen it only occasionally.

The efflorescence, which in the typical cases usually consists of papules, or vesicles and papules, may vary so as to simulate closely a common erythema, constituting the form called *lævis*, or may closely simulate a papular erythema. Again, the efflorescence may in certain cases be represented by minute vesicles or milia, characterizing the form called *miliarius*. Any of these forms may be confluent, but not usually anywhere except on the face. There is another form of efflorescence which occurs in measles, is rare, and is of a more serious nature than the common benign forms which you will meet with ordinarily. This is called the hemorrhagic or malignant form, and is represented on the skin by small capillary hemorrhages. It is often rapidly fatal, and at times appears to be part of a general hemorrhagic diathesis represented by epistaxis, hæmaturia, and hemorrhages from other localities. The temperature in this form is not typical, as it does not remit in the prodromal stage, thus depriving us of an important means of diagnosis; but a doubt as to the nature of the disease does not last long, as the other symptoms soon become prominent. The more prolonged the course of this form the better the prognosis, for if fatal it is usually quickly so. It may be complicated by a malignant broncho-pneumonia.

The efflorescence, besides differing in its form, may vary to a great degree in its intensity. Thus, we may have every grade of papule or macule, from the smallest to the largest, and varying from a dark purplish to a light pink color. In like manner, although the arrangement of the efflorescence, especially on the chest, is somewhat crescentic, yet during epidemics of undoubted measles this crescentic shape is often absent. Instead of the efflorescence first appearing on the face and then extending to the thorax and extremities, we may find in undoubted measles that it begins first on the chest or some other part of the body; or the efflorescence may appear on the face and thorax simultaneously. We may also find that in certain cases the efflorescence appears first on the abdomen, or on the thighs, and yet the presence of other typical and undoubted cases of measles in the vicinity or in the same house assures us that we are dealing with the same disease. The efflorescence instead of lasting for a number of days may be evanescent and may subside within twenty-four hours. The entire absence of efflorescence is said to occur in some cases, but must be considered very rare, and its possibility has been doubted.

The desquamation of measles is of so light a grade that it is not surprising that in some cases no desquamation whatever is detected. Cases where desquamation occurs without efflorescence are highly improbable, although such have been reported.

During certain epidemics of undoubted measles cases have not infrently been noted where the post-aural and cervical glands were en- ed.

There is a form of measles, called the recurrent, which is closely allied elapsing fever. The main characteristic of this form is the high fever. temperature will sometimes be raised for five or six days, will then me normal for seven or eight days, and will then rise again with a re- ence of the symptoms. This is a very unusual form, and one which ls merely to be mentioned here. It is accompanied by the general ptoms connected with the nose, eye, and bronchi which are met with he typical form of measles.

Relapses have been reported to occur in measles, but they must be very ommon. I have never met with such cases.

In reviewing the pictures which I have endeavored to give you of these iations, it must be evident to you that, although in the large proportion cases measles runs so typical a course that the diagnosis is very easily le, yet such great variations in type are always liable to occur that should be extremely careful not to make a diagnosis of certain other eases, such as rubella, except under unusual circumstances. This is portant, because we know that during epidemics of well-marked measles these great variations as to incubation, prodrome, efflorescence, desqua- tion, and the entire course not infrequently arise.

A case which occurred in my wards at the City Hospital during an demic of measles which took place in that institution illustrates how atly the symptoms and appearance of the disease may vary. The cases urring in the hospital were almost without exception of the typical form, which no mistake could be made as to the diagnosis of measles.

A girl (Case 253) who was in the hospital, and who was exposed to infection from patients with measles, after feeling perfectly well on the previous day, was found in the ning to have slight coryza, cough, and a papular efflorescence not confluent even on face, small in size, light pink in color, and not crescentic. While the efflorescence d the appetite was somewhat lessened, and the temperature was about 37.5° C. (99.5° At the end of twenty-four hours the efflorescence had almost faded, and in a few days general symptoms passed away, the patient's appetite had returned, the temperature become normal, and she seemed perfectly well.

If this case had been met with as a sporadic one it would have been impossible to e the diagnosis of measles, and from its mild nature it would have been supposed to ome slight form of disease, such as rubella.

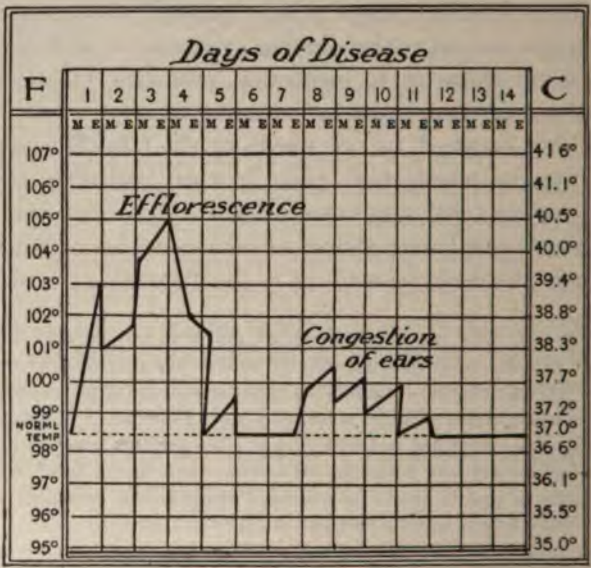
I have met with cases of this type quite frequently, both during epi- nics and sporadically; their cause is always obscure, and in them the gnosis between measles, rubella, and papular erythema is often impossible.

In this next bed is a little girl (Case 254) who is convalescing from measles and is htly desquamating. The record states that she was attacked with cough, coryza, hrymation, a temperature of 39.4° C. (103° F.), a pulse of 120, and respirations slightly ckened. On the second day of the attack these symptoms abated somewhat, and the perature fell to 38.3° C. (101° F.). On the following day the temperature rose to ° C. (103.5° F.) in the morning, and in the evening to 40.5° C. (105° F.). At this time an efflorescence, papular in character, appeared on the face, and by the fourth day had spread rapidly to the body and limbs. On this day the temperature fell to 38.8° C. (102° F.) towards the middle of the day, and by evening to 38.6° C. (101.5° F.). On the morning of the fifth day the temperature was 37° C. (98.5° F.) in the morning and 37.5° C. (99.5° F.) in the evening. The temperature was then normal for two days, but on the eighth day the child was found to have a temperature of 37.7° C. (100° F.), and to be complaining of pain in the left ear. A marked congestion of both membranæ tympani with a slight serous effusion was all that was detected. The temperature, after fluctuating from 36.6° to 37.7° C. (98° to 100° F.) for two or three days, fell to the normal, and the congestion of the ears subsided. During the time when the ears were affected the eyes were very sensitive to light, and there was considerable conjunctivitis, of which the child complained greatly. The cough was also very troublesome, and was evidently caused by an irritation of the mucous membrane of the throat, as at no time was any bronchial irritation detected.

It is to be noticed in this case that the congestion of the membranæ tympani occurred, as I have already described, during a period closely following the efflorescence on the face.

Here is the chart (Chart 18) which represents the temperature during the stage of invasion and efflorescence in this case, and also the accompanying mild congestion of the membranæ tympani which is so common in measles.

CHART 18.



Measles with congestion of membranæ tympani during stage of efflorescence.

COMPLICATIONS AND SEQUELÆ.—There are quite a number of complications and sequelæ which may occur in the course of measles. The most common of the serious ones are pertussis, pneumonia, and tuberculosis.

The first-named disease seems to have an intimate connection with measles, and its occurrence in the course of measles renders the prognosis more grave.

The bronchitis which is so common an accompaniment of measles sometimes appears in a more severe form, attacking the smaller bronchi as well as those of medium size, and may result in a broncho-pneumonia, which is much more common as a complication of measles than is lobar pneumonia.

The broncho-pneumonia does not, however, appear to be more severe when it arises as a complication of measles than when it occurs separately from that disease. Broncho-pneumonia as a complication of measles may occur very early in the course of the disease, even during the stage of invasion; but it occurs most commonly towards the end of the second week.

When, therefore, after the efflorescence has faded and the fever has subsided, the temperature again rises without evidence of local irritation in the throat, ear, or glands, we should suspect that a broncho-pneumonia is developing.

The additional symptoms of quickened respiration and the movement of the alæ nasi will render still more probable the supposition that this complication is arising, even though nothing abnormal is detected in the lung itself. This absence of abnormal physical signs in the lung in the early stage of broncho-pneumonia is quite common, and I shall defer a further description of them, as well as of the disease itself, until a later lecture (Lecture XLIX., page 962). In infants the temperature of tuberculous broncho-pneumonia does not seem to differ very much from that of ordinary non-tuberculous broncho-pneumonia.

The congestion of the larger bronchi, which appears to be almost a part of measles, may become subacute and chronic, instead of, as is usually the case, passing off soon after the maximum of the temperature and efflorescence.

Pleurisy may occur in the course of measles, but is not so common as pneumonia.

Among the rarer complications of measles are empyema, endocarditis, pericarditis, and membranous laryngitis.

Catarrhal laryngitis and tracheitis are not infrequent accompaniments of the acute stage of measles. Œdema of the glottis is rare, but has been known to occur.

When an otitis occurs as a complication of measles it is characterized by the symptoms which I have already described (page 575). In treating this complication the nose and naso-pharynx should be kept as clear as possible. The ear should be, as in the cases which I have already described in speaking of the treatment of the ear in scarlet fever, gently inflated by means of the Politzer bag, and the atropine solution (Prescription 70, page 559), together with dry warmth, should be used.

A case which came under my observation illustrates so well this aural complication occurring in measles that I shall report it to you.

A girl (Case 255), one year and seven months old, previously well, was attacked on March 6 with coryza, cough, lachrymation, a heightened temperature, quickened respirations, and a quick pulse. An efflorescence of measles appeared on the face on the following day, and the child felt sick, coughed continuously, and had a hoarse voice. The respirations varied from 36 to 40, the pulse from 170 to 180. The skin was hot and dry, and the throat was somewhat reddened. In the afternoon the temperature in the axilla was found to have risen to 40.2° C. (104.8° F.). She vomited and had a convulsion. The nervous symptoms passed off in a few hours, free perspiration followed, and the cough became somewhat looser. At 8 o'clock in the evening the temperature was 40.1° C. (104.4° F.), the respirations were quickened, and the pulse was rapid. Nothing abnormal was found on physical examination of the chest. During the night she was somewhat delirious, and very wakeful and fretful. The temperature remained at about 40° C. (104° F.), the respirations were rapid, and the alæ nasi moved so perceptibly that it seemed as though a pneumonia must be developing. Frequent and careful examinations of the lungs, however, failed to show anything abnormal. She continued to be very restless during the night, and the efflorescence appeared thickly on the abdomen and legs, but very slightly on the chest. She complained of pain in the chest from the continuous cough, but did not show any symptoms of pain or discomfort elsewhere. Towards morning it was found that an otitis of the left ear had developed, which in a few hours caused perforation of the membrana tympani. As soon as there was a free flow of pus from the ear the temperature fell to 38.3° C. (101° F.), the respirations became quiet and normal, the alæ nasi ceased to move, and the child fell into a quiet sleep. On the next day the efflorescence was pronounced all over the body, face, and extremities. From this time the measles ran its usual course, and was followed by desquamation and complete recovery.

The aural complication, however, proved to be very intractable, and, although it was carefully treated by Professor Blake, lasted for many months. The perforation of the membrana tympani did not completely heal for over a year, but the case finally resulted in complete recovery without any disturbance of hearing.

In addition to the conjunctivitis which is a common accompaniment of measles, and which, as a rule, requires no treatment beyond the protection of the eyes from light, the inflammatory process may extend to the deeper tissues of the eye and cause other grave lesions, such as blennorrhagic conjunctivitis, keratitis, and iritis. These complications should be treated at once by a skilled oculist.

Tobeitz has called attention to the deleterious influence of measles in rendering more active any subacute or chronic affections of the eye which may have existed previous to the disease.

In a number of cases an acute swelling of the thyroid gland may take place during the course of measles. This swelling of the thyroid gland may even cause marked dyspnœa by pressure, but it usually disappears in two or three days. In some cases, however, a formation of pus has taken place, followed by destruction of a part of the gland. In intractable cases of this kind it has been found that the external application of iodine is useful.

Enlarged cervical glands are not so common in measles as in scarlet fever, but they may occur, and may even prove serious from the occurrence of suppuration.

At times, at the height of the efflorescence, albumin may appear in the urine; but this is frequently merely a transient congestion of the kidney, due to the high temperature, and corresponding to the same condition in the period of efflorescence in scarlet fever. Nephritis may complicate measles, as it does scarlet fever, but it is comparatively rare.

The irritation of the intestine, which I have already referred to as occurring commonly during the height of the efflorescence and temperature, sometimes becomes much more severe from the development of colitis as a complication.

The most common sequela of measles is tuberculosis. This may occur her as a general miliary tuberculosis or as tuberculous disease of any the organs or the joints. Tuberculous disease of the joints seems to ow a special predisposition to follow attacks of measles. It is noticeable at where a patient with a tuberculous joint has an attack of measles e process in the joint is apt to become temporarily more active, and the ognosis is consequently more grave. The organ which in measles is st commonly affected by tuberculosis is the lung, and the most common rm of tuberculosis of the lung is a tuberculous broncho-pneumonia. You ust remember, however, that a tuberculosis of the lung may often occur as sequela of measles where pneumonia has not been present. In infants the mperature of tuberculosis, as has been observed by Holt, does not seem differ very much from that of an ordinary broncho-pneumonia. In gard to the relation which exists between measles and tuberculosis, we ould appreciate the danger, which seems to be a serious one, that the micro-ganism of measles will render active an old and quiescent tubercular dus, whether it be in the bronchial or the cervical glands or elsewhere.

I have here a case to show you which represents the infection of a patient ith measles by the bacillus tuberculosis.

This girl (Case 256), six years old, was always well until about one year ago, when she d an attack of measles. Although there was no acute pulmonary affection following the ack of measles, she began to be affected with slight dyspnœa and a cough about one nth after the measles had ended. Since then these symptoms have increased, and she s lately had swelling of the feet and has complained of a general malaise. She has o lost considerably in weight and strength. On physical examination dulness is found the apices of both lungs, and over the dull areas coarse and fine moist râles. Nothing normal is found in connection with the heart or kidneys. The temperature varies from 7° to 38.8° C. (100° to 102° F.), the respirations from 30 to 50, and the pulse from 120 130. An examination of the sputum shows the bacillus-tubercle to be present. This is dently a case of pulmonary tuberculosis following an attack of measles.

Another sequela, though a rare one, is paralysis. Cases thus complicated ve shown mostly a paraplegia, and, according to Osler, frequently can be ssified as post-febrile polyneuritis, although it is possible that some of em may be due to a rapidly ascending myelitis.

A very rare sequela of measles is the disease noma (cancrum oris). I ve here in one of the isolating rooms a case which illustrates the sequence measles of broncho-pneumonia and noma.

This child, a girl (Case 257), four years old, had a severe attack of whooping-cough. hen the whooping-cough had lasted six weeks, she was attacked with measles. Towards end of the second week of the measles the child was attacked with a broncho-pneumonia. is pneumonia was not of an unusually severe type, but it lasted for five or six weeks and ft the child in a very weakened and debilitated condition. During the pneumonia the child as not well cared for, and this complication arose, for which she has entered the hospital.

As I shall refer to this case later (page 793), when speaking of diseases of the mouth, hall show it to you now merely as a case of noma which I am having actively treated, ut in which the prognosis is very unfavorable. When noma occurs as a complication of easles and pneumonia it is generally fatal.

RUBELLA (Rötheln).—It is now almost universally believed that there is, in addition to variola, varicella, scarlet fever, and measles, a highly infectious acute disease accompanied by an efflorescence on the skin which is distinct from these other members of the group of exanthemata. While we must recognize the propriety of mentioning the existence of this disease when speaking of this class of affections, we must also acknowledge that it is the weight of opinion, and not of proof, which has characterized rubella as a disease *sui generis*. The cause, the symptomatology, and the resulting diagnosis and treatment of rubella must be left for future investigation, until the special micro-organism which produces it and that which produces measles can be separated bacteriologically. The difficulty which arises in differentiating rubella from the other diseases of this class is chiefly in distinguishing it from measles. We cannot describe a typical case of rubella in such a way as to enable us to diagnosticate the disease in a sporadic case. On the other hand, this can be done so readily with the other exanthemata that we can at once diagnosticate a sporadic case of these diseases. Rubella is described in many ways by observers in different localities, but is usually spoken of as essentially a highly infectious disease, with an incubation of two or three weeks, with slight or no prodromata, and with a slightly raised temperature, accompanied by mild catarrhal symptoms, and often by sore throat and swelling of the cervical and post-auricular glands.

The efflorescence is commonly described as papular or macular in form, of light grade, often evanescent, and seldom showing any desquamation. Complications or sequelæ following rubella are said to be uncommon. If you will bear in mind what I have told you concerning the variations which occur commonly during epidemics of undoubted measles, you will see at once that this description of rubella is one which may be applied to many mild cases of measles. As, however, epidemics arise in which these characteristically mild symptoms occur in many cases, and as these give rise to like cases, it is probable that in the future a micro-organism distinctive of rubella may be found.

Bearing these facts in mind, we can merely say, regarding rubella, that its diagnosis cannot be made in a sporadic case, that the prognosis is good, and that the treatment is the same as that of a mild case of measles.

It may perhaps aid you to carry in your minds more clearly the characteristics of the group of exanthemata, which I have endeavored to explain to you, if in a few words I speak of this group of diseases as a whole.

In none of these diseases has the specific organism been determined. When it shall have been, its detection will enable us to state definitely which disease we have to deal with, whether measles or scarlet fever, and even in the atypical cases of measles we can decide whether we have a case of true measles or of some disease such as rubella, which closely simulates its irregular forms.

By referring to this table (Table 97) you can at a glance ascertain the chief points of differential diagnosis in the exanthemata. The figures and atements are merely approximate, but in this way the diagnosis of diseases is much simplified and their characteristics are made more nent.

TABLE 97.

	Variola.	Varicella.	Scarlet Fever.	Measles.	Rubella.
tion . .	12 days.	17 days.	4 days.	10 days.	21 days.
mata . .	3 days.	A few hours.	2 days.	3 days.	A few hours.
scence .	Macules. Papules. Vesicles. Pustules.	Vesicles.	Erythema.	Papules.	Papules.
mation .	Large crusts.	Small crusts.	Lamellar.	Furfuraceous.	. . . . . .
ications sequelæ.	Larynx. Lungs.	. . . . . .	Kidney. Ear. Heart.	Eye. Lung. Tuberculosis.	. . . . . .

lthough what I have shown you in this table is far from definite, and , were one of the diseases to be diagnosticated, be very misleading, yet fferential diagnosis between all these diseases I think you will find it ble because of its simplicity.

addition to the leading points which I have indicated in the table, neral symptoms and the temperature of these diseases provide us with nt material by which to distinguish one from the other.

e slow progressive development of variola is very distinct from the rapid course of all the others. The vomiting and sore throat of fever are usually quite distinct from the coryza, lachrymation, and of measles. In variola the rise of temperature during the prodromal its decided lessening at the time of the appearance of the efflores- and its gradual rise again during the stage of suppuration, are listinct from the sudden rise of temperature in scarlet fever during rodromal stage and up to the height of the efflorescence. In like r the temperature in measles differs from that of the other diseases sudden rise on the first day of the prodromal stage, in its lessening second day, and in its rise on the third day and up to the height efflorescence. The manner of the decline of the temperature differs iola, in scarlet fever, and in measles. While in variola it is slow rolonged, in scarlet fever it is rather rapid, although it declines by and in measles the fall is often by crisis. In contradistinction to a, scarlet fever, and measles, varicella and rubella differ markedly absence of a prodromal stage, in their short duration, and in their scent and moderate temperature.

DIVISION XI.

DISEASES OF THE NERVOUS SYSTEM, AND THE MYOPATHIES.

LECTURE XXVII.

INTRODUCTION.

TO-DAY, gentlemen, we shall begin to study a class of diseases which is the most difficult to understand of any that are met with in early life. This difficulty exists necessarily from the complex organism of the parts affected, and on account of the important *rôle* which the nervous system plays in all diseases which occur in human beings during the process of their development. We are much more likely to meet with nervous phenomena of the most diverse varieties in children than in adults. In like manner we meet with the most widely differing clinical symptoms. If you had studied the clinical symptoms of nervous diseases in the adult only, you would be insufficiently prepared to diagnosticate properly from similar symptoms in the case of the child. Symptoms which if occurring in adults would be significant of serious lesions of the nervous system may arise in children from simple reflex conditions which only simulate and do not represent actual disease.

Children are much more apt to become unconscious, to have convulsive attacks, and to show disturbance of the functions of important nervous centres from reflex irritation, than are adults. The whole cerebro-spinal system in infancy and early childhood is so impressionable, so excitable, and so hypersensitive to even slight grades of irritation, that diseases of a nervous type, whether primary or secondary, dominate all others.

We have, then, not only well-recognized pathological lesions with their characteristic symptoms, as in adults, but also the same groups of symptoms caused by different pathological conditions, and, again, reflex nervous phenomena without organic lesions, *ad infinitum*.

These reflex phenomena are so much more numerous than those which arise from organic lesions, and are so irregular in their manifestations, that, from a diagnostic point of view, they are most important. They also enter all disturbances of the nervous system, whether functional or organic, ich a degree that what we have learned concerning cerebral localization he adult becomes of much less value in the young subject. Attempts ocate minutely diseases of the nervous system by means of cerebral lization are so indefinite, and in the hands of the general practitioner ruitful of incorrect conclusions, that I have thought it better to pay little attention to this branch of neurology, which for the present ld be referred to the nervous specialist.

Difficult as the study and clinical recognition of these manifold condi- s are, far greater becomes our task when we attempt to classify and nge in simple form for the purpose of teaching the complex nervous nomena which we meet with in our nursery practice. Diseases of the ous system constitute in themselves the study of a lifetime, and we are busily engaged in general medicine cannot hope to obtain the exact iled knowledge of the nervous specialist. The nervous specialist, on contrary, who has not worked practically among children, studying n in all their various phases of excitement and rest, disease and health, fail to grasp the special phase of nervous disease by which he is at s confronted.

The various pictures of nervous diseases which I shall present to you those which you will be most likely to meet with in practice. As it is roscopic rather than microscopic knowledge which is most needed for ical observations in childhood, I shall treat the subject broadly, leaving finer touches for your later and more extended study of the works of led neurologists.

We must adopt some division for teaching which by its simplicity will us to keep in mind the various diseases in a connected series. As the d grasps more readily symptoms produced by a distinct pathological n than those where such a lesion has not been proved to exist, I shall k first of the principal organic lesions of the brain and cord, reserving later lectures what I have to say about the various nervous phenomena h from our indefinite knowledge concerning them we term *functional.* ve adopted this division simply for the purpose of clearness in teaching. not that of any especial authority on nervous diseases, but it is what ve found to satisfy practically the needs of the many classes of students m I have met from year to year.

The terms anæmia and hyperæmia of the vessels of the brain, as desig- ng distinct diseases, have been used frequently in connection with the ussion of diseases of the brain and cord. These terms should in the ent state of our knowledge be restricted to represent symptoms, and diseases, for in the majority of cases they are only symptoms which are ndary to some primary disease.

Nervous diseases can as a whole be divided, as may be seen in this le (Table 98, page 592), into—I. *Organic;* II. *Presumably Organic;* . *Functional.*

TABLE 98.

I. ORGANIC	Where there is a distinctly definite pathological condition.
Examples	*Meningitis. Hydrocephalus.*
II. PRESUMABLY ORGANIC .	Where there is no definite lesion, so far as we can at present determine, but where we suppose that a pathological condition may in the future be discovered, and that the disease may then be relegated to the organic class.
Examples	*Chorea. Epilepsy.*
III. FUNCTIONAL.	
1. **Probably Central** .	Where apparently the symptoms arise from a disturbance rather than a lesion of the nervous centres.
Examples	*Hysteria. Temporary aphasia from fright.*
2. **Reflex**	Where the symptoms are caused by peripheral irritation of various parts of the nervous system.
Examples	*Convulsions from foreign bodies in the stomach. Asthma dyspepticum.*

Under each of these divisions I have tabulated the various diseases which belong to it, and I shall speak in detail only of those which you will be likely to meet with in general practice.

By referring to this second table (Table 99) you will see at a glance which diseases I am about to describe to you, and the order which I have followed in describing them.

I would also call attention to the fact that this table is not arranged on either a purely pathological or a purely symptomatic basis. On the contrary, wherever it seemed expedient to designate a disease by the name of its principal symptom I have done so, although in most cases I have used a pathological term. The table, then, does not represent a recognized scientific classification of nervous diseases, but is merely a list of the different diseases in the order in which I shall speak of them.

TABLE 99.

Nervous Diseases.

I. *Organic.*	II. *Presumably Organic.*	III. *Functional.*	
		Probably Central.	Reflex.
Non-tubercular meningitis.	Chorea.	Hysteria.	Pavor nocturnus (peripheral).
Tubercular meningitis.	Epilepsy.	Hypnotism.	Dental reflex.
Thrombosis of the cerebral sinuses.	Insanity.	Catalepsy.	Reflex nystagmus.
Hydrocephalus.		Simulated diseases.	Reflex of ear.
Cerebral abscess.		Insolation.	Reflex of larynx.
Cerebral paralysis.		Concussion.	Paroxysmal gasping.
Athetosis.		Temporary amnesia.	Reflex of lung.
		Temporary aphasia.	Reflex cough.
		Arrested psychical development.	Reflex of heart.

TABLE 99.—*Continued.*

Nervous Diseases.

nic.	II. *Presumably Organic.*	III. *Functional.*	
		Probably Central.	Reflex.
tumors.		Retarded speech.	Reflex of stomach.
syphilis.		Headaches.	Reflex of bladder.
		Vertigo.	Reflex of vagina.
g.		Sensitive spine.	Reflex of rectum.
		Tetany.	
anterior.		Pavor nocturnus (central).	
m caries			
e.			
taxia.			
taxia.			
ia.			
rebro-spi-			
is.			
al menin-			
ritis.			
the new-			

impress upon you the fact that the classification which we may to adopt to-day will in all probability in the next five or ten years modified by the further study of nervous pathology. You will ever, that in my classification I have allowed for this progress in ought, and that the various diseases which I have tabulated in the ' Presumably Organic" and "Functional" can be placed in the Organic Diseases" as soon as it has been proved that they belong

dying the various diseases of the nervous system which I shall you, and the cases which illustrate them, I have received so from the special knowledge of these diseases possessed by Dr. . Bullard that I wish to acknowledge my indebtedness to him.

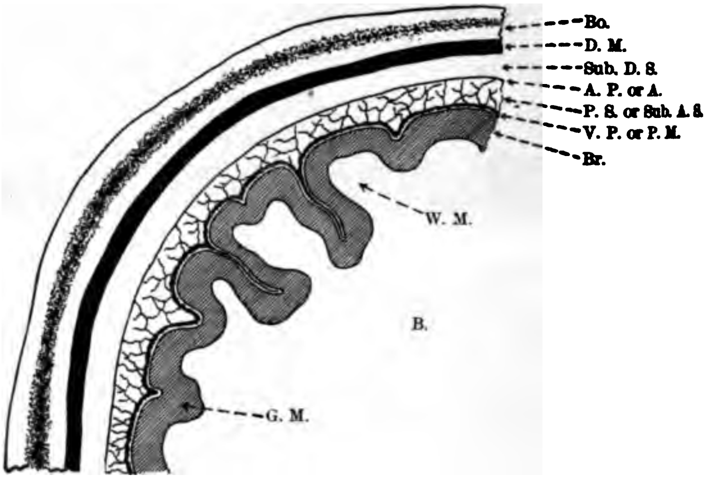
LECTURE XXVIII.

I. ORGANIC NERVOUS DISEASES.

BRAIN.—CORD.—BRAIN AND CORD.—PERIPHERAL NERVES.

BRAIN.—Before speaking of the diseases of the brain I should like to have you examine a section of the skull as shown in this diagram (Diagram 8). It represents the relations between the bone, the dura mater, the subdural space, the arachnoid, the subarachnoid space, the pia mater, and the brain. The diagram is useful for the clinical investigation of nervous diseases, and will, I think, aid you in understanding what I am about to describe.

DIAGRAM 8.



Section of skull and brain.

Bo.	Bone.
D. M.	Dura mater.
Sub. D. S.	Subdural space.
A. P. or A.	Arachno-pia or arachnoid.
P. S. or Sub. A. S.	Pial space or subarachnoid space.
V. P. or P. M.	Visceral pia or pia mater.
Br.	Brain.
W. M.	White matter.
B.	Brain.
G. M.	Gray matter.

The dura mater is closely attached to the skull at all ages, but especially so in childhood.

The subdural space lies between the dura mater and the arachnoid.

The subarachnoid space is crossed by fibres, thus making a connection between the arachnoid and the pia, which some anatomists are now inclined to speak of as one structure.

MENINGITIS.—If you will again glance at this table of classification able 99, page 592), and at the anatomical diagram (Diagram 8, page 594), u will see that I should naturally first speak of diseases of the cerebral ninges. Of these diseases *meningitis* is the most common. Cerebral ningitis may affect the *dura mater* or the *pia mater*. In the first case it called *pachymeningitis*, and in the second *leptomeningitis*. The form may *acute* or *chronic*.

Pachymeningitis.—Pachymeningitis is in early life so rare, except om certain local traumatisms, or as a lesion in some specific disease, such syphilis, that we need merely mention it, and can at once proceed to udy the inflammatory conditions of the pia mater.

Leptomeningitis.—Leptomeningitis, on the contrary, is very frequent childhood. It may be divided primarily as to its *locality* into (1) menin- tis of the convexities, and (2) meningitis of the base; as to its *pathology*, to (1) a simple non-tubercular inflammation of the pia, and (2) a growth miliary tubercle in the meshes of the pia producing inflammation.

This is only a general division, but it serves to prepare you for the mewhat more minute description which is necessary to make you under- and the varied clinical symptoms which are met with in these diseases, as e lines cannot be drawn sharply as to locality, pathology, or symptoms.

(1) NON-TUBERCULAR MENINGITIS.—Non-tubercular meningitis is ten called *purulent meningitis*, but the latter term does not seem so appli- ble as the former, because we also meet with a purulent exudation in certain ses of tubercular meningitis. The pathological process may, although in general way and to the greatest extent affecting the membranes of the con- exity, attack the membranes of the central and basic regions of the brain. ollowing, however, the rule that where we are making a clinical division diseases we should emphasize the salient lesions by which we can in most ses distinguish them, I shall leave the minute pathology of these diseases r your more extended pathological studies.

Non-tubercular meningitis in its acute form is a disease which may tack robust as well as debilitated children, and may occur at all ages. It rare in the first year of life. It is most common in the middle period of ildhood.

Clinically, we sometimes find the non-tubercular form in infants simu- ting in its symptoms, on account of the locality affected, the tubercular rm of the older child. On the other hand, the tubercular form in infants sometimes so acute in its symptoms as to simulate the non-tubercular rm of the older child.

Some rare cases of non-tubercular meningitis have been reported where e disease was apparently primary, and for the present, therefore, we can peak of this class of cases as idiopathic until further light has been thrown pon the subject. I myself have never seen a case of non-tubercular eningitis which was undoubtedly idiopathic. The great majority of cases s secondary.

Non-tubercular meningitis is most frequently of traumatic origin, as from some injury to the head, or the disease may arise from disease in the ear with its local inflammation extending through the petro-squamosal suture to the cerebral meninges. A comparatively small number of cases of non-tubercular meningitis appears to be caused by the specific microbe of such diseases as scarlet fever, measles, erysipelas, pneumonia, possibly typhoid fever, rheumatism, and syphilis. It also occurs in cerebro-spinal meningitis, and in rare cases it is secondary to a group of symptoms to which the name insolation has been given.

This table (Table 100) designates the different causes which may give rise to non-tubercular meningitis.

TABLE 100.

Non-tubercular Meningitis.

- *Primary* (said to exist).
- *Secondary.*
  - Traumatic.
  - Ear.
  - Specific diseases, such as
    - Scarlet fever,
    - Measles,
    - Erysipelas,
    - Pneumonia,
    - Typhoid fever,
    - Rheumatism,
    - Syphilis,
    - Cerebro-spinal meningitis,
    - Insolation.

PATHOLOGY.—The pathology of non-tubercular meningitis is practically, where infants and young children are concerned,˙ an inflammation of the pia mater. This, according to Delafield and Prudden, may be acute, chronic, tubercular, or syphilitic.

In any case of acute meningitis the inflammation is apt to extend downward and to involve the pia mater of the cord. In young children it especially happens that the inflammation may involve the ependyma of the ventricles and cause a distention of these cavities with serum.

In one form of acute non-tubercular meningitis the pia mater, according to Delafield and Prudden, from whose observations I shall freely quote, is somewhat congested. Its surface is dry, lustreless, and somewhat opaque. These changes in the gross appearances of the membrane are not marked, and may be overlooked, but the minute changes are more decided.

There is an abundant production of cells somewhat resembling the cells which coat the surfaces of the membranes and fibres which make up the pia mater. The cell growth is general, involving the pia mater over most of the surface of the brain. The inflammation is one which results in the production not of fibrin, serum, or pus, but of new connective-tissue cells. This form of meningitis, which may be called *acute cellular meningitis*, is of frequent occurrence, and is attended with the ordinary clinical symptoms of acute meningitis.

Another form of acute non-tubercular meningitis has been termed the *exudative*, because it is characterized by the accumulation, chiefly in the meshes of the pia mater and along the walls of the blood-vessels, of variable quantities of serum, fibrin, and pus. Sometimes one, sometimes another, of these exudations preponderates, giving rise to serous, fibrinous, or purulent forms of inflammation. The absolute quantity of the exudation varies greatly. In some cases death may be caused with so slight a formation of exudation that to the naked eye the pia mater may look quite normal or, perhaps, only moderately hyperæmic or œdematous. The microscope, however, in these cases will reveal pus-cells in small numbers, and sometimes flakes of fibrin in the meshes and along the walls of the vessels. In other cases turbid serum in the meshes of the membrane is all that can be seen, and the turbidity is shown to be due to pus-cells or to a small amount of fibrin. Again, either with or without marked œdema of the pia mater, yellowish stripes are seen along the sides of the veins, sometimes appearing like faint turbid streaks, and at other times dense, opaque, thick, and wide, so as almost to conceal the vessels. These are due to the accumulation of pus-cells and fibrin in large quantities along the vessels. They can be seen best and are most abundant around the larger veins which run along over the sulci. In still other cases the infiltration with pus and fibrin is so dense and thick and general that the brain-tissue, the convolutions, and most of he vessels of the pia mater are concealed by it. This is usually of a greenish-yellow color, and is sometimes so thick as to appear like a cast of he brain-surface at the seat of the lesion. Sometimes extravasated red blood-cells are mingled with the other exudations as the result of diapedesis. Microscopic examination shows numerous white blood-cells sticking in the walls of the veins and capillaries, or the vessels may be blocked with them. t is evident that a large part of the pus-cells accumulates as the result of migration. The connective-tissue cells of the pia mater may be detached from their places or degenerated. In some cases there are considerable ccumulations of pus between the pia mater and the brain-substance and long the vessels which enter the latter. More rarely, pus is found upon the ree surface of the membrane. The brain-substance may be compressed by he accumulated exudation so that the convolutions are flattened. The cortical portion of the brain may be simply infiltrated with serum (œdematous), r it may undergo degenerative changes and may be the seat of punctate hemorrhages. Not infrequently the inflammation extends to the ventricles, which may contain purulent serum, and to the pia mater of the cord. This form of infiltration is most frequent on the convexity of the brain, but may extend or even be confined to the base. It may be localized, but it frequently extends widely over the surfaces of the hemispheres. Bacteria are often present in the exudation, and I shall explain their relationship to the lesions when speaking of cerebro-spinal meningitis.

When recovery occurs from the *acute exudative form* of non-tubercu meningitis, there may be fatty degeneration of the cells which have accun lated in the pia mater, particularly along the vessels, and this may prod white patches in the membrane and threads along the blood-vessels, wh resemble the accumulation of exudation in the acute stage. Fatty deger ation of the blood-vessels and cells of the pia mater may also occur with acute inflammatory changes. Sometimes in children inflammatory char in the ventricles persist for days and weeks after the subsidence of the flammation of the pia mater.

The non-tubercular form of meningitis may also be chronic, in wh case the pia mater at the base of the brain alone may be inflamed (bas meningitis), or only the pia mater over the convexity, or the entire mater, or certain circumscribed patches of the membrane. In these cases pia mater is thick and opaque, and there is a formation of new connec tissue, with a production of pus, fibrin, and serum. The relative quan of these inflammatory products varies in different cases, and results in s cases in firm and at times extensive adhesions between the dura mater the pia mater. Other conditions which represent the results of chr inflammation may also be present, but need hardly be referred to h further than to say that the ventricles of the brain may in this chronic f contain an increased amount of serum and may be dilated. The ependy also may be thickened and roughened.

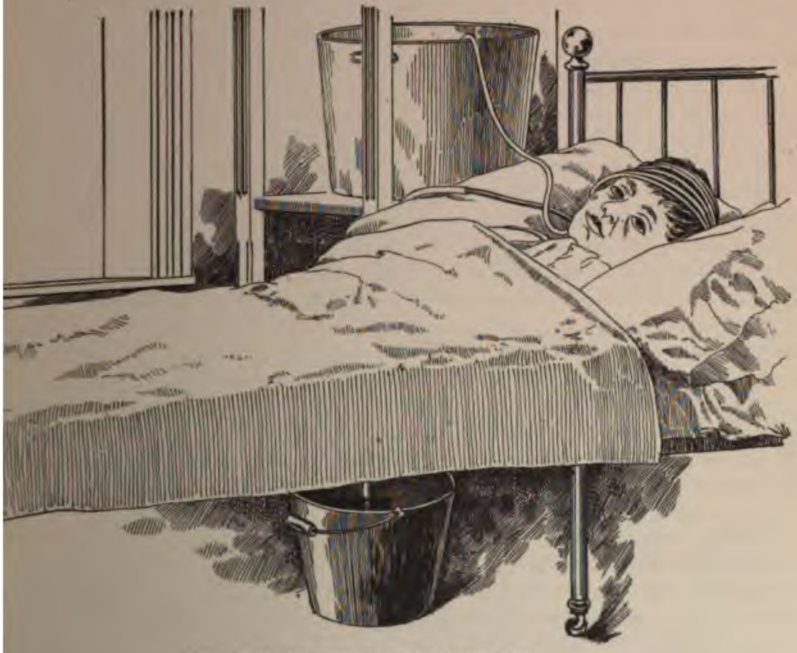
SYMPTOMS.—Where *non-tubercular meningitis* is secondary to inju or to other diseases, the characteristic symptoms may of course be com cated and even obscured by symptoms resulting from the especial cause. the supposed primary or idiopathic cases the symptoms, especially where child is over two years of age, are rapid in their development. The cou is short, from seven to eight days, and the disease may often prove f in forty-eight hours. The disease, when affecting the convexities chie begins with intense headache and a high temperature, 40°–40.6°–41.1° (104°–105°–106° F.). The respirations are rapid, 30–40–50, and compa tively regular. The pulse is quick, 150–160–170, but is usually regu Vomiting, photophobia, contracted pupils, and delirium are present. C vulsions occur early. Later we may have blindness and paralysis.

DIAGNOSIS.—As the diagnosis of non-tubercular meningitis is chiefl differential one from tubercular meningitis, I shall reserve what remains be said on this subject until I speak of the latter disease.

PROGNOSIS.—The prognosis is very unfavorable. It is possible, ho ever, for the child to recover completely from an attack of non-tubercu meningitis. Perhaps only a changed mental condition will remain, b appearing effeminate or more easily excited than would be conside normal. Some of the more acute forms affect also the brain, and we f their results in idiocy and contractures. We must always bear in mi that children have wonderful recuperative powers. Their nervous org isms, although sensitive to the least shock or the slightest irritation, fr ivity of their growth present opportunities for repair which do not n adults. So long as a disease of a necessarily fatal character is sent, the possibility of recovery should not be lost sight of. Vio- f the nervous manifestations does not by any means always indicate issue.

EATMENT.—The treatment of non-tubercular meningitis varies with the disease or condition to which it is secondary. The child should t in a cool, dark room and protected from noise. In the treatment of itis, whether it is a symptom or whether it is idiopathic, the indica- re to reduce the temperature of the body and to support the general h until the disease has run its course. The former is accomplished the application of mustard derivatives to the lower extremities, by ng the entire body every three or four hours with water at a temperature from 15.55°–22.22° C. (60°–70° F.), and by the application of cold head. The strength should be supported by the administration of nd, when necessary, of stimulants.

Case 258.



Treatment of meningitis with Leiter's coil.

e method of applying cold to the head by means of Leiter's coil is a le one, and I have here in this bed a child (Case 258) with meningitis being treated in this way.

e apparatus called Leiter's coil is very simple, and consists of a light metallic or preferably rubber tubing, which can be bent in any way and applied to any part of the body or limbs as well as to the head.

Two vessels are needed: one at a height somewhat above that of the child's head acts as a reservoir for the water, while the other stands under the bed to receive the water after it has passed through the tube. In this way we can have water at a constant temperature, warm or cold, continually running through the tube several times around the child's head.

In addition to the local treatment, bromide of soda in varying doses, according to the age of the child and the severity of the disease, can be given.

Dr. Fraser reports the case (Case 259) of a male infant, fourteen months old, unusually well developed and previously perfectly well. It began to be irritable and to lose in weight. These symptoms continued for about a month. When it was brought to him it had a temperature of 37.4° C. (99.5° F.), and it had no other symptoms beyond what would be expected from the condition of the gums, which were hot and tender. Three days later a convulsion occurred, and two days later hemiplegia of the left side. The pulse was 130, small and irregular. The temperature was 38.6° C. (101.5° F.). Sensation was perfect on both sides. On the following night the infant began to have convulsions, which continued with irregular intervals until the next morning. The entire voluntary muscular system was then found to be in a state of tonic spasm. The legs were rigid, the head was retracted on the trunk, and there was opisthotonos. This tonic spasm was interrupted at intervals of half an hour by a clonic seizure involving chiefly the extremities. While these nervous manifestations continued, the thumbs and the fingers were bent into the palms, and the forearms were flexed and extended upon the arms with short rhythmical movements.

The inferior extremities were similarly affected, though to a milder degree. The movements also extended to the face, giving rise to contortions. The respiration was irregular, but there was no lividity of the skin. The pulse was 140. The temperature was 38.8° C. (102° F.). Three days later, the previous symptoms having in the mean time continued, there was a diminution in the convulsions, but consciousness almost entirely disappeared, and there was an increasing tendency to coma. The pupils were contracted, there was an entire inability to swallow, and the infant gradually sank, dying at 6 P.M.

The post-mortem examination was made twenty-four hours after death. On opening the skull and deflecting the dura mater the convolutions appeared flattened, as if they had been slightly compressed. The veins of the cerebral cortex were much engorged. The outer surface of the visceral layer of the arachnoid was smooth and dry, but at a spot about 1.2 cm. (½ inch) in diameter, situated about the middle of the ascending frontal and parietal convolutions of the left hemisphere, the pia mater was covered by a thin, yellowish layer of lymph. During the removal of the brain several ounces of clear serous fluid escaped from the lateral ventricles. On section of the hemispheres the centrum semi-ovale did not present any unusual number of vascular points on either side, but the substance of both hemispheres, especially that of the left, was very soft.

The optic thalamus and lenticular nucleus of the left hemisphere were so much softened as to be almost diffluent. The ependyma of the lateral ventricles was soft and uneven, and it appeared in parts to be covered by a layer of lymph, but the surrounding tissues were so much softened that it was doubtful whether the layer consisted of lymph or of the smooth and softened ependyma. At the base of the brain a layer of lymph 0.3 cm. (⅛ inch) in thickness was found in the interpeduncular space underneath the visceral layer of the arachnoid. The inner surface of the dura mater at the base of the skull was smooth and without a trace of opacity.

There was in this case a softening of the brain-substance, which was probably secondary to the meningitis.

This boy (Case 260), four years old, whom I have here to show you, is apparently suffering from the results of non-tubercular meningitis. He was always well and strong until the onset of the present attack, which occurred twelve weeks ago. He never has had any disease, with the exception of measles when he was three years old.

ttack, in all probability, was produced by a fall, in which he struck the back
No cut or bruise was detected. Later, on the day of this fall, he began to com-
in his head and to vomit. He was very feverish, and lay in bed protecting
the light, as there was great photophobia. The bowels were regular, and he
ntities of food. A week later he became delirious, and this condition con-
weeks. He was then brought to the Children's Hospital, and from time to
ious during a period of five weeks. The delirium was sometimes active, and
disappear and he would recognize his parents. He was very cross in the in-
delirium, and would roll his head from side to side. His appetite was poor.
any convulsions or paralysis. For a time, however, he had incontinence of

attack he has been gradually growing better, and he is now comparatively
he sometimes complains of slight pain in his head, at which time the head
an at others. He also sometimes has a little photophobia, and when exposed
at or excitement is rather restless and fractious. His pupils have seemed to
ated, but their reaction is normal.

ment has been simply to keep him perfectly quiet. His diet has been care-
d, and 0.18 gramme (3 grains) of bromide of potash has been administered
luring the day. At present his pulse is 98 and regular, his temperature is
F.), and his respirations 25 and rhythmical.

nosis is probably traumatic non-tubercular meningitis.

case (Case 261), which I have had brought to show you as possibly one of
r meningitis, is a child two and one-half years old.

ealthy at birth, and remained so until he was eleven months old, when he had
ing for two or three weeks, characterized by high temperature, but no other
oms beyond apparent irritation connected with the teeth.

was seventeen months old he had a similar attack, only more severe, accom-
irium, photophobia, high temperature, and, in a few days, paralysis of the
arm, while he could only move the right arm slowly. He had a tendency
ad to the right, and his head was retracted. The fæcal movements and the
rmal. He was unconscious for two days. An examination showed that there
bnormal in the ears, nor was anything abnormal found on physical examina-
. He cried out as though he had severe pain in his head when the attack
a few days he began to improve rapidly, and, although he had never talked
egan to express himself in words.

he following year he had some trouble with his ears, and grew very weak, so
not walk. Later he had an attack of croup, accompanied by perforation of
næ tympani.

s you see, he is comparatively well.

to the diagnosis of these last two cases, we are only justified in saying that
ue well, and do not show a return of cerebral symptoms, the most probable
f their condition is a non-tubercular meningitis.

xt bed is a little girl (Case 262), four years old, who fell and struck the back
She did not complain of much pain until the next day, when in the evening
lushed, she vomited, was restless, and was constipated. On the next day the
reased in severity, and two days later she entered the hospital. Her head was
here was an erythematous condition of the skin of the face, elbows, and knees.
re equal and reacted well. She was very restless, but showed no evidence
pulse was 132, the respirations were 44, and the temperature was 39.1° C.
She was able to take nourishment and to retain it. She had marked opisthot-
the following night and day she moved her hands continuously, and early
g vomited. The erythema of the skin gradually faded away. She talked
ost of the time. The eyes were fixed. The feet and hands were cold.

at times she showed labored breathing and the pupils were dilated. The
retracted so much, but the muscles of the neck were very stiff. The pulse
re feeble and slower. The abdomen was retracted, and there were petechiæ

on the face, elbows, and knees, most marked on the right side. Last night she was very restless and her breathing was again labored.

To-day, as you see, there is considerable twitching of her arms and legs. The patellar reflexes are absent; the plantar reflexes are diminished. Nothing abnormal is found on examination of the ears, throat, chest, and abdomen, or of the urine.

This case is probably one of traumatic non-tubercular meningitis. The erythematous efflorescence and the petechiæ would make me suspect that we might possibly be dealing with a case of cerebro-spinal meningitis. The symptoms, however, are not of so severe a grade as I should expect in the latter disease, while the acute onset following trauma would naturally point towards a simple inflammation of the pia mater. We cannot, however, in cases of this kind definitely determine the diagnosis without an autopsy. It is evident that there are no other diseases, such as typhoid fever or pneumonia, developing, and the possibility of its being a tubercular meningitis is exceedingly small, considering that she is at a period of childhood when the typical signs of this disease are most marked, and its type is of a subacute character.

(Subsequent history.) On the following day she sank rapidly, and she died, without any spasmodic movements or convulsions, on the eighth day of the disease.

LECTURE XXIX.

BRAIN.—(Continued.)

TUBERCULAR MENINGITIS.

form of leptomeningitis which I shall describe to you is
r meningitis, and I happen to have a number of children
disease in the wards of the Children's Hospital to show you

meningitis is a disease caused by the tubercle-bacillus at-
mater; it occurs most commonly in early life, runs a sub-
d is invariably fatal. The disease presents many irregu-
ıanifestations, and its typical symptoms vary according to
patient. The most typical cases of the disease are seen in
d. It occurs more commonly between the ages of five and
ıy other period of life. It is rare in the first year of life,
early months; the number of cases increases rapidly in the
decreases as rapidly after the eighth year. It is compara-
adult life that out of the large number of adult patients
et in my service at the City Hospital only a few cases
ıeningitis have come under my care in the last ten years.
ingitis, then, can be considered to be essentially a disease of
be most common in the middle period of childhood. In
of cases there is a tubercular history of one or both parents.
in the sense that the individual inherits tissues which are
eptive to and which provide a favorable material for the
the bacillus of tubercle.

should be protected in all possible ways against tubercular
er by its food or by human beings. The tubercle-bacillus
to attack individuals in cases where the question of inheri-
tely be eliminated. We should, therefore, take the greatest
en should not be under the care of tuberculous nurses, as
member of the family who comes into the closest relation
As an illustration of the truth of this statement I shall
seen by me in consultation with Dr. W. L. Richardson and
es.

33), five years old, died of tubercular meningitis. The autopsy showed
r lesions of the meninges, with enlarged bronchial lymph-glands and
he apices of both lungs. The child up to the time of the attack had
ly well. There was no history of tuberculosis on either the father's or
There were several other children in the family, none of whom had ever

shown any symptoms connected with tuberculosis. This boy at the age of fourte[illegible] months was placed in the charge of a nurse about twenty years old, who remained wi[illegible] him until he was four and a half years old. Just before leaving the child she w[illegible] brought into especially close connection with him while his parents were away for so[illegible] weeks. The child was very fond of his nurse, insisted upon being in her lap a great de[illegible], kissed her on the mouth, slept in her bed, and kept her in the nursery with him contin[illegible]ously. This nurse had a sister who died of pulmonary tuberculosis. She herself w[illegible] taken sick with the same disease while taking care of the child, and subsequently di[illegible] of it.

Other cases of this kind have been known to occur. Of course the possibility of coincidence must be thought of, but the fact that a robust child with no hereditary tube[illegible]culous history lives in close connection with a tuberculous nurse and dies of tuberculos[illegible] of the bronchial glands and cerebral meninges is at least significant.

It is not unusual to meet with a tubercular meningitis secondary t[illegible] tubercular disease of the spine. This complication occurred in a child fou[illegible] years old whom I saw in consultation with Dr. Scudder.

The child (Case 264) was being treated for Pott's disease with lateral deviation of t[illegible] spine. He was placed on a frame for five weeks, and at the end of that time he lost [illegible] appetite and weight and began to have a cough. Nothing especial, however, was found [illegible] the lungs. The bowels became constipated, and he then began to have some mental di[illegible]turbance and to vomit. A few days later he became unconscious, and on examining hi[illegible] his pupils were found widely dilated, uneven, and not responding to light. His temper[illegible]ture was usually about 38.8° C. (102° F.). The pulse and respirations were somewh[illegible] quickened. His head was retracted, and on the day of his death he had a convulsion.

Tubercular meningitis may also occur in connection with disease of t[illegible] hip, the latter being much more common than when the spine is affecte[illegible] I have seen a case of this kind in consultation with Dr. Brown which illu[illegible]trates the importance of recognizing the occurrence of this complication.

A child (Case 265), four years old, was being treated by an irregular practitioner f[illegible] disease of the hip-joint. The child had been allowed to drag itself about, and the tre[illegible]ment had been with drugs and not by apparatus. When the child was placed under D[illegible] Brown's care he had him taken to the country and placed in a house and room where [illegible] the hygienic surroundings were good. He kept the child in bed and treated it by mea[illegible] of the method of extension usually employed in these cases. The child at first began [illegible] improve, but after a few weeks lost in weight and in appetite. Its temperature, which h[illegible] been varying from 37.2° to 37.7° C. (99° to 100° F.), rose to from 39.4° to 40° C. (103° [illegible] 104° F.). A few days later the child became somnolent and had convulsions.

When I saw the case with Dr. Brown it was evidently one of tubercular meningit[illegible] apparently secondary to disease of the hip-joint, and the child died within twenty-four hou[illegible] after I had examined it.

In this connection I might mention that the tubercular form of otitis [illegible] not uncommon, and that it may be the starting-point for tubercular menin[illegible]gitis. Surgeons should, therefore, watch carefully the possible complicatio[illegible] of tubercular meningitis when treating tuberculous disease of the bones an[illegible] joints.

A knowledge of the general pathology of tubercular meningitis is o[illegible] great practical importance in acquiring a clear picture of the diseas[illegible] We must look upon the tubercular lesions as secondary manifestations of sion of diseases of the nervous system.

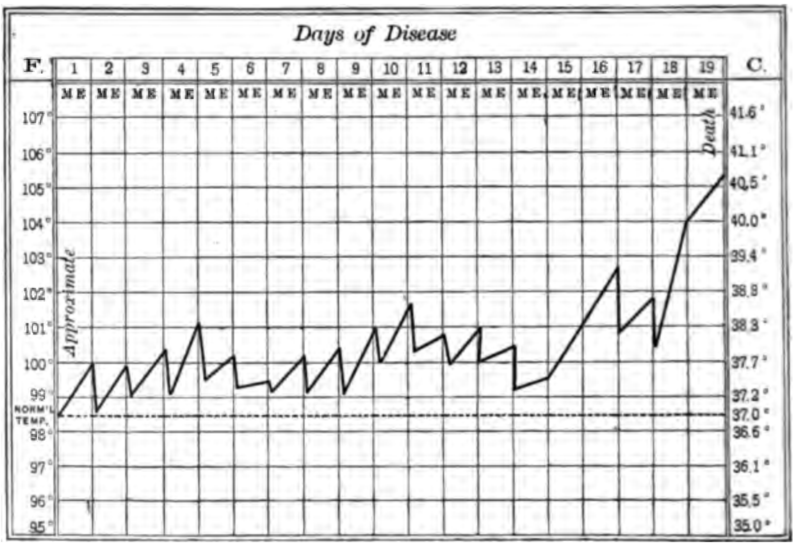
PATHOLOGY.—Although the nidus of the tubercle-bacillus which pro- s the pathological lesions of tubercular meningitis is in some other part he body, and the lesions of the brain and its meninges are always ndary, yet, as the clinical characteristics of the disease are those of a ıary cerebral nature, I shall describe only the morbid lesions which r in the brain.

The macroscopic pathological condition which is seen in the brain as a lt of the action of the tubercle-bacillus is a growth of miliary tubercle ıe meninges and in the cerebral substance. This growth is especially ked in the meshes of the pia mater along the course of the blood-vessels he base of the brain. These small granulations are conspicuously erous in the choroid plexus and cause great irritation in the neighboring s. This irritation is followed by a transudation of greater or less extent the ventricles. Accompanying this transudation is also a fibrino-puru- exudation between the pia mater and the cerebral convolutions at the of the brain, notably in the fissures of Sylvius, but at times covering whole convexity of the brain. The amount of exudation is not pro- ionate to the number of tubercles. The ventricles are sometimes so nded as to burst the septum. Pressure is thus brought upon the al portions of the brain, involving especially the optic thalamus, the us striatum, and the corpus callosum. While, as I have stated, the ptoms vary in different individuals and at different ages, the patho- al lesions, on the other hand, with the exception of their location, are paratively stable. What is of especial interest to us clinically, however, at, although in a typical case of tubercular meningitis in middle child- the symptoms, as a rule, correspond to the pathological lesions, yet me cases we find an entire lack of such symptoms as would naturally t from the wide-spread and prominent lesions.

ymptoms.—From what I have already told you in describing the

the nutrition is affected, that there are a lessened appetite, loss in weight, anæmia, and in fact symptoms which warn us that something is affecting the child's general health. This condition may last for many weeks, or even months, varying as to the time when the tubercle-bacillus has left its original nidus and migrated to the cerebral meninges. Only after this has occurred do we begin to get symptoms of cerebral irritation. The child now becomes peevish and capricious, and is in some cases easily frightened. As the tubercular growth increases and causes further congestion of the blood-vessels, the sleep is disturbed; the child complains of dizziness and slight evanescent pains in the head; it staggers slightly in its walk (static ataxia); sometimes it cries out sharply, especially at night (hydrocephalic cry). Vomiting not apparently connected with the food, and usually without nausea, is a common symptom. These are symptoms of irritation of the nervous centres, and may last for a week or two, according to the development of the pathological lesions. The temperature is usually moderately raised, 37.2°–37.7°–38.3° C. (99°–100°–101° F.), but on some days it rises a degree or so higher, and just before death a considerable elevation may occur. This chart (Chart 19) shows the temperature of a

CHART 19.



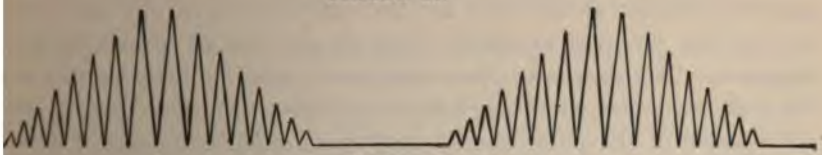
Tubercular meningitis. Male, 4 years old.

child five years old who lately died in the hospital. It represents very well what you will usually meet with in uncomplicated cases of tubercular meningitis. Of course it is impossible to determine the exact day of the beginning of the disease in such an affection as tubercular meningitis, so that the first day marked on the chart is merely approximate and serves as a starting-point to show the character of the temperature.

The pulse at first is somewhat quickened, but it soon becomes slower than al, and is apt to intermit. The respirations may in the early part of isease be quickened, and at times are of a sighing character. Obstinate ipation is a common symptom. Hyperæsthesia of the skin, with occa- l waves of congestion, especially of the cheeks, is sometimes met with. pain in the head increases, and the child is apt to hold its hand to its . Drowsiness, at first slight, soon becomes very marked. The child is etic and lies in bed, refusing to eat. The urine is scanty. There is phobia, and the pupils are contracted. Tubercles in the fundus of ye are rarely seen during life. Abdominal pains are quite frequent, depression of the abdomen (boat-shaped) is noticed in a certain num- f cases. Drawing the finger over the skin usually produces a bright ine, which becomes in a few minutes quite intense, and lasts perhaps ten teen minutes, which is much longer than would be the case in a healthy . This phenomenon is called the *tache cérébrale*, and is quite frequently with in tubercular meningitis, though it may be absent. This sign is, ver, in no sense typical, and is seen in a number of other diseases. child at this stage of the disease is apt to roll its head on the pillow st continuously.

he pathological irritation has now gone on to exudation, and we begin et symptoms of pressure. If diarrhœa appears, we should suspect rcle of the intestine. Sopor now comes on rapidly, and the child can roused only at times. Strabismus, nystagmus, and ptosis may appear. pupils are dilated and irregular, and their reaction is lost. The Mei- ian secretion is sometimes markedly increased. Convulsions, generally al, and of a rather mild type, may appear. At times paralysis of the or of the arm and leg (hemiplegia), and interference with sight (optic itis), may occur. In some cases the pulse now becomes markedly slow irregular, 50–60–70, and it is very common to find an intermission in the

CHART 20.



Cheyne-Stokes respiration. Tubercular meningitis. Child, 4 years old.

e, though this must not be considered as diagnostic of tubercular menin- . The respirations may not be perceptibly diminished at first, but soon me slow, 10 to 15 in a minute. A peculiar form of respiration, called yne-Stokes, usually occurs at this stage of the disease. This type of iration is characterized by complete or almost complete cessation of the iratory movements for a number of seconds. This is followed by a return of the respiratory movements, which gradually increase in h, rising for five or six inspirations and then fading away again so as to perceptible. This chart (Chart 20) represents this type of respirations occurring in the third week of the illness of a child four years old who died of tubercular meningitis.

A heightened temperature in tubercular meningitis indicates a complication of some kind, such as pulmonary tuberculosis, pneumonia, or tubercle in the intestine. At the end of the disease, however, the temperature rises rapidly, as do the pulse and respirations. Hearing, taste, and smell seem to be unimpaired for some time. The position which children with tubercular meningitis often take is somewhat characteristic. In all forms of meningitis they are apt to bury their heads in the bedclothes. There is often spasmodic retraction of the head, and they are inclined to lie with their knees drawn up. There may be spasmodic opisthotonos, as in this case which I shall presently show you (Case 272, page 618). The disease varies in its length, but usually lasts for from three to six weeks. Death may be preceded by continued convulsions for perhaps several hours. A striking feature which not infrequently occurs in the course of these general symptoms is a partial return to consciousness after the child has been lying in a stupor for several days. This phenomenon often induces the parents, and sometimes even the physician, to entertain hopes of improvement. It is, however, always delusive, for it has no favorable significance, and is soon followed by a more profound state of unconsciousness. These symptoms which I have mentioned do not, of course, always appear together, but may be present in different groups, varying with the individual. All the symptoms may disappear temporarily. There may be tonic as well as clonic contractions of the limbs and rigidity of the neck.

DIAGNOSIS.—The diagnosis of tubercular meningitis in the middle period of childhood, and with the sequence of symptoms which I have just enumerated, is not difficult, but you will at once perceive that the diagnosis in the early days or even in the first week of the disease must necessarily be very difficult. It is by watching the course of the symptoms and their general grouping, rather than by the consideration of any one symptom, or even one group of symptoms, that we are justified in making a definite diagnosis. The diagnosis, then, must, as a rule, be held in abeyance for many days. Reflex vomiting, with a moderate temperature, irregularity and intermission of the pulse, apathy, and many other symptoms of tubercular meningitis, I have often seen, both alone and in combination, in cases where they represented no cerebral lesion whatever. The active development and sensitive condition of the nervous system in childhood are so exaggerated in comparison with those of adults, that whatever disease may be present is liable to produce so profound an impression on the child's nervous centres that actual disease of these centres is readily simulated. Thus for days these apparently cerebral symptoms may mask by their undue prominence the symptoms of the real disease.

Illustrative of this difficulty are certain cases (Cases 466, 467) of pneumonia, which I shall describe to you in a later lecture (Lecture XLIX., page 984), in which the children had constant vomiting, soon became apa-

(2) poliomyelitis anterior, (3) *pneumonia*, (4) *malaria*, (5) *typhoid*
syphilis, (7) *rheumatism*, (8) *nephritis*, (9) *cerebro-spinal meningitis*,
non-tubercular meningitis.

iseases of the Stomach.—Unless the child is very young, acute
mptoms are, as a rule, not difficult to recognize after the first few
'e may at times, however, be suspicious of cerebral disease on
n an infant with continual vomiting and an elevated temperature
ere is no discoverable source of reflex irritation to account for the
s. This is especially the case if there are some irregularity of
n and a slow pulse. These may be cases of tubercular menin-
as I have described that disease in the first year of life. Again,
they may be simply cases of reflex vomiting. As illustrative of
of reflex gastric disturbance I shall cite this case:

e infant (Case 266), eight months old, was attacked with vomiting which lasted
intervals for two days. There were apathy and slow, intermittent pulse. The
e was 37.2° C. (99° F.). There were irregular respiration and rapid emaciation.
t made a perfect recovery in four or five days, and the case was evidently of
in. The slow, intermittent pulse, and the moderate temperature, which would
o alarming in an older child, led me in this case, as in others in the first year
liminate tubercular meningitis. In my experience this interpretation of symp-
roved to be correct.

oliomyelitis Anterior.—The following case of poliomyelitis an-
embled tubercular meningitis: it is, however, the only one which
en where the resemblance of the two diseases was so striking:

(Case 267), eighteen months old, showed for over a week symptoms closely simu-
e of tubercular meningitis. Obstinate constipation and apathy were present, fol-
nconsciousness; there were also a marked *tache cérébrale*, distended fontanelle,
ulse, contracted pupils, eyes turned upward, and convulsive attacks. Finally,
f one of the arms appeared, the general symptoms passed off, and the diagnosis
elitis anterior was easily made.

A male infant (Case 268), twenty months old, with a history of tuberculosis on the mother's side, began to show symptoms of anæmia and malnutrition with no perceptible cause, such as either improper food or bad general hygiene, to account for it. After two or three weeks it had attacks of unconsciousness lasting for hours; at other times drowsiness, with irregular pulse and respirations, was present. The temperature was 39.5° to 40° C. (103° to 104° F.). There were slight convulsions, and the fontanelles were distended. At first there was no periodicity of the symptoms, but a week later the attacks were evidently more pronounced every other day, and the infant was brighter on the intervening days. It lived in a malarial district.

On the administration of quinine and on removing the infant to a non-malarial region, these symptoms entirely disappeared.

The detection of the plasmodium would, of course, have determined the diagnosis in this case, but it could not be obtained.

Another case, which I saw in consultation with Dr. Parker, of Princeton, is also very instructive in warning us how careful we should be in making a diagnosis of tubercular meningitis in cases where there is a possibility of malaria being the cause of the symptoms.

A male infant (Case 269), fourteen months old, had always been well until fourteen days previous to the time when I first saw it. It then began to be fretful and to have diarrhœa. This condition continued for about a week, when it fell into a stupor, became very anæmic, and it was necessary to feed it by means of a dropper. At times it would cry out sharply. The temperature varied from 37.2° to 38.7° C. (99° to 102° F.). The respirations were usually regular, but at times were of the Cheyne-Stokes type. The pulse was about 120, sometimes regular, but at times intermitting. The pupils were sometimes contracted, but showed no irregularity. No other abnormal conditions were noticed, such as paralysis or symptoms connected with the lung, ear, heart, or throat, but the abdomen during the twenty-four hours previous to my examination was beginning to be depressed. The *tache cérébrale* was very distinct.

On close inquiry I found that there was a slight periodicity in the symptoms, shown by a rise of temperature on each afternoon and followed by the stupor becoming somewhat less. Although the infant had been unconscious for a week, and was becoming weaker and taking less nourishment every day, yet, on the supposition that it might possibly be an obscure case of malaria, I decided that quinine should be administered in suppositories. On the next day a slight improvement was noticed in the infant in the afternoon. It appeared less comatose, but its temperature and pulse remained as on the previous days. On the following day, which was the second from the time that it had begun to receive the quinine, it rapidly became conscious and began to drink milk. On the following days it was reported to have had a restless night and to have had two slight convulsions. Its temperature in the morning was 38.2° C. (100.9° F.), and the pulse was 115 and not intermittent. On the following day there was marked improvement in every way, and this continued without interruption for the next four days. The infant then continued to improve rapidly, the temperature and pulse becoming normal, and some months later it was reported to be perfectly well.

(5.) **Typhoid Fever.**—In my experience typhoid fever in young children is the disease which, next to non-tubercular meningitis, is most likely to simulate and be mistaken for tubercular meningitis. We may also have considerable difficulty in differentiating tubercular meningitis from the non-tubercular meningitis which may occur in the course of typhoid fever. The extreme cerebral congestion which at times arises as a symptom of typhoid fever may also add fresh difficulties to the differential diagnosis. The isive point, however, between typhoid fever and meningitis, whether bercular or non-tubercular, is the absence of leucocytosis in typhoid er and its presence in meningitis, provided that the latter is to any gree purulent.

According to E. S. Wood, in meningitis the chlorides in the urine di- nish rapidly; heating the urine precipitates the phosphates readily, and amount of indoxyl is increased: the reverse of these reactions occurs in hoid fever.

(6.) **Syphilis.**—The history and general symptoms of syphilis are to be ght for where a syphilitic meningitis is suspected. The temperature is especially high, and the symptoms are seldom acute. The pathology is d to be usually that of a chronic basic meningitis.

(7.) **Rheumatism.**—Rheumatism is said to occur as a cause of menin- is, but this must be rare, and I shall merely mention it, as I have never t with a case of this kind. A high temperature and acute symptoms are d to be the rule in rheumatic meningitis.

(8) **Nephritis.**—In addition to the other diseases which may simulate bercular meningitis should be mentioned nephritis, in which the symp- ns of uræmia simulate, to a certain extent, those of tubercular men- gitis. The urine should always be examined in doubtful cases of this nd, as where uræmic symptoms resulting from nephritis are present the sease will be shown by such examination, and we shall thus be able to fferentiate it from tubercular meningitis.

(9) **Cerebro-Spinal Meningitis.**—It is often quite difficult to differen- ate the early stages of tubercular meningitis from those of cerebro-spinal eningitis. In typical cases, however, the diagnosis is easily made, as the ng prodromal period of tubercular meningitis, as a rule, does not occur in erebro-spinal meningitis, and the temperature in the latter disease is almost lways high, while in the former it is, as I have already told you, raised to nly a moderate degree. In fact, all the symptoms of cerebro-spinal men- ngitis are markedly acute in comparison with those of tubercular menin- itis, which is essentially a disease of a subacute character. I shall pres- ntly show you a case of tubercular meningitis (Case 272, page 618) which imulated cerebro-spinal meningitis very closely.

(10) **Non-Tubercular Meningitis.**—On closely studying what I have lready told you of the symptoms of meningitis in general, you will be ble in the great majority of cases to differentiate it from other diseases, rovided that you do not attempt to make the diagnosis too early. Re- nember that you are seldom warranted in making an early diagnosis, in iew of the wide range of possible nervous symptoms which can be met vith in young children. Having determined that the disease is of cerebral rigin, we must next differentiate between the tubercular and non-tubercu- lar forms of meningitis by means of the broad rules of which I have just poken, and which I have condensed and simplified by means of this table (Table 101, page 612).

TABLE 101.

Cerebral Meningitis.

Non-tubercular.	Tubercular.
Usually secondary (possibly primary).	Secondary.
Not hereditary.	Hereditary.
Acute.	Subacute.
Prodromata short, if any.	Prodromata long, decided.
Headache severe at once, with delirium early, and soon followed by somnolence.	Headache less severe at first, but gradually increasing; delirium less common and milder.
Photophobia extreme.	Photophobia not so marked.
Convulsions violent.	Convulsions less violent.
Temperature high.	Temperature moderate.
Pulse and respiration rapid.	Pulse and respiration slow and irregular.
Duration short.	Duration long.

Transudation into the ventricles may occur in either form. The younger the infant the nearer the two forms approach each other in the similarity of their symptoms. Caillé has lately shown the value for diagnosis of Quincke's method of tapping the spinal canal.

Infantile Tubercular Meningitis.—According to some extended observations made at the hospital in Stockholm, infantile tubercular meningitis is characterized in the first year by an absence of prodromata, the sudden development of acute symptoms, a short course, and a fatal issue. The temperature is high, 38.8°–39.4°–40° C. (102°–103°–104° F.). The respirations are quickened and comparatively regular, 30–40–50. The pulse is high, 130–140–150. Clonic spasms and strabismus often occur. Paralysis is quite frequent, and diarrhœa is present rather than constipation. Bulging of the fontanelles is usual. Sinking of the abdomen is rare. Vomiting may occur, but is not especially common. Sharp cries are occasionally met with. The differential diagnosis from non-tubercular meningitis is difficult. Sopor and coma at the end are frequent in both diseases. The duration is seldom more than a week. It may be only two days, yet in rare cases the infant, like the child, may live for a month.

During the second year the symptoms of tubercular meningitis become of rather an irregular type, sometimes assuming the character of those which are seen in the first year, but soon corresponding more nearly to those which are met with in the middle period of childhood.

Prognosis.—Where we are sure of our diagnosis, I believe that in our prognosis we should give no hope of recovery whatever, except that in the extremely rare cases which I have just mentioned a temporary remission may take place. The reported cases of absolute recovery from tubercular meningitis cannot but be looked upon with scepticism. Indeed, the non-tubercular forms of meningitis simulate the tubercular so closely that without post-mortem verification recoveries can be supposed to be possible, but can hardly be accepted as proved.

Treatment.—The treatment of tubercular meningitis up to the time when the diagnosis is established should be purely symptomatic; later we ollowing case illustrates very well the tubercular meningitis of hildhood:

(Case 270), five years old, had always been well and strong. On December 3, eavoring to climb into bed, he fell and struck the back of his head. He cried , but the blow left no mark, and nothing was thought of it. The following day, ing, he fell and struck the back of his head, but the blow was no more serious ad often had before. On the next evening he went to a children's party, ate nusual, went to bed early, and slept all night. On the following day he was eat and was somewhat fretful, both of which conditions were unusual for him.

ecember 29 he had a slight follicular tonsillitis. His pulse and temperature were e cheeks were flushed, the eyes dull, and the pupils normal. His head was t, and he was dull and drowsy. He did not have any movement of the bowels for but on the third day they were moved by means of medicine. He continued to be he same condition until January 2, when his temperature was 37.2° C. (99° F.), lse 64, regular and strong; his face was flushed, and his eyes were vacant and He vomited once on that night, passed his water involuntarily, moved his left leg lly, and clinched his hands occasionally. He was evidently uneasy, and moaned l.

e following day the pulse was occasionally intermittent. In the mean time he re and more drowsy, and finally relapsed into a state of unconsciousness.

e 3d of January the pupils were normal, but he was completely unconscious. rature was 38.1° C. (100.6° F.), the pulse 180, and the respirations 30.

the child on January 4, and on making a careful physical examination found normal, except a slight congestion of the ear in the neighborhood of the malleus, e back over the apex of the lung was a slight elevation of pitch on percussion. erature was 39.1° C. (101.2° F.), and the pulse was 89 and strong. There was le twitching of the arms, chiefly on the right side, lasting from ten to twenty The pupils were slightly contracted, but were alike. That night he drew his l across the face with a quick trembling motion, the right leg being drawn up hole body trembling; occasionally there was moaning and sighing respiration.

. J. Blake, who examined the ears, reported that there was a slight congestion in ior canal of both ears and also in the neighborhood of the right malleus. Both e tympani were clear, normal, transparent, and without injection of the manubrial els. There was, in fact, no evidence of disturbance of the ears. On the posterior ch external auditory canal at the anterior third, more pronounced in the right than

The extremities of the right side were absolutely motionless, and sensation was apparently absent. The child lay, as a rule, perfectly quiet, as though asleep, and at times would present the picture of a perfectly healthy child sleeping.

On January 9 the extremities became cold, the face very pale, and the pulse imperceptible. This condition lasted fifteen minutes, when he improved in appearance. During the night the breathing grew very rapid, he was restless, moved the left arm continually, and moaned. After some time he opened his eyes, looked around the room, and then became quiet and slept. The next day he was slightly unconscious, and the fingers were flexed, with a very strong contraction of the muscles. The breathing then became more difficult, the nostrils being widely dilated with every breath. During the night he was conscious for some time, swallowed water without difficulty, and the eyes were wide open.

On January 11 there was ptosis of the right eyelid. The pulse became regular, compressible, and intermittent. The left arm was occasionally raised to the head with a quick spasmodic motion, the child moaning as if distressed. Later the eyes became fixed, the pupils dilated, the nostrils expanded, and a bluish color appeared around his mouth and nose. The breathing became very difficult. During an attack of this kind he had every appearance of being moribund, and each attack was thought to be his last.

The change from day to day in the child's general condition was almost imperceptible. He was, however, gradually becoming emaciated.

On January 12 the pupils of both eyes were much dilated; the right eye was almost motionless, with ptosis of the right lid, while the left eye moved occasionally from side to side in a circle. The face was livid, and the hands were mottled with bright red spots. Later, the left eye became quiet and had a slightly contracted pupil.

On the following day, January 13, the movements of the left eye were repeated, the right pupil being dilated, while the left one was contracted. During all this time the enemata were retained, the bowels moved regularly, and the urine was passed normally. The pulse was so weak that at times it could not be found at all at the wrist, and the breathing was at times inaudible and almost imperceptible.

On January 17 there was slight discharge of pus from the mouth, and also from the left eye. During the next day his breathing grew more and more difficult, and it seemed as though he could not possibly live much longer. In the evening, however, his respiration was much easier and his whole appearance was greatly improved. His breath was very offensive, and there was a loud bubbling sound in the throat.

On January 19 the right nostril was much more dilated during inspiration than the left. The forehead was shiny and slightly œdematous, and the veins were plainly mapped out. Occasionally he moved his right hip-joint and shoulder, which had been motionless for days. There was another slight discharge of pus from the mouth, and when his lips were wiped he seemed more sensitive to touch than before. During the night his left arm and left leg were constantly moved, and he moaned as though he were still in pain. His forehead was still œdematous.

During the next day he was in a state of deep coma for four hours. He then drew a deep sigh and seemed somewhat conscious. The pulse was soft, intermittent, and fluctuating.

On January 20 he partly opened and shut his right eye, which was very sensitive to light. The breathing was difficult and noisy. The face was covered with perspiration. At 10 P.M. the sighing respiration began again, and at 10.15 he died quietly, on the thirty-first day of the disease.

The autopsy was made eighteen hours after death by Dr. W. W. Gannett, and the report was as follows:

The body was much emaciated. There was slight lividity of the dependent portions. Rigor mortis was present. Nothing unusual was observed about the calvaria or dura mater. The sinuses of the latter contained partly congested blood. The pia mater of the convexities of the brain was very dry, and the minute vessels were injected. The convolutions were flattened. The sulci were obliterated. The pia of the base along the chiasma and in the fissures of Sylvius was thickened, and there was an opaque yellowish-gray color from the presence of a fibrino-purulent material in its meshes. In the above situations, on the under surface of the frontal and temporal lobes, also on the pons and inner lers of the occipital lobes, were to be seen very numerous, gray, translucent nodules ut 2 mm. ($\frac{1}{12}$ inch) in diameter. The lateral ventricles each contained about 50 c.c. ounces) of a slightly opaque fluid. The ependyma was thick, grayish, and opaque. choroid plexuses and velum interpositum were markedly injected. In the latter were e seen several small nodules similar to those described in connection with the pia of the .

A section of the hemispheres showed nothing remarkable, the puncta cruenta being of ıt the usual size and number.

The basal ganglia, pons, medulla, and cerebellum also showed no appearances worthy pecial note.

The heart was normal.

The pleural surfaces on both sides were free from adhesions; the pleural cavities coned no fluid.

Both lungs retracted readily, and were crepitant everywhere except at the apices, where ll nodules could be felt within the tissue.

On section an opaque, grayish-yellow, cheesy nodule, 6 mm. ($\frac{1}{4}$ inch) in diameter, ounded by a narrow border of gray and translucent tissue, was found at the top of the lung. At the top of the right lung were several closely aggregated nodules of a similar earance, forming together a mass about 2.5 cm. (1 inch) in diameter. The other pors of the lungs were normal.

The bronchial lymph-glands were enlarged to 1.2 cm. ($\frac{1}{2}$ inch), showing on section a ow, opaque, crumbling material.

The spleen was of the usual size, color, and density. On section the follicles and eculæ were found to be fairly distinct; the pulp was firm and of a pale red color. Two hree gray, translucent, sharply defined, slightly projecting nodules, 1 mm. ($\frac{1}{25}$ inch) in neter, were to be seen. The kidneys were normal. In the lower third of the ileum a of substance of the mucous membrane was found in several places. The edges of these ns were elevated and their bases granular. The liver was found to be normal.

The **pathological diagnosis** was—

Tubercular meningitis,
Acute hydrocephalus,
Ependymitis,
Tuberculosis of the velum interpositum,
Tubercular nodules in the lungs,
Tuberculosis of the bronchial lymph-glands,
Tuberculosis of the spleen,
Tubercular ulcerations of the intestines.

I have here in Bed 3 an interesting case of tubercular meningitis to w you.

This boy (Case 271) is three years old. There is no history of tubercular or syphilitic ase in the parents.

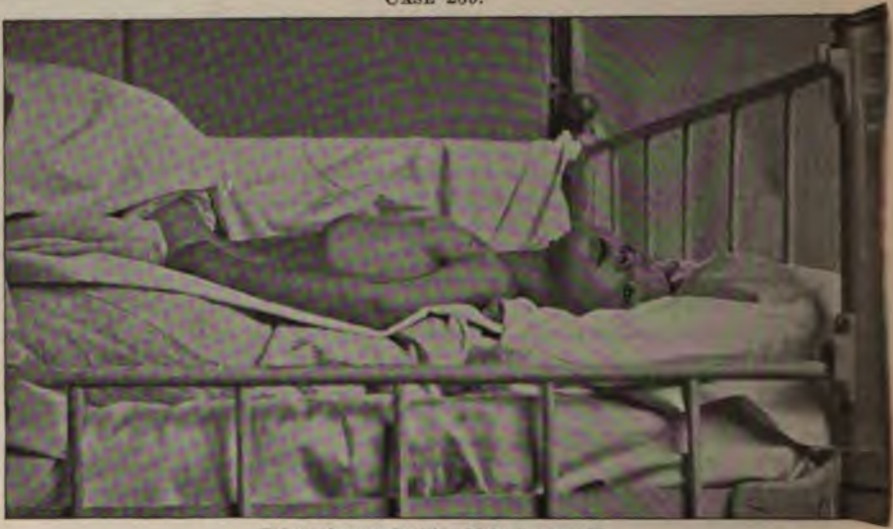
Three weeks before entering the hospital, the child, who had previously been healthy, an to complain of pain in the abdomen, and to have anorexia and a feeling of general aise. Somewhat later it was noticed that the eyes would at times turn inwards and that head would be drawn back. He was in this condition for two weeks before entering the pital.

On March 13 he was brought to the hospital, and was found to have a temperature of ° C. (101.2° F.), a pulse of 120 and not intermitting; the respirations were 40. He in an unconscious and drowsy condition. His head was drawn back, and he did not h to lie on his back. The tongue was not coated. An examination of the heart, lungs, urine showed nothing abnormal. An examination of the eyes, made by Professor O. Wadsworth, showed the pupils to be dilated, but equal in size and reacting to light. ere was internal strabismus of both eyes. There was optic neuritis and the beginning of an atrophy following the neuritis. The patellar reflexes were absent, and there was no ankle-clonus. The superficial reflexes were normal. There was no tenderness of the head or spine. An examination of the ear, which was made by Professor J. O. Green, showed nothing abnormal.

On March 16, as you remember, I examined the child before you with Dr. Bullard. At this time he showed nystagmus with conjugate deviation to the right or to the left, according to the side on which he lay. No *tache cérébrale* was found.

On the 17th an erythematous congestion was noticed on the right cheek, and he became still more somnolent.

Case 269.



Tubercular meningitis. Male, 3 years old.

On the 18th the head was much less retracted. He had vomited once during the night and once in the morning.

On the 21st he had a convulsion, which was the first that had occurred during the course of the illness. He was also found to have partial opisthotonos. The legs did not participate in the contraction, but the head was drawn back almost to the buttocks. He was found to have Cheyne-Stokes respiration. During this day he had four or five convulsive attacks, and remained in a condition of opisthotonos in the intervals between the attacks. These convulsive attacks lasted about half a minute each, and the intervals between them were about four minutes. There was incontinence of urine and of fæces. The pulse was rapid and irregular, and the extremities were cold. The *tache cérébrale* was obtained on this day, and lasted for twelve minutes. 0.12 gramme (2 grains) of chloral and 2 grammes ($\frac{1}{2}$ drachm) of brandy were given subcutaneously. The convulsions ceased, the opisthotonos disappeared in twenty minutes, and the child remained quiet.

On the 26th the record was that for two days the child had been decidedly better, the retraction and strabismus were less, the nystagmus had disappeared, and he had recognized and spoken to his father. The *tache cérébrale* could be obtained, but was less distinct, and the temperature was normal.

On the 28th he became worse again. His head was again retracted, but he was not wholly unconscious. There was retention of urine, for which he had to be catheterized.

On the 29th he had a convulsion lasting three minutes, in which the right arm was jerked up over his head. This was followed by partial opisthotonos, and then by a general convulsion lasting two or three minutes, during which his eyes rolled up. At times he would have convulsive movements and tremor without actual convulsions.

To-day (April 7), as you will notice, the right hand lies motionless by his side and is in a state of extreme pronation. He is unconscious, and all the abnormal symptoms have rned. You see that he has the characteristic aspect of a typical case of tubercular ingitis. The eyes are open and staring, the head is drawn back, the abdomen is icted, and on drawing the finger over the thigh you see the *tache cérébrale* is very ked. The respirations are of the Cheyne-Stokes type, the pulse is intermittent. The berature has varied from 37.2° to 38.8° C. (99° to 102° F.), but has risen within the last ve hours to 40° C. (104° F.), which indicates that the fatal issue of the case is very .

(Subsequent history of the case.) On the following day there were a number of con- ons occurring in rapid succession, especially involving the left side. The child groaned sighed a number of times; his arms and legs were rigid, his eyes were rolled upwards. wo o'clock in the morning he took some milk, but after that refused it, and from that until his death, at 7.25 A.M., he was in a condition of continued convulsions.

I shall now ask you to come to the autopsy-room, in order that you may the results of the post-mortem examinations of some children who have of tubercular meningitis. Dr. Gannett has some specimens here to v you of a case which has just died in the hospital. When the patient alive the case simulated cerebro-spinal meningitis very closely, and you e already seen it in the wards. It is a very instructive case, as it is an sual one, and illustrates an important fact in connection with tubercular ingitis,—namely, that the patient may recover temporarily from an ck of the disease and finally die of a recurrent attack. This is, how- , a very rare occurrence.

You may remember that when this infant (Case 272) was alive I explained to you the ulties which may arise in making a definite diagnosis in cases where cerebral symptoms present.

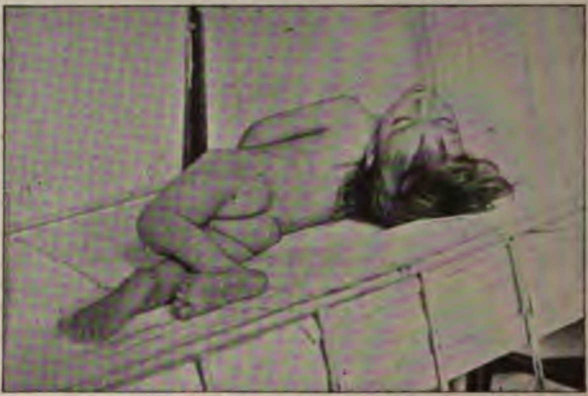
It was twenty-one months old when it entered the hospital. The history obtained the mother was that she had always been healthy, but that the father was supposed ave had the primary lesion of syphilis three years previously, although no secondary ifestations had appeared. The infant was born after an unusually long labor with a onged forceps delivery.

It was stated to have been healthy until it was nine months old. At that time it had nvulsion, which first affected the right and then the left side. It was unconscious for days, and was somnolent for four weeks. Two or three weeks later its general condition roved. During this time the infant did not use the muscles of its left side or limbs, it could laugh only with the right side of its face. Its body was turned continuously he left; sensation was not interfered with. It gained slowly in strength, and the symp- s gradually disappeared, until it was thirteen months old, when it seemed to be com- atively well, all motor disturbances having ceased. In the following months it had ew slight attacks of the same nature. The final attack from which it died occurred en it was twenty months old, and began with a convulsion on the right side with tching of the muscles on the left side and frothing at the mouth. There was also sis of the left eye. It did not cry out when going into the convulsions, but had marked sthotonos, which lasted, to a greater or less extent, for five weeks. During these five eks it was unconscious, and there were several slighter attacks.

On entering the hospital, physical examination showed that the infant was of me- m size, pale, poorly developed and nourished, and unable to stand, the left leg being aker than the right. Nothing abnormal was found in connection with the heart or gs. She could use her extremities partially, but there was an evident motor disturbance the whole of the left side, and she took hold of objects with her right hand only. The ex and little finger of the left hand were frequently found to be extended, the second and rd fingers being flexed partially. There was also slight drooping of the left eyelid, and lines of the left side of the face were obliterated. There was a very slight drooping of the left corner of the mouth. There was slight strabismus of the left eye, and an apparent lack of power of the left external rectus muscle. The patellar reflexes were exaggerated on the left side. Examination of the ankle-clonus was negative. The epiphyses of the wrists were somewhat enlarged. The child could not speak, and apparently could not understand readily. No evidence of a history of cerebral injury could be obtained. The circumference of the chest was 1 cm. ($\frac{3}{8}$ inch) larger than that of the head. The cause of the disease was so obscure that at this period the diagnosis could not be definitely made, the supposition being that the child was suffering from the results of an attack of cerebro-spinal meningitis, or possibly from tertiary syphilis, or that a cerebral hemorrhage had taken place, with a resulting spastic paralysis.

While in the hospital the child presented a number of different nervous phenomena. At times she would appear to be for days semi-comatose and would not take notice of anything about her; the eyes rolled up and she would have slight twitching of the body, but this was not localized, and there were no convulsions. At another time, while sleeping quietly during the night, she was found to be unconscious in the morning, and to have her head slightly drawn back and her eyes turned up. Nystagmus was present, and the pupils were dilated and did not react to light, but were equal in size. Clonic twitching of the right foot and the muscles of the right side, flexion of the fingers of the right hand over the thumb, and twitching of the muscles of the wrist sometimes occurred. There was twitching of the fibres of the sterno-mastoid muscle on the right side. There was also twitching of the right side of the face. There was no spasm on the left side, except of the left sterno-mastoid, but there was a nystagmus of the left eye. These clonic twitchings were rhythmical and occurred 180 times a minute. The pulse was 172, and was very feeble. The respirations were 80, rapid and rattling; the temperature was 39.4° C. (103° F.).

CASE 272.



Recurrent tubercular meningitis. Female, 21 months old.

From 2 A.M. until 5 A.M. 0.36 gramme (6 grains) of chloral was given by enema, and 0.36 gramme (6 grains) of bromide of potassium was given every three-quarters of an hour by the mouth, alternating with the chloral. The spasms became less marked after 3 A.M., but continued in a mild degree up to 11 A.M. During the remainder of the day the child lay in a stupor, but had no convulsions. It was able to swallow brandy and milk, which were given to it by the mouth in small quantities at different intervals.

On the day following this attack the report was that the child had slept well, and that there was more or less stupor, but there had been no convulsions.

On the following day the condition remained about the same, but on the next day she apparently had attacks of pain, when she would straighten herself out, throw back her head, and cry out.

On the following day, about 11 A.M., she began to have the same twitchings as in the attack previously mentioned. They were of the same character, except that the extensor les of the left foot contracted feebly. The convulsions ceased at about 2 P.M., and hild remained in a stupor.

On the following day it was reported that she had had no convulsions, but apparent ks of pain, when she would cry out and throw her head back, and that she had had ttack of opisthotonos, in which condition you will remember you once saw her. This ition of opisthotonos at times would be much more marked than when you saw her, at the heels would almost touch the back of her head. The next symptom which ared was stupor. The temperature at this time was considerably elevated.

On the following day there were no convulsions, and her condition was about the same the previous day, but the head was drawn back and was rigid, and the legs were drawn nd were held rigidly. She lay in this condition, most of the time in a stupor, crying ccasionally, and moving her left hand and arm more than she did the right. At times ould appear to be sleeping naturally and the rigidity would pass away.

The opisthotonos gradually became more marked and more frequent in its occurrence, although the bowels were moved regularly every day, she took less nourishment, and mperature continued to rise, and varied from 37.7° to 40° C. (100° to 104° F.).

The time when you saw her in the condition of opisthotonos was the sixth week from ime of this last attack. During the last week of her life the opisthotonos became less ed, and at times passed away entirely. She opened her eyes, but the pupils reacted slightly. The left pupil became somewhat larger than the right and reacted slightly, the right pupil did not react at all. The spastic condition of the right wrist and left persisted, the patellar reflexes were equal and normal, and the child lay in a semi- r, with a temperature varying from 38.3° to 39.4° C. (101° to 103° F.). She took less ess nourishment, and had a slight cough. She gradually lost in weight and became er, and on the day before she died her respirations for a time were very rapid, running 100 a minute. Death took place apparently from exhaustion.

The long duration of this last attack, embracing a period of eight or nine weeks, made iagnosis very difficult, and prevented us from making the clinical diagnosis of tubercu- eningitis, which these specimens just found at the autopsy prove to be the disease by h the child was affected from the beginning.

This chart (Chart 21, page 620) represents the temperature, pulse, and respirations of ase during the last twenty-one days of its life.

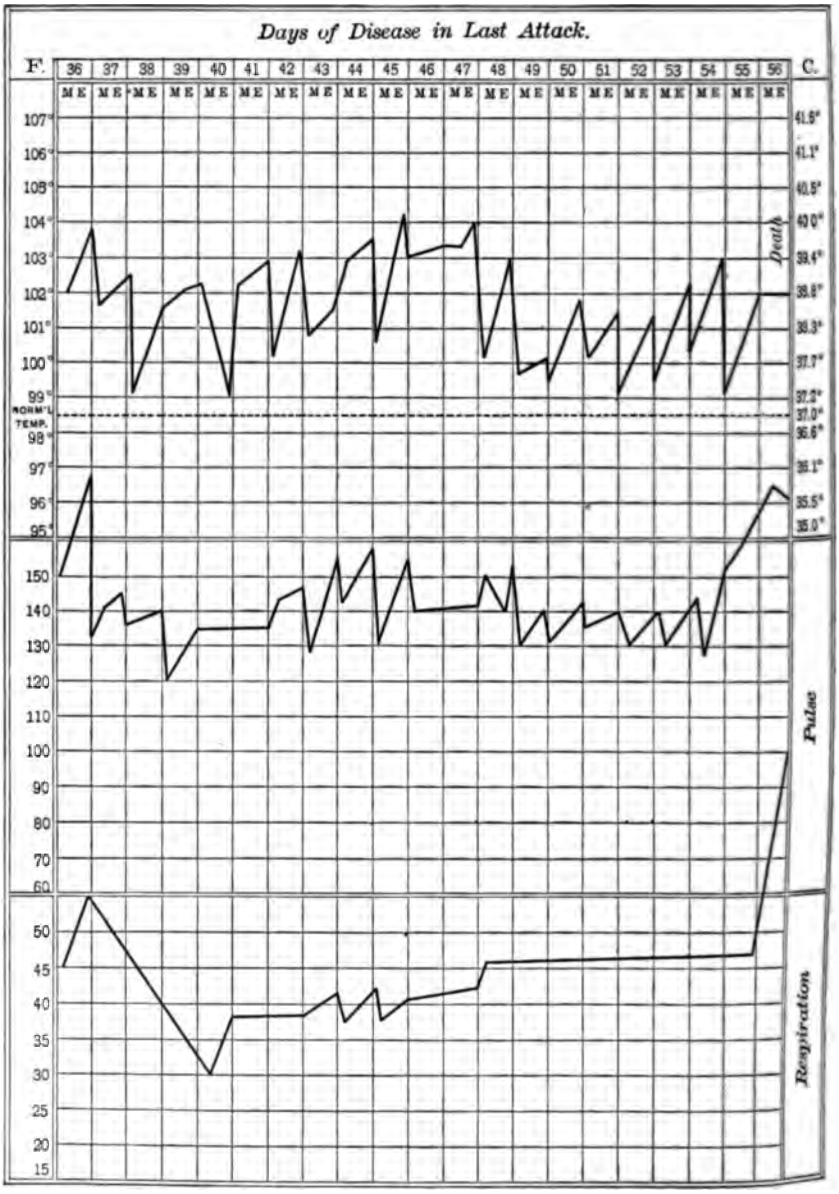
On examining the brain you see that the dura mater is normal, the pia mater of the xities is pale, and the cerebral convolutions are somewhat flattened. The pia mater base of the brain shows considerable infiltration with fibrin, which is quite firm, but is little or no injection of the blood-vessels. In many places in the portions of the pia at the base of the brain where the meshes of the pia are not infiltrated with fibrin, nodules as large as a pin-head are to be seen. The lateral ventricles are at least six the usual size, the layer of brain-substance between the cavity and the convexity being derably thickened. The ependyma of the lateral and fourth ventricles is thickened granular. On section the brain-substance is found to be pale, and the puncta cruenta . Sections of the basal ganglia, pons, medulla, and cerebellum show that the brain- ance is normal. The spinal cord shows in gross nothing unnatural. The heart is al. Beneath the pleura of both lungs numerous gray nodules the size of pin-heads are seen. At the apex of the left lung is a cheesy nodule 0.5 cm ($\frac{1}{5}$ inch) in diameter. lungs are extensively studded with gray miliary tubercles. The spleen and kidneys similar appearances, and the bronchial and lymph glands are very much enlarged and throughout their substance cheesy degeneration.

The **pathological diagnosis** in this case is—

Subacute tubercular meningitis,
Chronic granular ependymitis,
Chronic hydrocephalus,
Atrophy of the brain-substance,
Miliary tuberculosis of the lungs, spleen, and kidneys,
Chronic tuberculosis of the lung.

As Dr. Gannett has explained to you, some of the tubercular lesions are of recent growth, while others are evidently old ones and representative of a former attack. You see, therefore, that the presence of older tubercular lesions in the meninges, as well as of

CHART 21.



Recurrent tubercular meningitis. Last 21 days of life.

those which produced the symptoms in the last attack from which the infant died, proves to us that the case is one of recurrent tubercular meningitis.

These cases of recurrent tubercular meningitis are rather rare, and the disease is so uniformly fatal in the first attack that I shall recall to your minds the case which was under Dr. Townsend's care at the Good Samaritan Hospital.

A little girl (Case 273), five years old, entered the Good Samaritan Hospital with hip-disease on the left side and dorsal Pott's disease. She was treated in bed for these diseases, and did very well for a time, but on May 7, after a week in which she showed anorexia and loss of weight, she began to vomit, and on the following day she complained of headache and photophobia. She rolled her head from side to side. Her bowels were constipated, and could not be moved by enemata, and her abdomen was much retracted. This continued for four days, with at times delirium, accompanied by marked drowsiness. There were also ptosis of the left eyelid, slight convulsive movements of the limbs, and frequent putting of her hands to her head, as though she were in pain.

On May 12 she had recovered so much that she played with the other children and called for her books and toys. The left pupil, however, remained a little smaller than the ight.

On the 15th of May, and again on the 20th, 21st, 25th, and 27th, the patient became drowsy, and complained of headache. In the intervals between these attacks she seemed bright and well. During the drowsy periods her abdomen was retracted and her bowels were constipated.

From the 27th of May until the 20th of July she appeared as well as usual. On the atter date her temperature suddenly rose to 40.1° C. (104.2° F.). She had pain in the ead and photophobia, and the right pupil was larger than the left. This lasted only two lays. She then became bright and well again, and continued so for over ten weeks.

On October 2, having been perfectly well on the previous day, she began to vomit and o complain of headache. Two days later she fell into a stupor and became completely omatose.

On October 6 the left pupil was widely dilated and the right one was contracted to the ize of 2 mm. ($\frac{1}{12}$ inch); there were convulsive movements, and later in the day she died.

The post-mortem examination showed a recent tubercular meningitis. In addition to hese lesions there were found some older large tubercles of the brain and the remains of the revious attacks of tubercular meningitis.

Nothing else of importance was detected in the other organs.

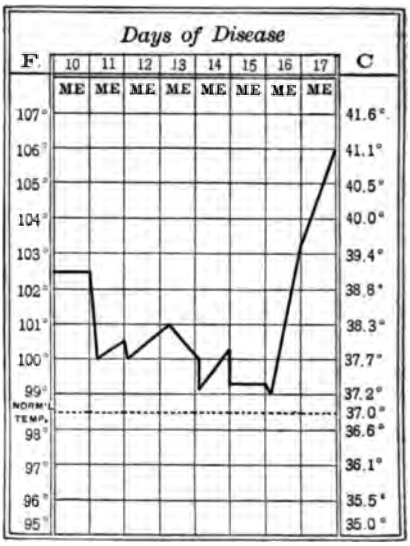
I will now ask you to return to the wards, to see a case of tubercular neningitis in a child, two and a half years old, who entered the hospital at vhat was supposed to be about the tenth day of the disease.

The history of the case (Case 274) is that the father's mother and the mother's mother nd brother died of consumption. When this child was one year old he had measles, otherwise he had always been well. About two or three weeks ago it was noticed that the child lept more than usual. At that time he appeared to be feverish and his tongue was noticed o be coated, but there was no nausea nor vomiting. A few days later he vomited once or wice during the day. The bowels were constipated. Eight days before entering the hospital he had a slight convulsion, and three days later he cried a great deal, as if he were n pain. Two days before entering the hospital he had a number of convulsions during he night, each lasting about ten minutes. On the following day the convulsions occurred gain. On the day he entered the hospital he began to have convulsions at three o'clock, which lasted about two and a half hours. He was also noticed to have marked internal trabismus of the left eye and slight strabismus of the right eye. The muscles of the neck were somewhat contracted. There was no paralysis of the extremities.

The pupils were equal, they reacted to light, and were somewhat dilated. The conjunctivæ were injected, the left one especially so. Sensation was not impaired. The knee-jerks and ankle-clonus were absent. There was a marked *tache cérébrale*. The respirations were irregular and sometimes of the Cheyne-Stokes type. The child was unconscious and was very pale. The heart was found to be beating very rapidly, sometimes as high as 200 beats in a minute. No souffles were detected. The temperature was 38.8° C. (101° F.). During the next day the child lay in a state of stupor. He continually moved the left forefinger and thumb, kept drawing the head to the left, and was very restless. He was reported to have cried all night and to have put his hand to his left ear. He lay with his eyes wide open, took nourishment well, and had less strabismus than when he entered the hospital. On the following day (about the thirteenth day of the disease) he was very restless, had sordes on the teeth, and his tongue was very dry. Examination of the ears showed nothing abnormal. The abdomen was somewhat retracted. He was less restless, and slept a good deal. The bowels were moved regularly, and the movements appeared to be well digested. He took about 90 c.c. (3 ounces) of milk every two hours. On the following day there was no especial change, except that the muscles of the neck were firmly contracted and the *tache cérébrale* came out more slowly than on the previous day. A slight paralysis of the left side of the face appeared on this day. The left eyelid moved rather slowly, and the left corner of the mouth seemed to drop a little. The pulse was irregular, of fair strength, and intermittent. He did not take his nourishment so well. Yesterday the child was in about the same condition.

To-day you see that he is lying in a comatose condition, with his eyes half closed. The pupils are rather irregular, dilated, and do not respond to light. The face is somewhat cyanotic, especially about the nose and the eyes. The respiration is decidedly of the Cheyne-Stokes type. The pulse is irregular and intermittent. On drawing my finger over his thigh you see that the *tache cérébrale* is well marked and that it lasts about ten or fifteen minutes. The head is somewhat drawn back. What I wish especially to call your attention to is the temperature chart (Chart 22). You will notice that the temperature had risen yesterday from 37.5° C. (99.5° F.) to 39.5° C. (103 2° F.), and that it is now rapidly rising until it has reached 41.1° C (106° F.).

CHART 22.



Tubercular meningitis. Male, 2½ years old.

This rise of temperature is very significant, and denotes that the child will die very soon.

(Subsequent history.) The child died quietly on the evening of what was supposed to be the seventeenth day of the disease.

The autopsy was made by Dr. Mallory. Rigor mortis was present; the left pupil was dilated; there was moderate lividity of the dependent portions of the body.

Heart.—The right ventricle was dilated and contained dark, clotted blood. The valves were normal.

Lungs.—A number of small, flattened, gray masses were found in the pleura; on n they were found to be miliary tubercles. The right lung was adherent to the tal pleura by strong fibrinous adhesions, beneath which were miliary tubercles, especially e areas covering the ribs, the diaphragm, and the upper third of the sternum. A small ber were also found in the substance of the lung. The bronchial glands were enlarged, f them being 1.2 cm. (½ inch) in diameter. This gland on section was yellow and some- broken down.

Spleen.—The spleen was of about normal size and showed many flattened miliary cles. Beneath the capsule, on section, there were found numerous tubercles of varying the larger ones were yellow and the smaller ones gray.

Peritoneum.—There were found scattered all through the omentum, on the surface e root of the mesentery, over the bladder, and particularly on the under surface of the side of the diaphragm, numerous miliary tubercles. The lymph-glands of the mesen- were considerably enlarged, particularly beneath the stomach. On section they showed cles, most of which were quite large and had yellow, cheesy centres.

Intestine.—In the intestine about the ileo-cæcal valve there were several small ulcer- s apparently in the process of repair. In the cæcum there were two narrow ulcers t 1.5 cm. (⅝ inch) long. The bases were injected. The walls were not broken down.

Liver.—Many rather large tubercles were found beneath the capsule of the liver. y were flat, but not cheesy.

Brain.—The convolutions of the brain were flattened. There was marked fibrino- us exudation at the base of the brain, covering the optic commissures and the adjoining s. The third nerve was chiefly injected. Many small tubercles were present in the fis- s of Sylvius and over the convexities of the brain. In the right half of the cerebellum, beneath the pia, about the centre of the base, was a yellow nodule about 6 mm. (¼ inch) iameter. In the left lateral ventricle anterior to the velum interpositum was a similar ule about 3 mm. (⅛ inch) in diameter projecting into the ventricles. Both ventricles e moderately dilated by the serous fluid. The ependyma was everywhere granular: condition was due to small, gray, transparent tubercles. No tubercles were found in the l or fourth ventricles.

Kidneys.—The kidneys contained a few rather large grayish areas with here and there lowish speck.

The **pathological diagnosis** of the case was—

Old tubercular ulcers of the intestine,
Chronic tuberculosis of the mesenteric and bronchial lymph-glands,
Solitary tubercle of the brain,
Miliary tuberculosis of the pia, lateral ventricles, pleura, lung, spleen, kidney, peritoneum, and liver.

In connection with the other cases of tubercular meningitis which I e spoken of, I shall now mention some cases which represent the earlier iods of life, when, as I have told you, we are led to expect a variation in symptoms and a consequent difficulty in the diagnosis. The first two es represent tubercular meningitis as it so often appears when occurring infants under one year.

The first case was seen by me in consultation with Dr. Kimbal, of lem.

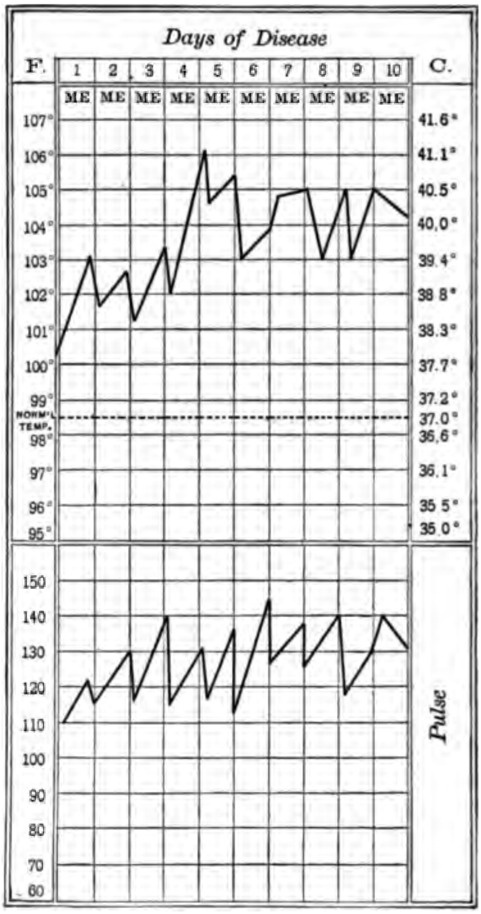
A male infant (Case 275), ten months old, had always been well and strong. For a days before I saw him he had been rather dull and feverish, but had shown no other ormal symptoms. He was evidently cutting some teeth at that time. On the day that w him, except that he was somewhat fretful and that he put his hands to his mouth as ugh his gums were disturbing him, he seemed very well, and careful physical examina- revealed nothing abnormal in the ear, throat, chest, or abdomen.

On the day following my visit the slight symptoms of indisposition which he had previously shown disappeared, and he played with a toy whistle, blowing it himself, and seeming to be very well. This condition lasted for two or three days, when he became stupid and unconscious, and about the tenth day from the time that I saw him he died in convulsions.

This case should impress upon you the difficulty of making a diagnosis in the early period of a tubercular meningitis, and how guarded we should be in giving a prognosis in young infants, even where the character of the disturbance is very slight.

The next case (Case 276) was the infant, eleven months old, whom I examined before you on the 18th of March.

CHART 28.



Tubercular meningitis. Male, 11 months old.

The history at that time was that he had always been well and strong, except that he had had bronchitis in December and that the cough had returned at intervals. He had at that time six teeth. There was a tubercular history on the mother's side of the family. He had remained well and thriving until you saw him here, when he seemed feverish and rather dull; there were anorexia and insomnia, and he was said to have become tired easily. The temperature had been rather high, 39.4° to 40° C. (103° to 104° F.), the pulse quick and regular, and the respirations rapid but regular. Nothing abnormal was found on physical examination, and, as the gums were swollen and hot, no diagnosis was given, and a guarded

›sis. He remained in this condition until March 17, when I noticed an apparent ıch to Cheyne-Stokes respiration and a little retraction of the head. There was no phobia, and the pupils were equal and reacted well. There were no vomiting and *he cérébrale.* The bowels were regular; the tendon reflexes were exaggerated, but ıg else abnormal was found in the lungs, heart, or abdomen. On the following day s found to have ptosis of the left eyelid. The fontanelle was depressed; the abdo- ras distended. He gradually grew worse, and died on March 22, about ten days after ınced symptoms of any disease had begun.

ere is the chart (Chart 23, page 624) of his temperature and pulse. The respirations the whole course of the disease varied from 80 to 100.

his next case, which I saw in consultation with Dr. Broderick, of ı Boston, represents tubercular meningitis as it appears in the second of life. You will notice how at this period it is rapidly approaching haracteristic symptoms of the disease which are met with from the to the seventh or eighth year, and even later.

boy (Case 277), fifteen months old, had always been healthy. His mother was ıy, but his father had died of tuberculosis. He had twelve teeth, and was cutting one canine teeth, the gum over which was swollen and tender. He had always had a ncy to constipation. He was perfectly well until he was fourteen and a half months hen he did not have a movement of the bowels for a week. He became fretful, and ds the end of the week his respiration was noticed to be of the Cheyne-Stokes type. ulse varied from 80 to 150 and was regular. The temperature in his axilla varied 37.4° to 38.1° C. (99.6° to 100.6° F.). At times there was rigidity of the hands, but were no regular convulsions. He vomited at the beginning of the attack, but not vards.

Vhen I saw him, in the second week of the disease, his eyes were rolling from side to nd there was much Meibomian secretion. He was unconscious, but he was said in the to have put his hand to his head and to have cried out as though he were in pain. e was some stiffness of the neck and back. There was a rather marked *tache cérébrale,* there was decided depression of the abdomen. The pupils were equally dilated and nded to light. There was considerable emaciation.

During the following week at one time for a few seconds he had decided opisthotonos. radually sank and died. The duration of the disease was four weeks.

LECTURE XXX.

BRAIN.—(Continued.)

THROMBOSIS OF THE CEREBRAL SINUSES.—HYDROCEPHALUS.

THROMBOSIS OF THE CEREBRAL SINUSES.—Thrombosis of the cerebral sinuses is a very uncommon condition. The disease is more frequent in infancy and early childhood than in adult life. It is caused by the formation of an ante-mortem clot in one of the sinuses of the brain. As a primary condition it is exceedingly rare. It is usually secondary to some condition which has produced a deep impression upon the child's vitality, such as profound anæmia, exhausting diarrhœa, or a collection of pus in any part of the body, but especially about the scalp, as in erysipelas. A purulent otorrhœa is perhaps the most common etiological factor. It is not necessary here to do more than refer to the traumatic cases of this disease, such as involve the ear and the scalp, as in fracture, or where the disease is caused by compression, as from a cerebral tumor. The pathology of the secondary cases includes the lesions of the different processes which have caused the thrombosis. That of the idiopathic or undetermined cases is well represented in these specimens which I am about to show you (Case 279). The thrombosis may take place in any of the cerebral sinuses, and at times may occur in the course of a meningitis. When the thrombus is formed, the venous branches behind the obstruction become distended mechanically, and thus give rise to capillary hemorrhage and softening of the floor of the ventricles. When the thrombosis has taken place in the neighborhood of some inflammatory focus, such as a purulent otitis media, pyæmia may result.

So few cases have been reported where the diagnosis has been established by a post-mortem examination, that the clinical description of the disease must necessarily be very limited. The symptoms which existed in cases where this condition has been found on post-mortem examination are not such as to suffice for making a differential diagnosis during life between this and other intra-cranial conditions, such as occur in profound anæmia. Where, however, convulsions occur in an atrophic child, especially if there has been chronic trouble in the ear, we can suspect the presence of this condition after carefully differentiating all other causes. Cases of thrombosis of the lateral sinus may be suspected where symptoms of a severe purulent affection follow a suppurative otitis, with involvement of the mastoid cells, and where there is a tenderness over the external jugular vein.

The prognosis in this disease is usually fatal, except where it occurs in the lateral sinus and can be relieved by operation. Pitt reports the recovery a boy (Case 278) ten years old who had chronic otorrhœa, followed by ute symptoms of fever and aural tenderness. Following these symptoms, week later, he had a rigor, and optic neuritis was developed on the right le. Exploration of the lateral sinus disclosed a clot, which was removed, d the boy recovered.

I will now show you the results of a post-mortem examination which s been made by Dr. Whitney on an infant nine weeks old.

This infant (Case 279) was seen by you with me in the wards of the Infants' Hospital o weeks ago, and at that time it was apparently well and strong. You saw it two days o unconscious and having an irregular type of convulsions.

When first seen by me, January 16, it was, so far as I could judge, strong and healthy, ighing 4805 grammes (about 10¼ pounds), which at six weeks is decidedly a greater ight than the average. The average weight of the male infant at birth, as I then told u, is about 3250 grammes (about 7 pounds 2⅗ ounces.) Allowing for a daily gain of 30 ammes (1 ounce), the weight of an infant six weeks old should be 4510 grammes (about pounds 14 ounces), so that this infant weighed 295 grammes (about 9⅝ ounces) more than e average infant of the same age.

On entering the hospital it took its food well, had two or three apparently well-digested jections daily, slept well, and seemed to thrive for the following week. No one would ve known from its outward appearance that anything was the matter with it if it had not en carefully weighed, when it was found that it was losing. The following is the record its weight from January 16 until its death, January 30 (Table 102):

TABLE 102.

Date.	Weight. Grammes.	Gain or Loss. Grammes.
January 16	4805	
January 18	4655	Loss, 150
January 20	4630	Loss, 25
January 21	4595	Loss, 35
January 22	4610	Gain, 15
January 23	4590	Loss, 20
January 24	4425	Loss, 65
January 25	4420	Loss, 5
January 26	4420	Loss, 0
January 27, 8.30 A.M.	4110	Loss, 310
January 27, 6 P.M.	3995	Loss, 115
January 27, 7 P.M.	3925	Loss, 70
January 28	3945	Gain, 20
January 29	3965	Gain, 20
January 30	3735	Loss, 230

On January 23 the loss of weight was very evident, and various changes were made in e infant's food, but with no good result, as he vomited and had thin watery discharges om the bowels.

On January 28 he seemed weak and did not look well. A wet-nurse was procured for m, but her milk did not agree with him, and in fact he became much exhausted when ying to nurse.

At 7 P.M. he was examined by Dr. Haven and myself, with the following result. His mperature was 38° C. (100.4° F.). His respirations were 35, and were natural. The pils were normal and reacted to light. The fontanelle was very slightly depressed. The ild did not seem to be in pain. Nothing abnormal was detected in the thorax, abdomen, throat.

On January 28 he vomited considerably during the day, and had a natural yellow fæcal dejection, but he would not take his food. His pupils were contracted equally, and he had rhythmical contractions of the arms and legs, first on one side and then on the other. Accompanying these movements was opisthotonos. The head and eyes were drawn to the right. There was no rigidity or paralysis of the legs or arms. The fontanelle was not depressed. There were rapid contractions of the eyelids, first on one side and then on the other.

On January 30 he had six rather watery fæcal movements. The muscular contractions ceased, but the opisthotonos continued until just before his death, which occurred at 6.30 P.M.

You see that the face is thin and pinched; the body is small and somewhat emaciated; there is slight rigor mortis; the calvaria is removed without difficulty. On close examination, nothing abnormal is noticed on the external surface of the dura mater. In the straight sinus and in the portion of the superior longitudinal sinus immediately adjoining this is a formed red clot, slightly decolorized in parts, but easily removed from the vessels, and evidently ante-mortem. The other sinuses contain a little loosely-clotted blood. The surface of the brain is moist, and the spaces between the convolutions are slightly opaque and cloudy from the presence of a serous fluid. The blood-vessels of the pia mater are injected. Upon opening the lateral ventricles and turning back the fornix the floor of the ventricles is seen to be covered with numerous thromboses of the blood-vessels and its surface to be universally reddened. The ependyma is roughened and infiltrated, and there is a bloody serous fluid in the cavity of the ventricles. The veins of the choroid plexus are filled with dark clotted blood which is directly continuous with that found in the straight sinus. The substance of the brain is moist. The spinal cord presents a moderate injection of the vessels of the pia mater. Both sides of the heart contain dark loose clots, and the heart itself is normal. The lungs are slightly œdematous. The other organs present nothing abnormal.

As a summary of the case we have an infant nine weeks old, apparently strong and well up to January 16, when it began to lose in weight. By January 23 it had lost over 200 grammes (6⅔ ounces) without showing any other symptom of disease. By January 27 it had lost 480 grammes (16 ounces). Two days later it was attacked with convulsions and died. The autopsy showed nothing abnormal except a capillary hemorrhage into the ventricles caused by a thrombosis of the straight cerebral sinus.

The pathological diagnosis in this case is that of a sinus-thrombosis of undetermined origin, a condition which is exceedingly rare, and instances of which established by autopsy have seldom been reported.

Tirard reports the case of a boy four years of age, which is of considerable interest and value as representing secondary sinus-thrombosis.

The child (Case 280) was well until he had measles; from that time he lost in weight and strength. Just previous to coming under medical observation he had several severe convulsions, had been stupid, and had not spoken to any one.

On examination he was found to be emaciated and to have a coated tongue; his teeth were covered with sordes; his bowels were constipated. He was semi-conscious occasionally, and had slight convulsions, in which the left arm was generally affected. There was no drawing of the face; the pupils were equal; there was no strabismus, no retraction of the head, no tenderness of the spine. The patellar and plantar reflexes were present, equal and normal. There was no anæsthesia nor analgesia; a *tache cérébrale* could be obtained. There was a purulent discharge from the left ear. The urine contained a trace of albumin.

In the next two days there were several convulsions and a rise of temperature, followed by a brief return of consciousness. Examination of the chest showed dulness and crepitation over the base of the left lung.

A week later the mouth was noticed to be drawn to the left. Trembling of the hand, resembling the oscillations of paralysis rather than the movements of chorea, then appeared. When the child was lying undisturbed these tremulous motions ceased, but they became ggerated when the limb was raised, and were then accompanied by tremulous move- nts of the face. Death occurred two weeks later.

The post-mortem examination showed thrombosis of the cerebral sinuses. There were merous small abscesses in the lungs, apparently from infarctions. The longitudinal and eral sinuses contained well-marked decolorized thrombi. In the latter they were soft; in former, firm. There was pus in the left tympanum and in the mastoid sinuses. There s no perforation of the membrana tympani, and no necrosis of the petrous bone.

HYDROCEPHALUS.—I shall next describe a disease of the brain hich is characterized primarily by an exudation of fluid into the mem- anes of the brain or one of its cavities.

For lack of a better name, we designate the disease by the term repre- nting the most prominent pathological condition,—namely, hydrocephalus ater in the head).

In order that you may clearly understand what I am about to say, I all ask you to refer again to this diagram (Diagram 8, page 594), show- g a section of the skull, the cerebral membranes, and the brain.

The general shape and circumference of the head in infancy and child- ood vary in the individual to a considerable degree. This has already een spoken of in a previous lecture (Division II., Lecture III., page 61), nd is merely referred to here for the purpose of illustration, because the kull and its contents have so close a connection in the mind of the student.

On the other hand, when these variations in size pass a certain limit, or re combined with certain nervous phenomena, they have a distinct patho- gical significance.

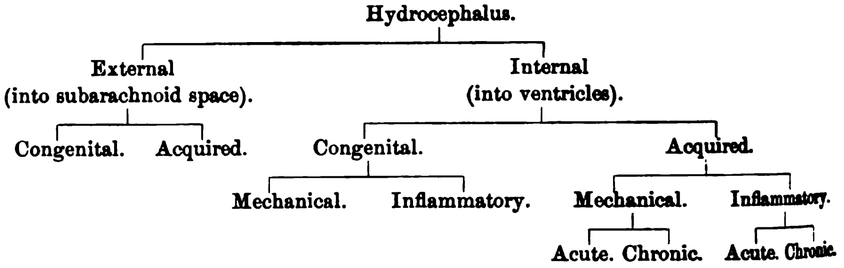
Hydrocephalus may be (1) external or (2) internal.

(1) The external variety consists in a transudation into the subarachnoid pace and the meshes of the pia, represented in this diagram by Sub. A. S. Diagram 8, page 594). This external variety is very rare, and may be ither congenital or acquired.

(2) The internal and common variety of hydrocephalus consists in a ransudation into the cerebral ventricles. It may be congenital (intra- terine) or acquired (extra-uterine), and its cause may be mechanical or nflammatory. Acquired internal hydrocephalus may be acute or chronic. The acute form of the disease usually occurs as a symptom or a cause of ymptoms in the course of such diseases as rhachitis, cardiac and renal isease, pertussis, and meningitis, and in various other diseases. It may be lso apparently idiopathic. The chronic form of acquired internal hydro- ephalus resembles so closely congenital internal hydrocephalus that we an consider them together, and, so far as the name of the disease is con- erned, the term hydrocephalus would be restricted best to (1) congenital nternal hydrocephalus and (2) chronic acquired internal hydrocephalus. n other words, there exists pathologically a certain class of effusions into he ventricles for which no cause is apparent. When these effusions reach certain amount the resulting symptoms are quite typical of what is called ydrocephalus, and clinically the term has therefore been confined to cases f this class.

This table (Table 103) will aid you in understanding the classification which I have just given you:

TABLE 103.



In addition to the effusion which takes place in either external or internal hydrocephalus, there may be a combination of both, as there is a communication between the fourth ventricle and the subarachnoid space by means of the foramen of Magendie.

(1) EXTERNAL HYDROCEPHALUS.—External hydrocephalus may occur as a *congenital* disease, but this occurrence is so rare that little can be said concerning it. I have met, however, with one instance of a somewhat analogous condition which I saw in consultation with Dr. Boughton, of Jamaica Plain, from whom I received the complete notes of the case. It seems very likely that this was a case of congenital cyst.

A female infant (Case 281) was born of a healthy primipara, the delivery being assisted by forceps. The infant appeared to be strong and vigorous and was not cyanotic. Its head was natural in shape and size, and there was no evidence of undue or prolonged instrumental pressure. Its weight was 3632 grammes (8 pounds). Nothing unnatural was noticed about the infant for several days, except that it did not nurse well. At these times it would cry and refuse to nurse. Its mother had a sufficient supply of good breast-milk.

When it was five days old it looked rather pale and thin, and on the sixth day, when the nurse was giving it its bath, she noticed that its right arm twitched convulsively several times. This twitching increased in frequency and force and was accompanied by a marked change in the infant's face. It became very pale and was cyanotic around the lips and eyes. At times it would cry out sharply both during the convulsive twitchings and in the intervals. Sometimes it would pass into a state of semi-collapse and would be cold and very pale. At this time also it would jerk its right arm convulsively at the rate of 75 times a minute. These spasmodic movements seemed to be confined to the right arm. There was no muscular contraction elsewhere, no frothing at the mouth, unconsciousness, or other evidence of general convulsions. Sometimes the respirations would be very faint and scarcely perceptible. The pulse would be weak, about 90 per minute, and then the infant would suddenly begin to breathe with great rapidity and the pulse would increase to 120. The area of cardiac dulness was not increased. There was a moderately loud, double cardiac souffle, most marked at the second left intercostal space, but heard all over the upper part of the sternum. In the right back, at the angle of the scapula, there was a patch of dulness about 2.5 cm. (1 inch) in diameter, but there were no râles over this area of dulness. The expansion of the lungs was irregular. The temperature was slightly subnormal.

The infant was wrapped in cotton-wool and surrounded with heaters. Brandy and aromatic spirit of ammonia were given in alternate doses of 0.3 c.c. (5 minims).

On the eighth day it was reported that the convulsive twitchings had continued, and

had been twenty-five spasmodic attacks within the previous twenty-four hours.
it was still in a state of collapse, the pulse and respirations were very weak and
and sometimes it would actually stop breathing for a minute. It was semi-
us. Its pupils were dilated. The abnormal signs found in the chest were
d.

he ninth day the convulsive movements had ceased, but the child was still in a
ollapse and remained perfectly dormant and passive.

ie tenth day the convulsive movements began again, and at times the infant ap-
eless. On this day oxygen was administered for five minutes every hour. The
as increased to 0.72 c.c. (12 minims). The infant had been too weak to nurse for
ys, and the mother's milk was given to it by means of a dropper.

ie eleventh day the oxygen was given for ten minutes at a time every hour, and
henever signs of unconsciousness appeared.

ng the next few days the infant began to show signs of improvement. The
uffle became less distinct. The atelectasis of the lung remained unchanged.
he time when the infant was so ill there was no disturbance of the bowels or

he seventeenth day modified milk was substituted for the breast-milk. From this
nfant rapidly improved. The cardiac souffle lasted for six weeks, and the atelec-
lually disappeared, the last signs of it being a little diminished respiration in the
k. The oxygen was continued in small doses for six weeks; 8400 litres (2000
were used. Of this, of course, a certain quantity was not inhaled, but escaped, as
l was held rather lightly over the infant's mouth. The infant had become con-
emaciated, but when it was four weeks old it had greatly improved, and, although
only 3405 grammes ($7\frac{1}{2}$ pounds), looked fairly well.

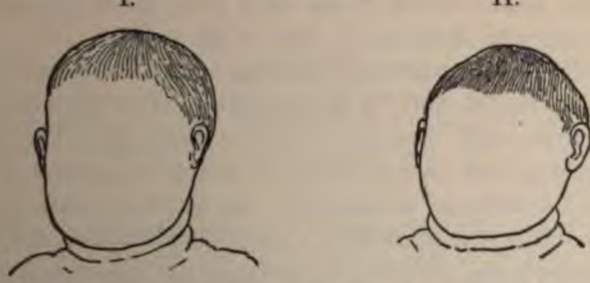
n the infant was a little more than four weeks old the head was perfectly normal in
l size. The fontanelle could be seen pulsating naturally and was normal in shape

ve here a tracing (Case 281, I.) from a photograph which was taken of the infant
ne.

he following day the head was found to be noticeably altered in shape, and this
acing (Case 281, II.) was made from a photograph which was taken when the

CASE 279.

I. II.



Congenital external hydrocephalus or congenital cyst.

somewhat older. The rounded, full forehead had disappeared, and, instead of look-
al, the child had the appearance of an idiot. The report of the nurse was that
was being dressed in the morning it had vomited some fluid like water, and that
had assumed this shape within the course of a few minutes. Both segments of the
d the parietal bones had flattened, and apparently had settled or collapsed. The
fontanelle had entirely closed, and the frontal suture could not be felt. A line
m the vertex to the root of the nose was entirely straight, instead of showing the
urve. The width of the forehead was also diminished. The entire frontal bone
t that it lay upon a lower plane than the parietal bones,—perhaps 1 cm. ($\frac{3}{8}$ inch)

tic atrophy can be found by means of the ophthalmoscope. In a consid-
ible proportion of cases of this kind there is a diminution of vision which
not explained by anything that the oculists tell us.

Henoch mentions a case of hydrocephalus in which the fluid drained
ough the nose to the amount of 100 to 200 c.c. (3½ to 6⅔ ounces) a day
quite a long time, so that the hydrocephalus was reduced considerably.

The *acquired* form of external hydrocephalus is exceedingly rare, and is
ually found in connection with cerebral atrophy (hydrocephalus ex vacuo).
ere are certain cases which can for the present be classed under this head-
until our knowledge derived from post-mortem examinations becomes
re precise. These cases are so rare that it is impossible at present to for-
late in detail their symptomatology and diagnosis. I have met with a few
es, however, which in their symptoms were so significant of a rapid devel-
nent, with its speedily fatal issue, of an external hydrocephalus, that the
gnosis of hydrocephalus by the elimination of other possible causes has
med to me rational, and has been supported by the post-mortem exami-
ion. The symptoms may develop, according to my experience, in young
nts who either have been fairly well or have been atrophic. Phys-
examination in these cases has revealed nothing abnormal about the
d or any of the organs, such as the heart. The infant, after a short
iod of indefinite symptoms, at times lasting only a few minutes, and
resented by nervous twitching, perhaps a convulsion and rapid collapse,
denly dies. I have met with three cases in my personal practice. Two
e, after minute post-mortem examination by Dr. William F. Whitney,
nd to represent as their only pathological lesion external hydrocephalus
h œdema of the cerebral substance. The third case showed this condition
rely as a symptom of pernicious anæmia, and I have spoken of it in a
vious lecture.

One of these cases was an infant (Case 282), ten months old, of healthy parentage, and
ays perfectly well, except that for two weeks before its death it had cried more than
l and was somewhat irritable. Five days before its death it was somewhat languid,
took its food well, and when I examined it the night before its death nothing abnor-
was found. On the following morning it had a few convulsive movements and died
denly.

On post-mortem examination nothing abnormal was found, except that a large amount
erebro-spinal fluid escaped from the cranium as soon as the skull and parietal dura
er were removed. There was also a general œdematous condition of the brain.

The other case was a female infant (Case 283), seven and one-half months old, which
been suffering from malnutrition for several months and was very weak and puny.
the day of its death I examined it carefully, and, with the exception of an atrophied
dition of the muscles and a weak action of the heart, nothing abnormal was discovered.
thin an hour after I had seen the infant it had a few convulsive attacks and died sud-
ly.

The autopsy, made twenty-four hours after death, showed nothing abnormal externally.
or mortis was present. There was great pallor of all the organs. The skull was normal
evelopment, and the fontanelle was normal. There was cerebro-spinal fluid in excess.
brain-substance was very moist and pale, but otherwise nothing abnormal was noticed
he brain or meninges. The heart was of normal size, and its cavities and valves were

normal. The ductus arteriosus and Eustachian valve were closed. The mus
on the right side of the heart was pale and opaque, while that of the papill
the left side was pale but not opaque. Microscopic examination showed the
stance to be filled with minute, highly refracting granules, which in pa
the addition of acetic acid, but were left undissolved in some places, wher
of the fibre was destroyed. On the left side of the heart granules were
could be wholly dissolved in the acetic acid. There was fatty degeneratio
marked of the cardiac muscles and also of the diaphragm, the fibres of
numerous granules, which dissolved in acetic acid in about one fibre to fifty.
were pale, but otherwise nothing abnormal, either microscopically or macro
found. The supra-renal capsules were normal. The liver was of normal size,
its surface was found to be dry, yellowish, and opaque. Microscopic exami
that the liver-cells were filled with fat-drops of varying size, especially nu
cells of the periphery of the lobule. The cells themselves had a sharp outl
defined nucleus. The pancreas was normal. The stomach was normal in
tained considerable milk, with but few curds. Nothing abnormal was de
microscopic examination. The small intestine contained a very little soft, yell
The large intestine contained a small amount of yellowish, soft fæces. No
Peyer's patches or of the solitary follicles was found. The mucous coat was

(2) INTERNAL HYDROCEPHALUS.—Internal hydrocepha
congenital or acquired. The earlier the hydrocephalic conditio
larger will the cranium become. We therefore find the very la
a rule, to be of the congenital variety. The head is at times o
as to cause difficulty in the delivery, or the fluid may collect
after birth, and the head soon assumes the characteristic ap
hydrocephalus.

This skull of a child three years old (Fig. 92) is an exag
of the congenital internal hydrocephalic head.

This other skull of a child, also three years old (Fig. 9
place beside the hydrocephalic skull, represents a normal head
age.

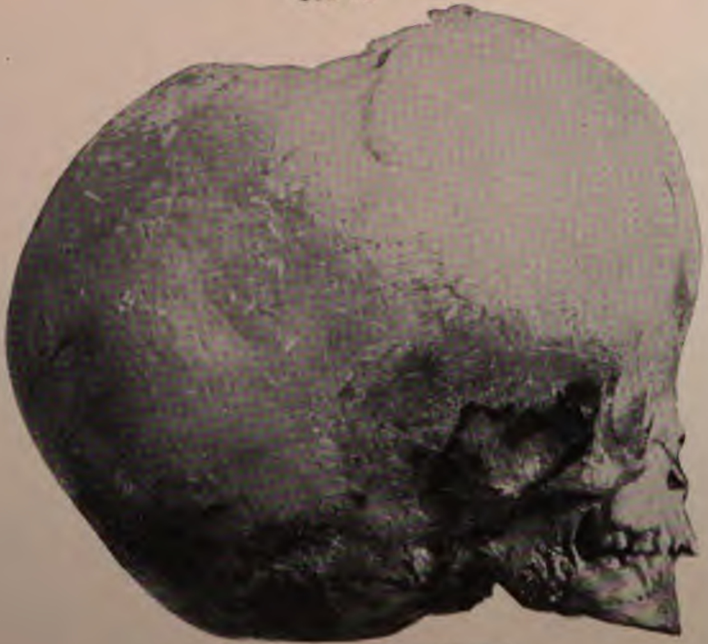
The face in these cases of hydrocephalus remains about the
it would be normally, but usually looks much smaller from th
tionate size of the head, which rests upon it from above like a g

Congenital Internal Hydrocephalus.—The cause of co
ternal hydrocephalus is somewhat obscure. In some cases it i
matory origin, in others no evidence of inflammation can be fou

PATHOLOGY.—The anatomical appearance of the brain itse
corresponds with and may be accepted as the result of pressure
ventricular fluid. This brain (Fig. 94, page 635) was taken fi
who died of congenital internal hydrocephalus, and well exe
pathology of the disease.

You see that the convolutions are flattened and that the
ventricles are much thinned by the intra-ventricular pressure, wl
tricles themselves are much dilated. In some parts the cortex is
cm. ($\frac{3}{8}$ inch) in thickness. The amount of fluid in these cases v
few cubic centimetres to three or four litres. The fluid has a spe
of about 1004.

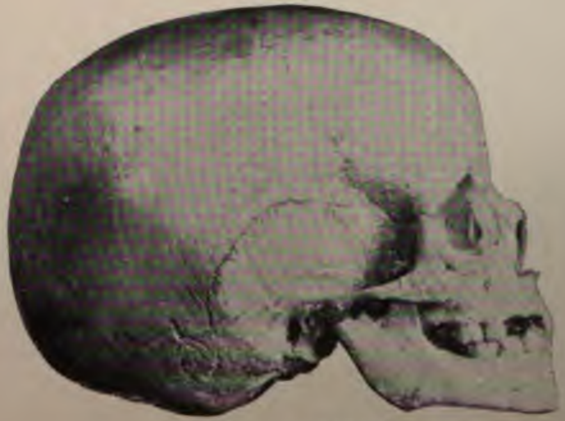
FIG. 92.



Hydrocephalic skull, child 3 years.

Warren Museum, Harvard University.

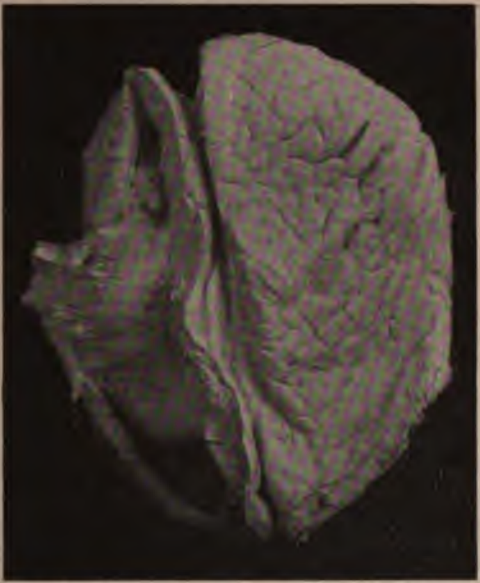
FIG. 93.



Normal skull, child 3 years.

IPTOMS.—The symptoms of congenital internal hydrocephalus are lly those caused by pressure. We naturally, therefore, find the lles bulging and fluctuating, and the bones thin and forced out of . As you see in this skull (Fig. 92), the temporal and parietal liverge as they extend upward, while in the normal skull they almost perpendicularly. If the disease has existed for some time, er wall of the orbit becomes flat and the eyeballs protrude. The

FIG. 94.



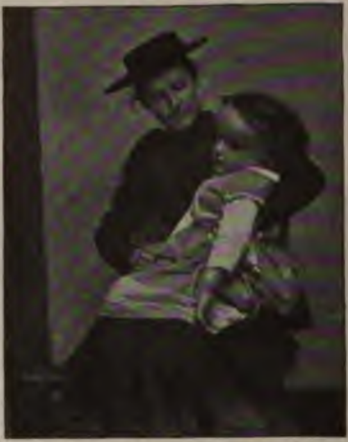
Hydrocephalic brain. Warren Museum. Harvard University.

ebral pressure often produces a strong collateral circulation in the d the forehead, where the vessels appear like tortuous blue cords. nal disturbances are numerous, and vary in almost every case. As he children are idiotic, but at times, even in marked hydrocephalus, the mental condition normal, even when paralysis is present. A instance of this fact is presented by this little girl whom I have had here to-day to show you.

child (Case 284, page 636) is five years old.

see that, although she has completely lost the power of using her legs, and has ead and distended, bulging fontanelle, yet she is bright and intelligent. She ed by her mother for over a year, and cut her first tooth when she was six months was always well and strong, but high-tempered. She has never had any disease. was five months old she fell from her crib and struck her head, but it did not urt her especially. Her head was always noticed to be of a peculiar shape. was eight months old she fell out of a chair and was stunned, but was not other-. She has had convulsions from time to time, but her mind has always been he was unable to hold up her head until she was three years old, and has always ed of more or less frontal headache. Her appetite has always been excessive, and her taste for food somewhat peculiar. She has gradually grown stronger, and is beginning to attempt to walk. She sleeps well, and her bowels are regular. Her head measures 57 cm. (22½ inches).

CASE 284.



Congenital internal hydrocephalus. Female, 5 years old.

As the various cerebral centres become affected by pressure, we notice that symptoms arise corresponding to the parts of the brain which are affected. Among these symptoms are nystagmus and, less frequently, strabismus. The pupils at first are usually moderately dilated. Later they become fixed, and sensibility to light is lost. The hearing lasts for a long time. The ability to walk is interfered with. Partial or general convulsions, paralysis (usually paraplegic), and contractures may occur. Pain in the head is often complained of, but, as a rule, is not so severe as in meningitis. There is difficulty in keeping the head erect, as it is so heavy. The digestion is often good, and the appetite usually extreme. The respiration is normal from adaptation. The pulse is usually not retarded. The temperature, as a rule, is normal. The adipose tissue is often abnormally increased.

DIAGNOSIS.—As congenital internal hydrocephalus is almost invariably attended by enlargement of the head and separation of the sutures, the diagnosis is not especially difficult, and is determined by comparing the measurements of the head with those of a normal head of the same age. I have described the measurements of a normal head in a previous lecture (Lecture III., page 61). In addition to the enlargement of the head, the symptoms of direct intra-cephalic pressure make the diagnosis very simple.

PROGNOSIS.—These congenital cases, as a rule, die before childhood has been reached, but they have been known to live to middle age. Death usually occurs from some intercurrent affection. Complete recovery is very rare.

TREATMENT.—The treatment of congenital internal hydrocephalus has been varied, but without marked success. When the effusion is not large and is not increasing, moderate pressure with a rubber bandage seems to have a favorable result. Where the disease is apparently not in an active state and is characterized by a very slight increase of fluid, aspiration hrough the anterior fontanelle of a small quantity of fluid at a time has een of temporary benefit. The point of aspiration should be 2 to 3 cm. ⅝ inch to 1¼ inches) from the median line, so as to avoid puncturing the lonitudinal sinus. In this way the condition of the patient is often rendered nore satisfactory.

An operation for chronic hydrocephalus presents no technical difficulties.)f course only certain cases are suitable for operation. Moderate effusions hould be let alone, also those cases where a rudimentary development of ne brain is suspected. Cases where an operation is especially indicated re comparatively both physically and mentally well developed up to the me when the enlargement of the cranium began. Such children should how the symptoms of direct intra-cranial pressure. They are evidently beoming weak-minded or idiotic. They do not learn to talk, or they quickly orget what they have learned. They may also have become totally blind. 'he power of walking is interfered with. Contractions and partial and eneral spasms are of ordinary occurrence. Unless the pressure is speedily emoved, atrophy of the brain results, and if they live they remain idiots or life. Such cases as these you can best refer to those who are skilled n neurology and surgery.

I have in my wards to-day a number of cases of congenital internal ydrocephalus to show to you, which are of considerable interest in view f what I have just told you.

Of these illustrative cases I will first show you this infant (Case 285), two years old, hich is sitting in its mother's lap.

The special point of interest in this case is that the circumference of the mother's head nd that of the child's head are almost identical, 52 cm. (20½ inches). You will notice the verhanging brow and deep-set eyes, the globe-shaped head and open bulging fontanelles, he small face and oblique parietal bones of the infant's head in comparison with the normal, round shape of the mother's head.

The history, so far as the mother is concerned, is negative. She has had no miscariages. The infant was born at term, and cut its first tooth when it was six months old. It now has sixteen teeth. It has had no convulsions. It has for some time supported its head alone and sits alone, but has never attempted to walk. The intelligence seems normal. It is apt to sleep with its eyelids partly open. Its digestion is good, and its appetite is very good. The bowels are rather relaxed. On examining the head you will see that it is abnormally large. From the root of the nose to the occipital prominence it measures 32 cm. (12½ inches). From the base of one mastoid to that of the other it measures 33.5 cm. (13¼ inches). The position and movements of the eyes are normal. You will notice, on ooking at the head from above, that it is triangular in shape, with the base of the triangle at the occiput. The anterior fontanelle you see is widely open, and is about 4 cm. (1¼ nches) in width and length. The protruding overhanging forehead makes the face look mall. The epiphyses are not enlarged. Examination of the lungs, heart, and spleen hows nothing abnormal. The child weighs 10,442 grammes (23 pounds).

In this next bed is a boy (Case 286, page 638), three and a half years old, whose head s typical of congenital internal hydrocephalus. The circumference of the head is 57 cm. 22½ inches).

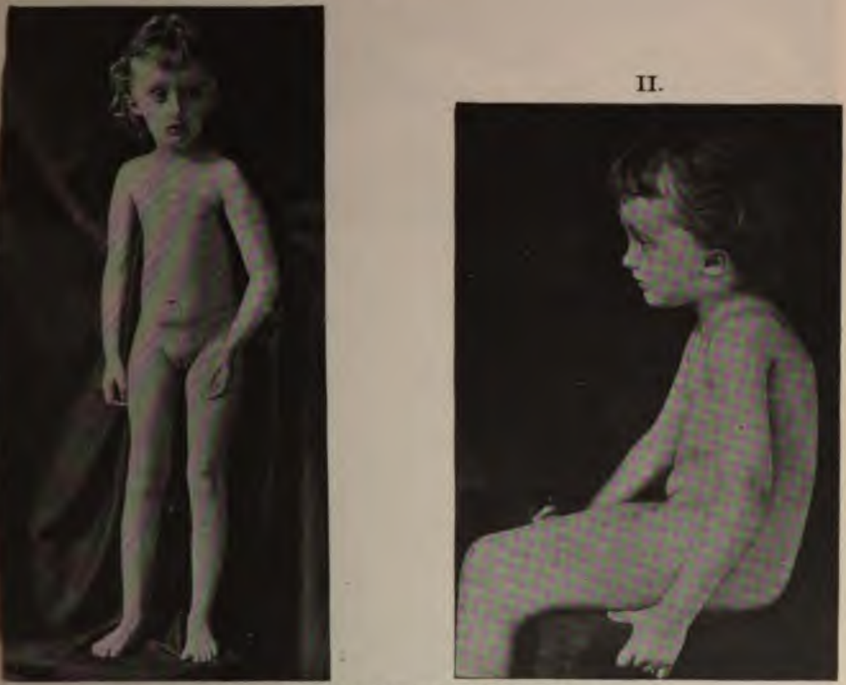
There is no history of disease in the parents, and the mother has had no miscarriages. The child's head has always been large since birth. He has never had any convulsions. He cut two teeth when he was four months old, and when he was a year old he had ten eeth. He walked when he was fourteen months old, but his legs never seemed strong.

the vertex. The superficial veins of the head are prominent. The face, as in the other child (Case 285), is small. The movements of the eyes are normal. From the tip of one mastoid process to that of the other is 41.5 cm. (16¼ inches). From the base of the nose to the occipital protuberance is also 41.5 cm. (16¼ inches). The anterior fontanelle is widely open and is 2.5 cm. (1 inch) long and 2.5 cm. (1 inch) wide. An examination of the heart, lungs, liver, and spleen shows that they are normal. The abdomen is prominent. The radial epiphyses are enlarged, and there is a slight outward bowing of each tibia. The spine is straight. The patellar reflexes are not increased, and there is no ankle-clonus. The urine is pale, thin, and clear, and contains no albumin. An examination of the eyes by Dr. Davis shows no marked diminution of vision in either eye. They are hypermetropic, and there is a convergent strabismus, probably accommodative. The optic disks are rather wider than usual, and their vessels diminished in size. There are no other signs of optic atrophy. There is no dilatation of the retinal veins or swelling of the disks. The examination, therefore, shows that, with the exception of an early stage of atrophy of the optic nerve from pressure, the fundus oculi is negative.

In this case a chronic congenital effusion was apparently actively increased by a blow on the head.

This little boy (Case 287, page 638), a patient of Dr. Haven's, is an interesting case of hydrocephalus, with its accompanying disturbance of the motor function of the legs, and also mental impairment. He is a characteristic picture of the disease. He cannot walk,

Case 288.


I.
II.

Internal hydrocephalus (probably congenital). Female, 6 years old.

ut is able to sit in a chair. His legs are atrophic, his abdomen is distended, and he is somewhat emaciated. His head, as you see, is decidedly enlarged, and he is mentally weak. His appetite is excessive. He is very fretful and peevish.

Cases of this kind are very apt to live for only a few years, and are especially liable to die if they are attacked by any intercurrent disease, such as pertussis.

This little girl (Case 288) is six years old.

She is said to have been normally developed and healthy at birth, but was unable to hold her head up until she was two years old. She has never walked.

You notice on looking at the head, both in front and in profile, that it is abnormally large. It measures 65 cm. (26 inches). The movements of the hands and arms are normal. She cannot stand unless she is supported, and there is a spastic condition of the legs, with an exaggeration of the knee-jerks. She articulates well.

She represents a case of partial recovery from chronic hydrocephalus, probably of the congenital variety. Her general development will probably always be interfered with.

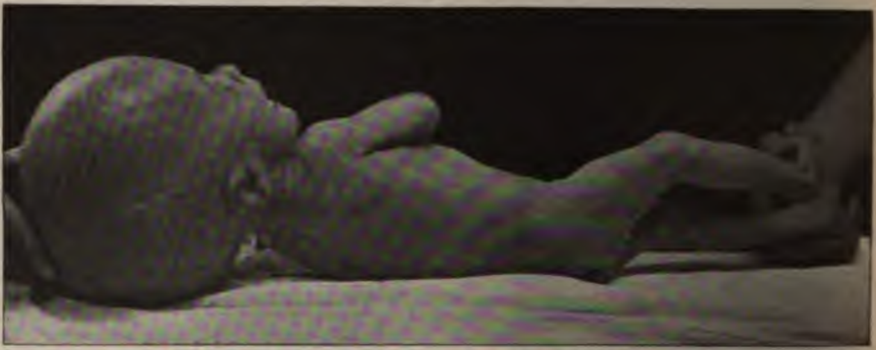
I shall now ask you to come to the operating-room and see some cases of chronic congenital internal hydrocephalus which Dr. Lovett is about to operate upon.

This first infant (Case 289, I.) is six months old.

It was noticed when the child was one week old that its head was beginning to increase in size. When two months old the circumference of the head is said to have been 41.5 cm. (16½ inches). Somewhat later the circumference of the head was 44 cm. (17¼ inches), and when it was three months old the circumference was 45.5 cm. (18 inches). When it was five months old the circumference was 55 cm. (21¾ inches). To-day, as you see, it measures 57.5 cm. (22¾ inches). There is no history of syphilis or of tuberculosis in the family. The infant has had no marked convulsions, although some twitchings of the hands and feet have been noticed. There has been constant nystagmus, and the infant's general condition is atrophic. You will notice the marked prominence of the eyes, and the great distention of the head. The anterior fontanelle is very large, and the skin covering it is distended to such a degree that it is thin and glistening. There is no doubt in a case of this kind that aspiration of the cerebro-spinal fluid should be made for the purpose of relieving the general condition.

CASE 289.

I.



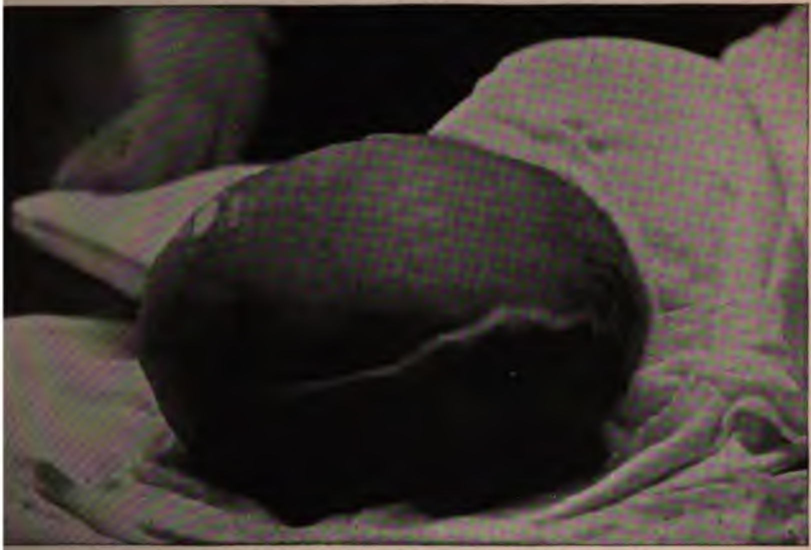
Congenital internal hydrocephalus. Male, 6 months old.

Dr. Lovett, as you see, has just made an exploratory puncture at the vertex of the right side of the head, about 5 cm. (2 inches) from the median line. In place of the small trocar he now introduces a larger one. Through this large trocar he has passed several strands of silk to serve as a drainage by capillary attraction. You will now notice (Case 289, II.) after withdrawing the fluid from the right ventricle that the right parietal bone has sunk in, its edge being beneath that of the left parietal bone, which is still pushed outward by the fluid in the left ventricle. On measuring, a considerable quantity of fluid is found to have been aspirated.

You see that, although the head is very much reduced in size, there are no symptoms of collapse nor any other alarming symptoms shown by the infant.

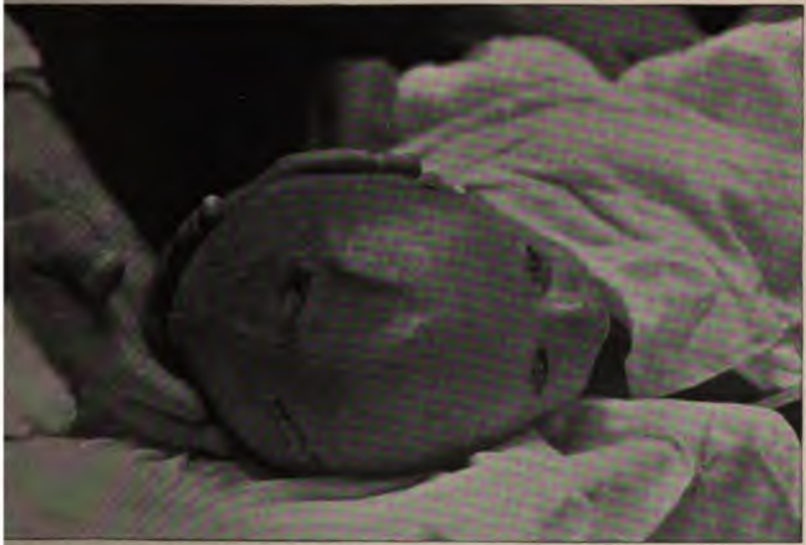
Case 289.

II.



Congenital internal hydrocephalus after aspiration of right ventricle.

III.



Congenital internal hydrocephalus after aspiration of both ventricles.

Subsequent history.) The infant was very restless during the following night, tossing ıd about and crying.

n the next day 270 c.c. (9 ounces) of clear fluid were withdrawn from the left ven- by introducing the trocar at a point corresponding to the point of aspiration of the ventricle. The head was then found to measure 55.5 cm. (22 inches).

No. 8 soft catheter was then introduced into each ventricle and sewed into place. xternal end of each catheter was closed by bending it upon itself and tying it tightly silk ligature.

he appearance of the cranium after the second aspiration is here shown (Case 289, You will notice the great depression of the anterior fontanelle.

hree days later 120 c.c. (4 ounces) of fluid were drawn through the catheter.

n the following day 138 c.c. (4⅗ ounces) of fluid were withdrawn, and the infant was to have a better facial expression.

n the next day, the fifth after the operation, 105 c.c. (3½ ounces) of fluid were ed, and the head was found to measure 51 cm. (20⅛ inches).

n the following day Dr. Dane began a series of observations on the fluid-pressure in se, which were the first of the kind that have been brought to my notice. He con- the catheter with a manometer and found a positive pressure of 7 cm. When the cried the pressure rose to 12 cm. On this day 120 c.c. (4 ounces) of fluid were ed.

n the following day the pressure was found to be 4 cm., and rose to 5 cm. when the cried. 90 c.c. (3 ounces) were removed.

n the following day the pressure was the same. 68 c.c. (2¼ ounces) of fluid were ed on this day, and the head was found to measure 49.5 cm. (19⅝ inches).

n the following day the infant failed rapidly, had convulsions, became unconscious, ied in the evening.

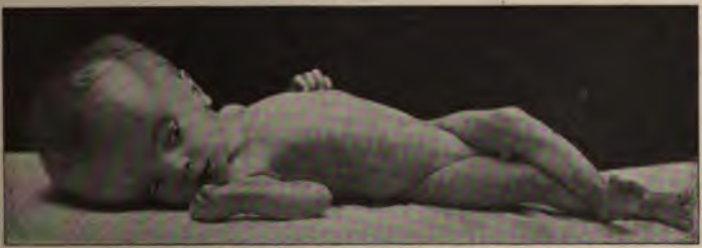
fter death 556 c.c. (18½ ounces) of cerebro-spinal fluid were removed. The specific y of this fluid was 1003. It contained 1⅞ grammes (28 grains) of albumin to the This was measured by an Esbach's albuminimeter.

he total amount of fluid withdrawn from the ventricles in this case was 720 c.c. (24 s) in seven tappings.

he next case is that of an infant (Case 290), seven months old, who was admitted to spital to-day.

t has always been nursed. When it was two days old it had convulsions. Three later it had bronchitis, and accompanying this disease a return of the convulsions,

Case 290.



Congenital internal hydrocephalus. Male, 7 months old.

h occurred as often as six or seven times in the day. They were localized in the left arm left leg. These convulsions lasted for three weeks, gradually growing less severe. e was at this time a certain amount of intestinal disturbance, which, however, has now peared. There was also a history of a purulent discharge from the ears before the t was admitted to the hospital. It cried out sharply at night. The measurements of ead are 56.5 cm. (22⅜ inches) in circumference, and 36.7 cm. (14½ inches) from ear to ver the vertex. The anterior fontanelle is bulging. The eyes, as you see, are markedly gent and protrude from the orbits. If you will observe the eyes closely you will see that there is at times a slight trembling and twitching. No other spasmodic movements are noticed. The chest measures 33.5 cm. (13¼ inches) in circumference.

You see that the child as it now lies on the operating-table takes no notice of anything. It has been decided to relieve the cerebral symptoms by aspiration on account of the great increase in the intraventricular fluid shown by symptoms of increased intra-cranial pressure.

As you see, Dr. Lovett has introduced a thoroughly aseptic aspirating needle into the right lateral ventricle through the much dilated anterior fontanelle. The aspirating needle is connected with a water manometer, which shows a pressure of 30 cm. Having determined the pressure by means of this water manometer, we can now remove a certain amount of the fluid. In order to do this, a whiff of ether is given to the infant, and you see that Dr. Lovett introduces a trocar in place of the aspirating needle. He then withdraws the trocar, leaving the canula in the cavity. Next, as you see, he introduces a No. 7 soft rubber catheter through the canula, and on withdrawing the latter the end of the catheter is left in the ventricle. 130 c.c. (4⅓ ounces) of clear fluid have been removed from the ventricle. The specific gravity of this fluid is 1006. You perceive that the axes of the eyes, which before the operation were divergent, are now parallel. The external end of the catheter is now closed in the same manner as you saw it done in the preceding case.

(Subsequent history.) The observations on the intra-cranial pressure in this case, as in the last (Case 289), were made by Dr. John Dane. On the day following the operation the pressure was found to be 14 cm. by the water manometer. When the child cried it was increased to 20 cm. 25 c.c. (⅝ ounce) were removed on this day, and the circumference of the head was then found to be 35 cm. (13⅞ inches). The specific gravity of this fluid was 1007.

On the second day after the operation the pressure was found to be the same. At that time 55 c.c. (1⅝ ounces) of fluid were removed, the specific gravity of which was found to be 1006.

On the third day after the operation the tube was found to have leaked a little, and there was a slight convulsion in the morning. 50 c.c. (1⅔ ounces) of fluid were removed.

On the following day the tube was found to be leaking freely, and the infant was in a state of collapse and refused to nurse. The head measured 52 cm. (20½ inches).

On the next day there was still some leakage around the tube, but the infant was in a better condition.

Three days later, the leakage around the tube having been controlled in the mean time, the infant seemed better, but it had a thick purulent discharge from both ears.

During the next few days the child began to grow weak, and there was again a slight leakage around the tube.

On the eleventh day following the operation the child died quietly, no convulsive symptoms having appeared.

There was no complete post-mortem examination, but the distended ventricles were found to contain 759 c.c. (25⅓ ounces) of clear straw-colored fluid,—the left ventricle containing 409 c.c. (13⅔ ounces) and the right 350 c.c. (11⅔ ounces). An examination of this fluid by Dr. J. H. Wright showed that it was turbid with a flaky sediment. It was slightly alkaline. The specific gravity was 1009. It contained about 0.1 per cent. of albumin. No sugar was found. Under the microscope nothing was seen resembling the lining cells of the ventricles. An inoculation of a guinea-pig with this fluid to determine whether it was of a tubercular nature or not gave negative results.

Acquired Internal Hydrocephalus.—Both the acquired and the congenital form of internal hydrocephalus may be of mechanical or inflammatory origin, but the acquired form shows evidence of an inflammatory condition oftener than does the congenital form, and occurs very frequently in connection with rhachitis. Acquired internal hydrocephalus may be acute or chronic. In its acute form it may occur at any age as a symptom of any of a number of diseases, such as meningitis, one of the exanthemata, tussis, and rhachitis. It may in any of these forms become chronic. disease may sometimes appear to be idiopathic.

The chronic form of acquired internal hydrocephalus occurs usually in first four years of life, and is represented pathologically by a small ount of intra-ventricular fluid, perhaps 100 or 200 c.c. (3⅓ to 6⅔ ounces). is this chronic form of acquired internal hydrocephalus that can best be sified under the name of hydrocephalus with the congenital internal lrocephalic cases which I have just shown you.

SYMPTOMS.—The symptoms of the acute form of acquired internal lrocephalus are so closely connected with the diseases in which it occurs a symptom that it is not necessary to speak of them here.

The symptoms of chronic acquired internal hydrocephalus are very ch the same as those of the congenital form. The firmer the union the bones the less likely is enlargement of the head to occur.

PROGNOSIS.—The prognosis as regards life is serious. Of those who over, many are left either with some mental defect or with permanent ndness, the latter the result of optic atrophy. Complete recovery may ur, but is exceedingly rare.

DIAGNOSIS.—The diagnosis of chronic acquired internal hydrocephalus the idiopathic form is in its earlier manifestations chiefly made by the nination of other cerebral diseases, though after the stage of inflamma-y irritation has passed and the symptoms of pressure have become estab-ied, a provisional diagnosis can usually be made. I say provisional ause the disease is rare, and a sufficient number of autopsies have not been made to justify a decided diagnosis such as can be made in the genital form of the disease.

TREATMENT.—The treatment is purely symptomatic in cases where the ures and fontanelles have completely closed, except where it is advisable perform craniectomy. Where they have not closed, the treatment is the ne as in the congenital form,—that is, usually operative.

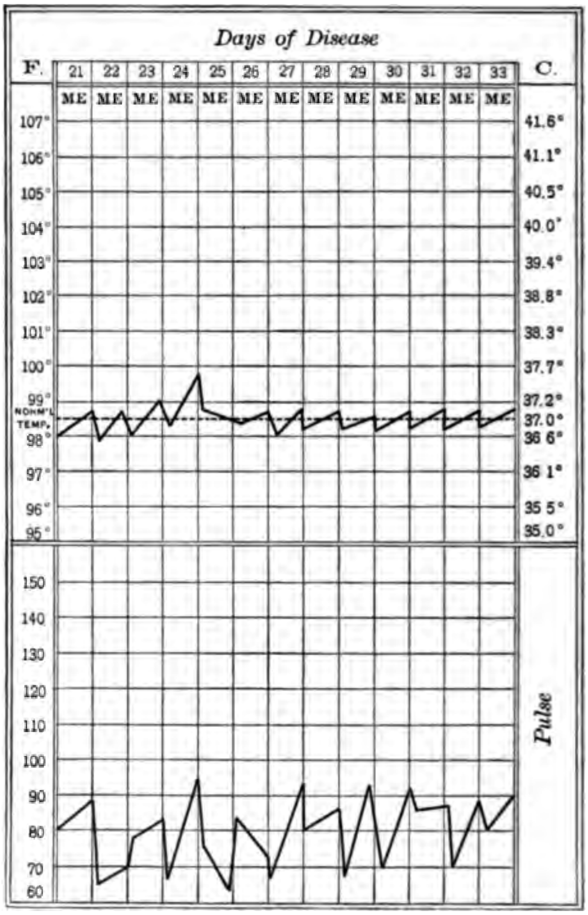
I have here three cases which I feel justified in reporting to you as bably representing chronic acquired internal hydrocephalus. Of course these cases we must allow that a tubercular or syphilitic taint may have n the starting-point of the intra-ventricular disease.

A boy (Case 291), four years and eight months old, was seen by me in consultation with E. J. Forster, May 27, 1885. The child's parents were healthy; his mother had other thy children and had had no miscarriages. The child had always been well, measles g the only disease which he had ever had. At the age of six months, while in the ess of cutting a tooth, he had three convulsions, from which he recovered entirely. appetite had always been capricious, but his digestion was good. His bowels had ays been regular. He had lately come from a malarial region, where he had lived in ther damp dwelling for a year.

On May 6 he vomited twelve or thirteen times. The vomiting then stopped, but rned later from time to time. He complained of pain in his stomach, had no fever, and etimes appeared to feel chilly. His bowels were constipated, and in the beginning of the attack his pulse was slow. He had been subject to night-terrors for some time previous to this sickness. The vomiting had lessened by May 27, and the report of my examination on that date is as follows:

Pulse 60, rhythmical; respirations regular; temperature normal; has had earache lately; no discharge from the ear since he was an infant; the examination of the ear was negative; yesterday morning he had a general clonic convulsion lasting for some time; his tongue is slightly coated; he lies in an apathetic state, though perfectly conscious; he is losing in weight and strength and has lost his appetite; urine normal. Nothing abnormal is found on examination of the thorax or abdomen. (The examination of the eyes, June 15, by Professor O. F. Wadsworth, showed that there was much swelling of the optic nerve, increased prominence of the retinal vessels, hemorrhages, and neuritis.) The child seems much brighter, and plays about. His intelligence is perfectly good; he has had no more convulsions and no paralysis, and seems perfectly well, except that his pupils are dilated and he is totally blind. The accompanying chart (Chart 24) records his temperature and pulse from May 27 to June 9.

CHART 24.



Chronic acquired internal hydrocephalus.

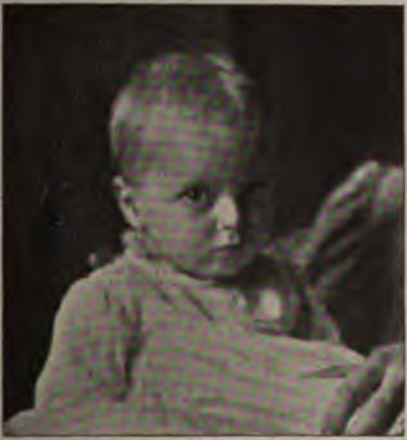
This child came to see me in May, 1893, when he was twelve years old. He had been and was at that time perfectly well. He was a bright, well-developed, healthy boy, twelve years old. His pupils reacted, but he had never recovered his sight. He weighed 19.8

(90 pounds). His bowels were regular; his appetite was good. His knee- increased. His head measured 49 cm. (19½ inches).

w call your attention to this little girl (Case 292), ten years old, who has been l for about two months. Her family history is negative, with the exception ternal aunts died of phthisis. The child has never had any disease except asles, and varicella. Her present trouble began one and a half years ago with of loss of consciousness without convulsions, lasting half an hour, after which asleep for some time. Eighteen months ago these attacks began to be accom- vulsions, which usually came about once a month, the intervals sometimes four months. The duration of the convulsions and the following sleep were e as in the earlier attacks. These convulsions have now not occurred for six the exception of one slight attack five weeks ago, when the right eye twitched a momentary loss of consciousness. The convulsions began with twitching ye, followed by twitching of the right hand. The rest of the body was not attacks were ushered in by intense headache, sudden vomiting, fever, flushed action of the head. The headaches, which began about one year ago, were ere and caused her to scream with pain. They were felt all over the head, in the region of the occiput; they would last an hour or more, until she then fell asleep. They came about every day, but were not always accom- niting. The headaches ceased altogether for a time, but she has had two or past five weeks. She has lately complained of dimness of vision. Six weeks pains in her right hand between the fore and middle fingers and began to f her hand. Physical examination reveals, as you see, nothing definite. She ard in using her right hand than her left, but all motions, you will perceive, nd strong. Her right foot seems to drag a little and is a little weak after run- e symptoms are not especially marked. The sensation of the hand is normal; is somewhat increased. For the past six weeks she has shown evidence lysis. Her pupils at times have been widely dilated. Dr. Dixon reports an th disks, with slight myopia.

Case 293.

I.



Acquired internal hydrocephalus. Protrusion of eyes.

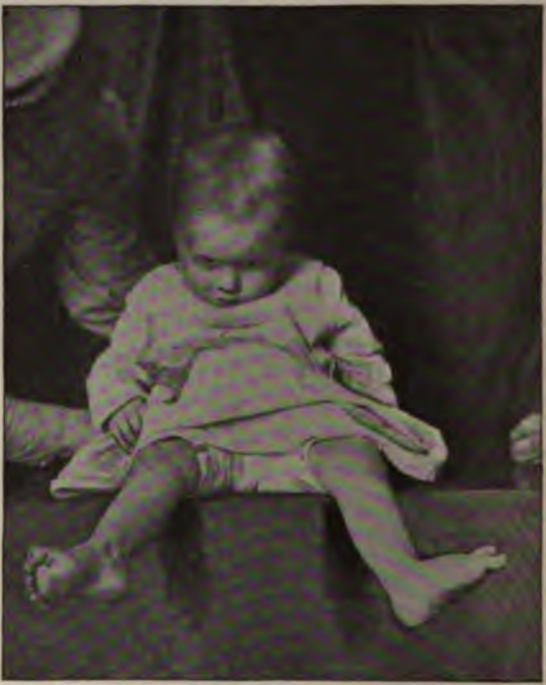
been sleeping poorly, and has had a fair appetite; the temperature has been o 38.3° C. (100° to 101° F.); the pulse 96 and regular; the respirations 24 She was treated with a good general diet and 0.30 gramme (5 grains) of tassium three times a day. The bromide was omitted one month ago. Lately d to be much better, and, as you see, she is now looking very well.

e boy (Case 293, I.) is two and a half years old.

He is said to have been well and strong at birth, and never to have been sick until two months ago, when he woke up screaming in the night, and this was followed by convulsions. For two weeks he did not recognize any one, cried out at times, and micturition and defecation took place unconsciously. In the early days of the attack he lay immovable. After consciousness returned he improved for five or six weeks, and no other especial abnormal condition developed. Two weeks ago he was attacked with convulsions, occurring at intervals of from thirty-six to forty-eight hours and lasting from one to one and a half hours. These attacks were ushered in by crying, which was followed by loss of consciousness, opisthotonos, kicking, and finally clonic convulsions. His mother states that during the early weeks of the disease he shrieked at times continuously and evidently suffered the most acute pain, apparently in the head.

CASE 293.

II.



Acquired internal hydrocephalus. Kernig's symptom. Male, 2½ years old.

On examining the child, you see that he is well developed and nourished. The terior fontanelle is still open. The fronto-parietal suture on the right side of the hea quite distended. His forehead is rather bulging. His eyes are somewhat prominent, rather depressed in the orbits. The pupils are dilated. The head measures 47.3 cm. (inches) in circumference, 33.2 cm. (13⅛ inches) from glabella to inion, 27.2 cm. (10¾ inc from ear to ear. The circumference of the chest is 49.7 cm. (19⅞ inches). There slight hemiplegia and paresis of the right arm and leg, but objects can be grasped with right hand. He cannot walk. There are no enlarged glands. Nothing abnormal is fo on examination of the heart, lungs, or spleen. The knee-jerks are increased, the right more than the left. There is no ankle-clonus. The teeth are in good condition.

On placing the child on the edge of a table (Case 293, II.), you will see that both legs become stiffened (Kernig's symptom).

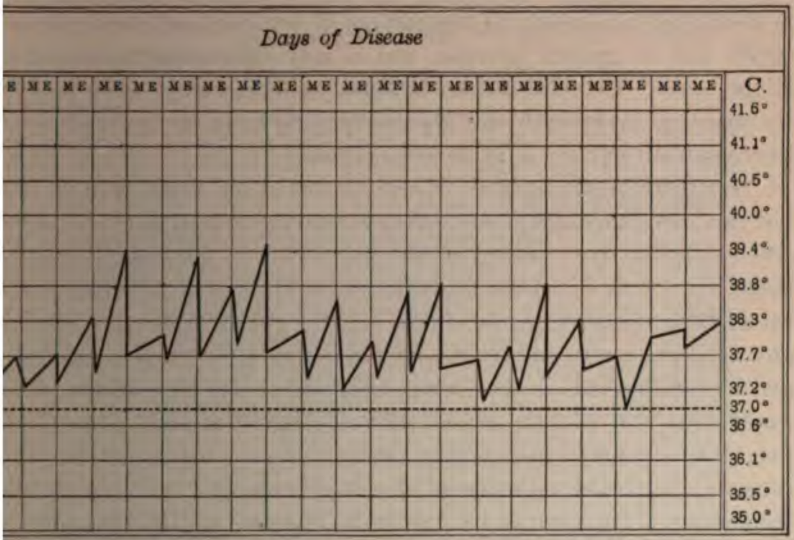
The child seems to be fairly bright and to be improving every day. He is much fretful than formerly. An examination of the eyes by Dr. Jack shows a beginning atro of the optic nerves, with retinal hemorrhages of the left eye.

child's temperature has varied usually from 37.7° to 38.3° C. (100° to 101° F.). e has been regular and somewhat quickened.

diagnosis of this case is evidently one of intra-cranial disease. The disease was its onset, and was accompanied by extreme pain in the head, convulsions, and ousness, followed by a partial paralysis of the arm and by loss of the power of . The protrusion of the eyes would indicate intra-cranial pressure, and the paralysis ra-cranial lesion, possibly of mechanical origin. What the nature of the original as cannot now be determined, but it was evidently of an acute inflammatory type, eems as though it must have been connected with an inflammatory condition probcting the ventricles. Following this inflammatory condition, the symptoms indiintra-ventricular effusion, and I think we can therefore assume that, whatever the cause of the disease was, the child may now be said to have chronic acquired hydrocephalus.

bsequent history.) During the following year the child improved slowly but y. He became less fretful; he learned to talk better, and finally to walk. His

CHART 25.



Chronic acquired internal hydrocephalus. Male, 2½ years old.

ture became normal, and when last seen, at the age of three and a half years, he to be perfectly well, the paralysis of the arm and hand having almost disappeared. re is the temperature chart (Chart 25), showing the course of the temperature for one days in the third month following the original attack.

LECTURE XXXI.

BRAIN.—(Concluded.)

CEREBRAL ABSCESS.—CEREBRAL PARALYSIS.—ATHETOSIS.—INTRA-CRANIAL TUMORS. —INTRA-CRANIAL SYPHILIS.—IDIOCY.—MIRROR WRITING.

CEREBRAL ABSCESS.—Cerebral abscess is a localized purulent encephalitis. It is probably always secondary to suppurative disease elsewhere. It may arise from a suppurative condition of the scalp, but its most common source is some purulent disease of the ear or its surroundings. It is also found as a sequel to traumatism of various kinds resulting in suppuration and in general pyæmia, and it may follow direct traumatic injury to the brain. Cerebral abscess is usually single, except when it is produced by pyæmia. Although the abscess may occur in any part of the brain, a very common locality is in the cerebellum.

SYMPTOMS.—A cerebral abscess may exist for a considerable time without producing any symptoms which can be recognized during life. In cases where suppurative disease of the ear exists, a cerebral abscess may be suspected where, in addition to the temperature, which would naturally be raised from this process, the child's general condition becomes worse without any apparent cause, and where indefinite symptoms, such as mental dulness and irritability, arise. The temperature may also suggest the presence of imprisoned pus, and the probability of cerebral disease, in cases where the pus cannot be found elsewhere. Cerebral abscess may, however, exist for a considerable period without rise of temperature, and even with a subnormal temperature. It is apt to be slow in its progress and to cause general constitutional rather than local symptoms. Local symptoms produced by the presence of cerebral abscess are rare. When present, however, they are represented by headache, vertigo, mental dulness, vomiting, and convulsions, and are followed later by coma. When the abscess bursts into the ventricles, symptoms of sudden collapse appear, and death rapidly follows. Tremor and convulsions may occur in cases of cerebral abscess, but neither of them should be considered as in any way symptomatic of this condition.

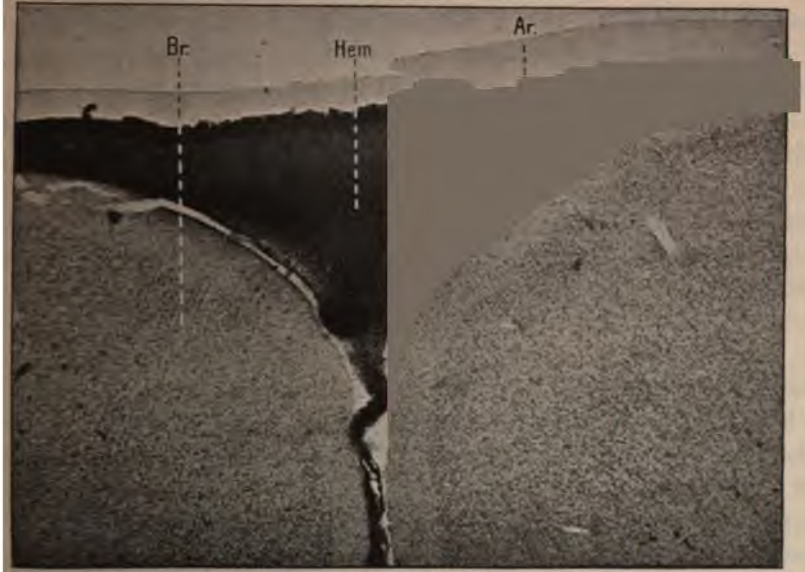
PROGNOSIS.—The prognosis is very unfavorable unless the disease can be reached surgically.

TREATMENT.—The treatment should be operative if the abscess can be localized.

CEREBRAL PARALYSIS.—In using the term cerebral paralysis it must be understood that it is not intended to describe every disease of intra-cranial origin from which a paralysis may result. We may have a resulting paralysis from many intra-cranial lesions, such as hydrocephalus, ebral abscess, cerebral tumors, and other causes. The class of cases iich I am about to describe under cerebral paralysis occurs usually in ldren under three or four years of age. In a certain proportion of these es hemorrhage, embolism, and thrombosis are the causes of the acute nptoms. Most of the autopsies which have been made have shown erosis, atrophy, or porencephalia, which are probably secondary rather in primary. In these latter cases the original primary cause is not own. Cerebral paralysis results in a spastic paralysis involving one more extremities, and may be in its distribution monoplegic, hemigic, paraplegic, or diplegic.

Etiology and Pathology.—I shall first speak of the three known mary causes which I have just mentioned,—namely, (1) hemorrhage (rupe of one or more blood-vesssls), (2) embolism (a foreign body brought to brain from some distant part of the circulatory apparatus), and (3) ombosis (an occlusion of one or more of the cerebral blood-vessels by a ıl coagulation of the blood).

Fig. 95.



Br., Brain. Hem., Hemorrhage. Ar., Arachnoid.

Of these three known primary causes hemorrhage is the most common. is hemorrhage is more apt to be meningeal than cerebral. It is for this tomical reason that these cases of hemorrhage in infants are less liable to ve fatal than those which occur in adults. I have already described to ı a case (Case 186, page 449) of presumably meningeal hemorrhage in infant three days old. This case illustrated the possibility of recovery in n severe cases of intra-cranial hemorrhage.

Through the kindness of Professor Northrup I am enabled to show you s specimen (Fig. 95) of a case of meningeal hemorrhage in an infant rn prematurely at the eighth month.

The mother had puerperal convulsions, and the delivery was by forceps. You see that the hemorrhage is in the locality where it is usually found in such cases, the *subarachnoid space* (vide Diagram 8, page 594). Intra-cranial hemorrhage in the infant and the young child may arise from various causes, such as increase of the intra-cranial pressure from various diseases, which produce stasis of the blood-current, or from traumata, whether from pressure or from direct injury to the skull and the brain. In addition to these causes, certain changes in the blood-vessels themselves, representing an atheromatous condition, are supposed to give rise to intra-cranial hemorrhage. Certain forms of degeneration may cause such a disorganization of the walls of the cerebral blood-vessels as to result in hemorrhage. In this latter class syphilis is a factor which must be considered, as must also, according to Sachs, general tuberculosis, meningitis, and cerebral tumors.

Next to hemorrhage, embolism is the most common cause of this class of cerebral paralysis. So few cases, however, have thus far been satisfactorily investigated by post-mortem examinations that I shall not dwell upon this condition, nor upon the still more rare resulting pathological lesion, thrombosis, except to explain that the emboli and thrombi act by cutting off the blood-supply of a certain portion of the brain, thus producing the disintegration of the cerebral tissue and the resulting paralysis.

As I have used the terms sclerosis and porencephalia, it may be well to define them.

Sclerosis consists of a shrinking and hardening of the cerebral tissues usually more or less strictly localized.

Porencephalia denotes a pathological hollow or depression in the brain running from the cortex towards the centre and usually communicating with a lateral ventricle.

The general pathological conditions to be remembered in cerebral paralysis, no matter what the original lesion, as has been so clearly summarized by Lovett in his paper on "Cerebral Paralysis in Children," are, *first*, a lesion of the brain involving, as a rule, some portion of the motor tract; and, *second*, atrophy and retarded development of the brain, with a descending degeneration of the lateral columns of the cord and pyramidal tracts. Finally, there is a possibility that the cause may be a defective development of the nervous centres.

Cerebral paralysis may occur in connection with a number of diseases, such as the acute exanthemata, pertussis, diphtheria, parotiditis, typhoid fever, and after continued convulsions. Difficult parturition, with or without the use of forceps, seems to be responsible for a certain number of the spastic cases, both paraplegic and hemiplegic.

SYMPTOMS.—Having made this preliminary explanation of the kind and extent of the knowledge which I am endeavoring to convey to you concerning the cerebral paralyses of infants and young children, I can now state the important general features of the disease which I should like to have you remember.

lesion has been of intra-uterine origin, we may get only the later
ons of this lesion, just as we do in congenital syphilis. In like
the lesion has occurred at the time of delivery, the primary
are often masked, and the resulting symptoms of the more
athological condition are noticed later.

the disease develops in extra-uterine life it is usually acute in
r and is marked by more or less fever, convulsions, and stupor.
symptoms are merely those of a general nervous explosion fol-
irritation of the nervous motor centres. They may be the first
ons of a disease of any kind, or they may occur in the course of
liseases of which I have spoken under etiology. If they happen
night and are of short duration, they may be entirely overlooked,
er symptoms of a cerebral lesion may be the first ones to mani-
lves. The child may die from the severity of these initial lesions
later symptoms of paralysis have developed by which we can
te the disease. Screaming, vomiting, and delirium may at times
e attack. In the midst of or closely following these primary
come the pronounced indications of a central nervous lesion,
by hemiplegia (paralysis of an arm and a leg on the same side),
(paralysis of both legs), or diplegia (paralysis of both arms and
cases of hemiplegia being the most common. In rare cases we
ne extremity affected (monoplegia).

tion to the paralysis of the limbs, facial paralysis may occur either
gia or in diplegia, and, as a rule, spares the upper part of the
t the eyes can be closed and the brows raised, thus showing that
eripheral facial paralysis. This form of facial paralysis often
early.

mining the paralyzed limb we find a resistance to motion, the
s are exaggerated, and in most cases there is a feeling of rigidity
lyzed side. A few cases of flaccid paralysis have been reported.
s a rule, is not affected. When the child has come out of its
the convulsions have ceased, it may be found to be aphasic. The
is usually impaired, but this, of course, depends upon the loca-
tent of the lesion and the period when it occurred.

ra-uterine and early infantile cases show the greatest mental dis-
These children are apt to be very irritable, and, where the lesion
epileptiform convulsions are quite common. The electrical re-
e muscles is normal. In the more advanced stages of cerebral
lditional symptoms begin to appear. The child learns to walk
it has already walked, the gait becomes peculiar. Rigidity fol-
ontracture of the flexor and adductor muscles may occur. In
s the spastic condition is so pronounced that the patellar tendon
he ankle-clonus cannot be obtained. When walking is attempted,
is apt to stand on the toes, the knees knock together, and the
dity of the muscles produces what is called the spastic gait,

which the child's toes scrape along the ground and the feet t against each other on account of the contraction of the adduct

In the milder cases the same manner of progression occur sudden and jerky, and the foot can be raised from the ground steadiness thus results in these cases.

The affected limbs are apt to show some disturbance of the and some coldness. There are more or less atrophy and shor bone, but to a less degree than in cases of poliomyelitis an certain number of cases involuntary incoördinate movements the paralyzed limbs on voluntary effort (hemiataxia, Osler), an designated as post-hemiplegic chorea. There may also be cont ments (athetosis) of either a partial or a general variety. T are not affected, whether the case is one of hemiplegia or of par epileptiform convulsions which I have already referred to may early in cases of cerebral paralysis, but may also be delayed of years, so that the possibility of these children becoming e always be considered.

Diagnosis.—The general diagnosis of cerebral paralysis w to the special cause is of great practical importance to the pra cian, and should be thoroughly mastered before he attempts cate the exact nervous lesion or to locate it with the precision neurologist.

The diagnosis in a marked case of the disease is not diff determination of the exact lesion is often impossible after the p has passed and we are left with a resulting paralysis. If faci present, we can, as a rule, say that the lesion is in the brain; does not always hold good, as there have been very rare cas paralysis was present when the lesion was in the cord.

The symptoms on which we chiefly rely in making our diag bral paralysis are (1) the distribution of the paralysis, hemiple paraplegic; (2) increased tendon reflex; (3) wasting compara contradistinction to the chief diagnostic symptoms of cerebral paralysis which I have just given you, you will find in poliomyelitis anterior (1) that the distribution of the paralysis is usually monoplegic; (2) that there is an absence of tendon reflex; (3) that there is an absence of rigidity in the early stages; (4) that there is rapid and marked wasting of the affected limbs; (5) that the reaction of degeneration is present; and (6) that there is no mental impairment.

In certain cases also a difficulty may arise in correctly understanding the relationship between cerebral paralysis and idiocy. The cerebral lesion is in many cases probably the same, but, according to its extent and location, we may have either (1) a cerebral paralysis alone; or (2) a cerebral paralysis accompanied by mental impairment or idiocy; or (3) idiocy without cerebral paralysis. There is a certain class of low-grade idiots in which some impairment of motion exists, apparently due to a mental inability to coördinate the muscles of the limbs properly. This may sometimes be accompanied by a diminution of sensation, which seems to be due to a want of perception in the higher nervous centres rather than to any actual lesion of the sensory tract. When the idiot's attention can be kept centred on the limb, the actual sensation does not seem to be much impaired. The differential diagnosis of this condition occurring in idiots from cerebral paralysis is easily made, for it exists in those cases only of the former where the mental development is much impaired, and it is not, as a rule, accompanied by true paralysis, as there is no weakness, but simply incoördination; in these cases also the tendon reflexes are, as a rule, not increased.

Cerebral paralysis can be diagnosticated from the paralysis which occurs in connection with caries of the spine, principally by the presence of cerebral symptoms in one case and the prominence of the spinal vertebræ and the rigidity of the spine in the other.

I should also mention here that the rare cases of syringomyelia may be mistaken for cerebral paralysis. The points of differential diagnosis in these cases are that in syringomyelia, although the weakness of the limbs may be so extensive as closely to simulate paralysis, yet the diminution of thermic sensation, which I shall presently speak of when describing the disease to you (page 690), easily distinguishes it from the normal sensation which is present in cerebral paralysis in cases where the test for sensation can be employed. The disease, however, is so rare in children that it need not be dwelt upon.

PROGNOSIS.—The question which immediately arises when the physician is confronted with a case of paralysis in an infant or a child is, What will be the result of this attack? not, What is the special anatomical lesion which is causing it? Knowing, as I shall presently explain to you, that where the lesion is of spinal origin the chances for recovery are fairly good, you will at once appreciate the vast difference which your answer may make to those interested in the child when you state that the disease is in the cord and that recovery is probable up to a certain point without mental impairment, rather than that the brain is involved and possibly mental as well as physical incapacity for life may result. The prognosis for life in cerebral paralysis is soon determined in the early days of the attack, and of course depends on the location and extent of the cerebral lesion. Entire recovery is rare. The leg, as a rule, recovers more rapidly and to a greater extent than the arm, which seldom regains its entire usefulness. The spastic rigidity usually goes on to decided contracture. In some cases no mental change is apparent; in others the mental development is merely retarded, and the child learns to talk some years later than is normal. In a large number of cases, however, the mind is much enfeebled. The occurrence of epilepsy as a result of cerebral paralysis is so common that it should be especially mentioned in this connection, as it makes the prognosis much more serious both as to the degree to which the mental impairment may attain and as to the life of the patient.

Except in very rare cases, the children can be taught eventually to walk.

TREATMENT.—The treatment of cerebral paralysis must necessarily be unsatisfactory. It is to be directed to keeping the paralyzed limbs in as good a condition as possible and thus avoiding contractures of high grade. This can be accomplished in a measure by patient and continued massage and manipulation, chiefly in the direction of stretching the flexor muscles and cultivating the use of the extensors. The faradic current used three or four times a week for five or ten minutes is a useful adjuvant, and, if necessary, surgical interference to relieve undue tension of the flexor tendons is indicated.

The mental training of these cases is exceedingly important, and should be attended to carefully. In this connection it is well to remember that the division of the contracted tendons in some way seems to improve the mental condition. Trephining the skull over the supposed seat of the lesion does not, with our present knowledge of the usual nature of these lesions, present a particularly encouraging outlook. The few cases which have been operated upon have not been benefited.

It should be thoroughly understood that surgical operations to relieve the contractures do not influence favorably any pre-existing paralysis or incoördination, but that it often puts the limbs in a condition in which massage and electricity can be applied to greater advantage. The indications for the division of the tendons of the contracted muscles exist when the contracture is so firm that thorough treatment by massage and electricity produces no essential relief. Cutting the tendons in cases of low grades of idiocy has usually been considered contra-indicated, but this opinion is not shared by all observers, as in a certain number of cases at the Boston Children's Hospital decided benefit has been found to result.

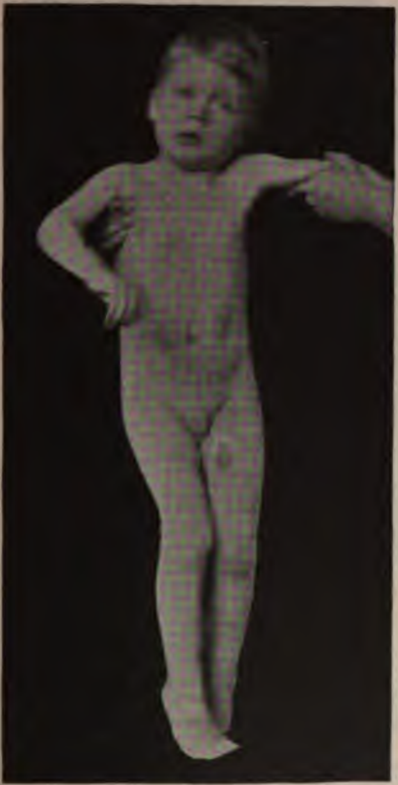
I have thus endeavored to give you a precise and practical idea of a very complicated subject. In order to do this I have used as few names as possible and have avoided many plausible but theoretical explanations of noted writers. As an instance of this, I have passed over Strümpell's brilliant

ved theory of *polioencephalitis* as one of the causes of cerebral
You will, however, now understand how inadequate are the
mes, such as *spastic paralysis*, *spastic rigidity*, *spastic diplegia*,
ease, and *infantile hemiplegia*, to cover the broad range of pathology
toms which is represented by the class of cerebral cases which I
nated under the general term *cerebral paralysis*.

a number of cases here to show you which represent this condi-
rebral paralysis in children.

t case is a boy (Case 294), five years old. Up to the age of seven months he is
been in a normal condition. The disease which was followed by the symptoms
ow presents occurred when he was seven months old. At this time he was
h fever and a convulsion, and later was found to have paralysis of the right arm
gs.

CASE 294.



ral paralysis. Right hemiplegia, with affection of opposite leg. Male, 5 years old.

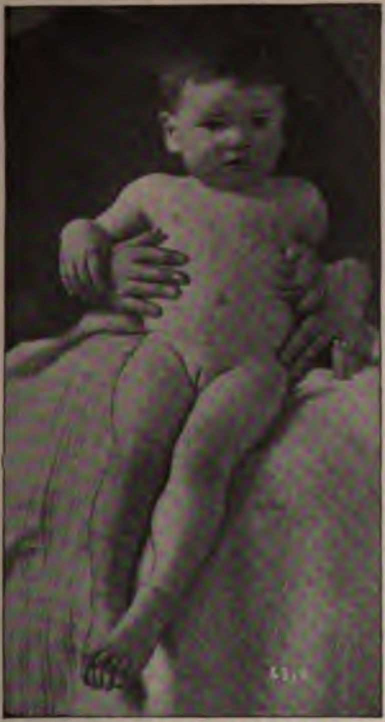
mining the child you see that the thumb is turned in on the palm of the hand
ers are slightly flexed and at times slightly extended. Both legs are somewhat
knees. The hamstring tendons are tense and unyielding. The knee-jerks are
The heels are raised from the ground.

ld is a case of hemiplegia with affection of the opposite leg.

d cases of this kind the treatment is by massage and electricity; in the more
apparatus is required. In a very severe form like this, operative interference

little girl (Case 296), two years old, was born after a severe instrumental labor.
has always from birth shown weakness of the arms and legs. She was unable to
til she was a year old, and she has never stood or walked. Her intelligence is
y normal. You see that the cranium is normal in shape, that the parietal emi-
e somewhat enlarged, and that the fontanelle is still open. There is no disturb-
he facial muscles. The right leg is slightly larger than the left. The teeth are
ent condition. The upper extremities appear alike, but she cannot loosen her
ter grasping an object with her right hand. The epiphyses of the wrist are much
and those at the ankles are slightly so. There is no definite rosary. The back is

CASE 296.



Cerebral paralysis. Congenital cerebral diplegia and rhachitis. Female, 2 years old.

t rigid. There is no marked deformity. There is a tendency to rigidity in both
tremities. The feet are inverted. The patellar reflexes are increased. Sensation
l. She can use her hands well, except as above described.

represents the class of cerebral paralysis which is called cerebral diplegia. You
he is also rhachitic.

is a little boy (Case 297, page 658), four years old, who was perfectly well at
t who when he was six months old had a number of convulsive attacks without
wn cause.

en he was two years old he had an attack of measles, followed by varicella, and
pertussis. He has never been able to sit or stand alone. He is fairly developed
ished, and his intelligence is normal. He has marked general kyphosis when
l by the arms. When he is assisted to walk he also shows the condition of cross-
rogression. The arms are somewhat stiff, and he holds the forearms slightly
. The triceps reflex is somewhat increased. The legs are usually held somewhat
the body, and the knees are also slightly flexed, with the feet in the position of
uinus. The knees are held closely together. Rigidity is less marked in the right

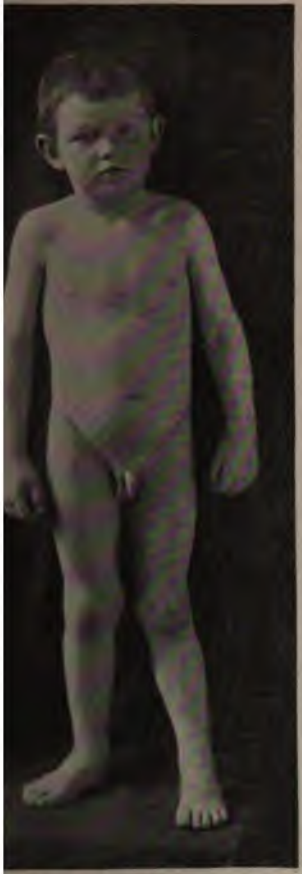
eighteen months old. He is stated to have had convulsions in his third year, and nvulsions occurred again when he was four years old. They were followed by the s for which he has come to the hospital to be treated. He does not use his left hand id the grasp of the left hand is less strong than that of the right. The triceps exaggerated on both sides. The left foot can with difficulty be flexed dorsally. ht knee-jerk is normal, the left is increased. He has flat-foot, and walks with his rotated in. He is now six years old, and is otherwise well and strong.

is is a case of left spastic hemiplegia.

treatment in this case is by massage and electricity. Apparatus does not seem to ated, as its only use is to support the limbs or to correct deformity.

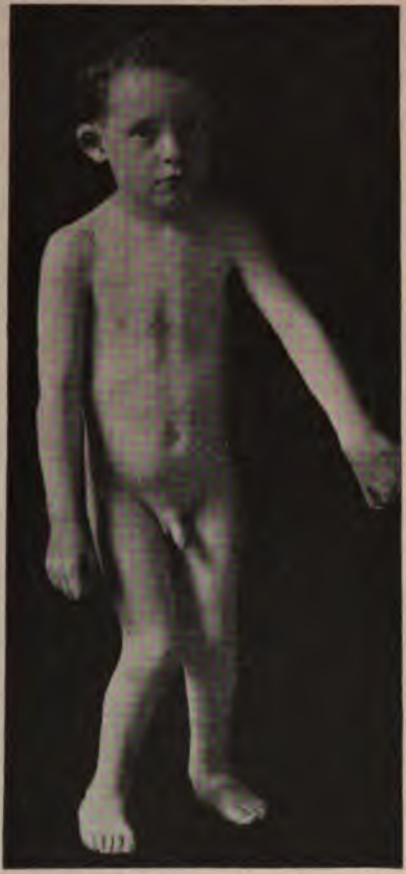
s boy (Case 300) is four years old. There is a history of phthisis on the maternal

CASE 299.



paralysis. Left spastic hemiplegia, ars' duration. Male, 6 years old.

CASE 300.



Cerebral paralysis. Spastic paraplegia. Male, 4 years old.

mother has four other, healthy children, but has a history of three miscarriages. ild was born prematurely, and the delivery was instrumental. He has always been , and had an attack of measles one year ago. He did not attempt to walk until three years old, and it was then noticed that he did not use his legs well. He is y normal. His arms appear to be normal. When placed on the floor he gets up in er like that which is shown in cases of pseudo-hypertrophic muscular paralysis. e stands his knees are highly flexed and adducted. He walks on his toes, with a y to cross the knees. This tendency can be only partially overcome. There is no apparent atrophy of the muscles. The knee-jerks are slightly increased, and there is slight ankle-clonus. The skin shows some disturbance of circulation.

He represents the class of cerebral paralysis which has been designated spastic paraplegia, the original cerebral lesion having affected the legs only.

If this child's condition is not much improved by passive movements of the limbs and massage, it may be advisable to resort to operative treatment and divide the tendons of the flexor muscles.

I happen to have here in the wards a case which apparently represents the symptoms of traumatic hemorrhage.

This little girl (Case 301) is four years and nine months old. She was brought to the hospital February 28, with a history of having fallen from the roof of a three-story building upon a brick sidewalk. She was unconscious. She vomited slightly, and she was found to have an ecchymosis on the left side of her head. Her pupils were equal and reacted to light. Her respirations were rapid; the extremities were cold. She moved all her limbs vigorously. Some clotted blood was found in and about the nostrils. The temperature was 36.3° C. (97.4° F.); the pulse was 90, and the respirations were 26. She ground her teeth and cried out in the night. The muscles of the left arm and leg moved actively.

On the next day it was found that she could swallow milk. She passed her urine involuntarily. She was still unconscious, and the movements of the left arm and leg continued.

On the following day, for a short time the right pupil was larger than the left, and would not react to light. Although she could not speak, her eyes would follow the finger; the eyes also had a restless movement. An enema produced a passage of a small amount of fæces and a few drops of blood. The respirations were very deep, and the face was flushed.

On the following day she still continued to move her left arm and leg, while the right arm and leg remained passive. The pulse was irregular and intermittent. She was reported to have slept more than at any time since the accident. She was still unconscious, but was less restless.

On the next day the pulse was irregular, as it was also two days later. The pupils were irregular, and she opened her eyes and fixed them on objects at times. She also rolled her eyes and yawned. She was still unconscious.

Two days later she had slight opisthotonos, and there were spasmodic movements of the left arm and leg. She slept a great deal.

On the following day she appeared brighter, and followed objects with her eyes. Her pulse was irregular, from 80 to 90.

Two days later she seemed brighter, and moved the left arm and leg less. She also made voluntary movements, such as to push objects away from her. On this day she gave evidence that she understood what was said to her. Three days later she seemed to recognize her mother.

On the following day she began to use her right arm very slightly. She ate a cracker, and was at times quite conscious.

The next day she appeared more intelligent, and on the day after that she began to speak single words. It was found, however, that she could move her right arm but very slightly. Since this time she has always been perfectly conscious, endeavors to say words, and notices the children in the wards, as well as her playthings.

To-day,—the twenty-ninth day from the time when the accident occurred,—as you see, she can walk, though with difficulty, as the right leg is very unsteady.

She apparently has had a lesion on the left side of the brain, represented by a hemorrhage and caused by traumatism.

(Subsequent history.) One week later she was discharged from the hospital. At that time she could use the right arm fairly well, but walked with some difficulty on account of the weakness of the right leg. Her articulation was labored, and her pupils were unequal.

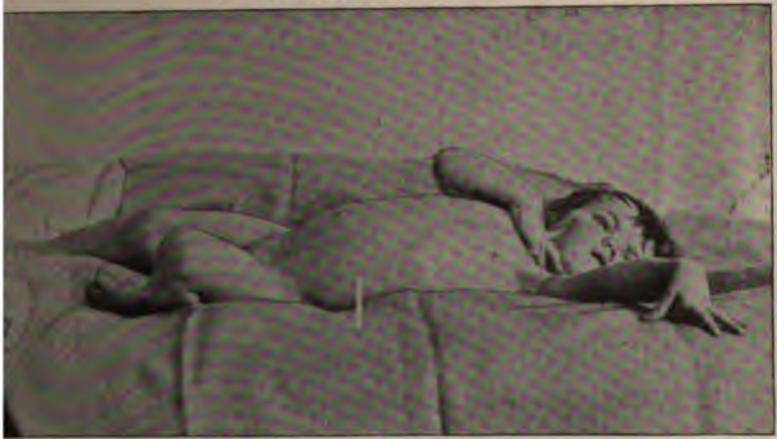
ETOSIS.—Athetosis is a symptom, and not a disease, and is repre- continuous incoördinate arhythmical movements of the extrem- face, and the body. This condition may be acquired or congenital. ired form may follow cases of hemiplegia or diplegia, in which event the paralyzed limbs. Certain cases of acquired athetosis occur ny accompanying paralysis. In congenital athetosis, and in the form without paralysis, the symptoms usually begin in the first

OLOGY.—The pathological condition which exists in cases of athe- upposed to be a chronic cerebral irritation in the neighborhood asal ganglia and in the internal capsule. The condition as we nically, therefore, is wholly a symptom of some organic lesion of

NOSIS.—The diagnosis of *acquired athetosis* is made by the charac- movements. These are continuous, and are distinguished from horea by being vermicular and less spasmodic.

liagnosis in cases of *congenital athetosis* is not difficult, as in no ase does an infant present at birth these peculiar movements and sque form of flexion and extension of the fingers and toes. The alled *congenital chorea*, in which involuntary arhythmical move- ist, is distinguished from athetosis by the character of the move- hich resemble those of ordinary chorea.

CASE 302.



Congenital athetosis. Female, 2 years old.

NOSIS.—The prognosis of athetosis in regard to recovery is un- . So far as the general health is concerned, the individual may fairly well and may live for years, as in the case of a man, twenty- s old, reported by Bullard.

TMENT.—There is no known treatment which has proved to be of n children. As they grow older the training of the affected limbs may be undertaken, but, as a rule, the results are unsatisfactory. Massage and electricity have proved to be of no value.

I have here a little girl (Case 302, page 661) who represents this condition of congenital athetosis.

She is two years old. She has never had any acute disease. She was born after a normal labor, and has received no subsequent injury. She has never talked nor shown much interest in her surroundings, nor has she been able to sit up or hold up her head without support. The bowels have always been regular and the appetite good. She is well developed, and, as you see, well nourished.

The disease is characterized by the continual incoördinate arhythmical movements of the head, trunk, and extremities; these movements are often quite rapid. There is constant flexion and extension of the hands and fingers, the fingers at times being bent backward and assuming most grotesque positions. This phenomenon is also seen in the toes. The expression of the face, as you see, is not that of ordinary intelligence. I find that I cannot determine the reflexes, on account of the resistance of the child to examination. She is usually irritable, but occasionally smiles slightly and takes some slight notice of those who are near her.

The prognosis in this case, so far as recovery is concerned, is bad. There seems to be no especial reason why she should not live.

INTRA-CRANIAL TUMORS.—In infancy and early childhood tumors of many varieties may occur in the brain and its meninges. The most common form of intra-cranial tumor is tubercular. The next in frequency are gliomata, sarcomata, and glio-sarcomata. The other varieties, such as carcinoma, lipoma, myxoma, and teratoma, are very rare; and syphilitic gummata, which are so frequent in adults, are exceedingly rare in infancy and early childhood. The parasitic cysts in the brain which occur quite frequently in individuals in other parts of the world, especially in Germany, are seldom met with in this country.

These tumors may be either of intra- or extra-uterine origin. Of these the tubercular is the most common.

PATHOLOGY.—The *tubercular* tumors of the brain or its meninges are, as a rule, secondary to a tubercular growth in some other part of the body, or to tubercular disease of some part of the skull, such as the orbit or ear. These tubercular tumors may be single or multiple, the latter being the more common variety. They may be found in any part of the brain or its meninges, and occur with especial frequency in the cerebellum. They may vary in size from a small collection of miliary tubercle to much larger masses. When one or more cheesy masses of a tubercular nature are found in different parts of the brain, the condition is called solitary tubercle. The *gliomata* grow most frequently in the white substance of the brain, but sometimes develop in the gray matter. According to Starr, they grow less rapidly than sarcomata, and never involve the membranes. They are usually primary, but may develop as secondary to glioma of the retina (Starr). The sarcomata are both of the round-celled and of the spindle-celled variety. Although not quite so frequently found as the gliomata, they are more frequent than the glio-sarcomata or myxomata. They are usually round in shape, and develop both in the nervous tissue and in the cerebral membranes, and in both the white and the gray matter of the cerebrum and cerebellum. The other varieties of tumor of the brain are so rare that they need not be considered here.

In connection with intra-cranial tumors, I might mention that *intra-cranial aneurisms*, according to Starr, are rare in childhood and are never very large. They increase in size rather more rapidly than aneurisms elsewhere, and show a tendency to rupture. They are found upon the larger arteries of the base of the brain and on the Sylvian arteries. The pathological condition of the brain in the neighborhood of these growths is such as would result from the impediment to the blood-current in the small vessels, or from compression of some of the larger arterial trunks. The condition is usually one of anæmia. The anæmia may be sufficient to impair the nutrition of the nervous tissue. As a still later pathological condition in these cases produced by pressure, areas of atrophy of the brain may occur.

SYMPTOMS.—The symptoms which result from intra-cranial tumors are very numerous, and are rendered all the more difficult to recognize in infancy and early childhood by the pronounced nervous phenomena which may result from even a slight degree of irritation or pressure in the young and undeveloped brain-tissue.

The general symptoms vary very much in accordance with the size and vascularity of the tumor, and according as it is growing or has become stationary. In the former case the symptoms are often apt to be more severe than later, when, the tumor having become stationary, the brain-tissue adapts itself to the new conditions produced by the morbid growth. Intra-cranial tumors in infants and in young children are often latent, present no symptoms, and are sometimes discovered only after death. A certain number of cases, on the other hand, present only general symptoms, such as headache, cerebral vomiting, attacks of vertigo, convulsions, and optic neuritis, which cause us to suspect intra-cranial disease, but give an indefinite idea of its location. Again, these tumors may produce local symptoms in addition to the general ones. These local symptoms are represented by paralyses of different kinds, anomalies of sensation, affections of the special senses, and staggering. These later symptoms arise according to the site of the tumor and the parts of the brain which are affected by it, and by means of them we can more or less approximately judge of its situation, size, and rapidity of growth.

I shall not enter here into the various complex questions of brain localization, but shall refer you for further information to works especially devoted to that subject (Keating's "Cyclopædia of the Diseases of Children;" Starr). I may, however, say that paralyses of the extremities are caused by an affection of the motor cortex, the internal capsule, or any portion of the motor tract on the opposite side of the brain above the crossing of the pyramids. Staggering or cerebellar ataxia is suggestive of cerebellar disease, while the involvement of the intra-cranial nerves suggests a tumor of the base of the brain or pressure on these nerves at some point, and more rarely an affection of their nuclei. The tendon reflexes are apt to be exaggerated, but in some cases are normal, and in others are said to be absent. The symptoms of cerebellar ataxia which at times occur where the tumor is situated in the cerebellum consist of a staggering gait resembling that of an intoxicated person, the steps being irregular in length and the body swinging from side to side. The child in these cases has a subjective sense of falling or turning back, and grasps for support or sinks into a chair or to the floor. This form of ataxia is to be distinguished from that which is found in spinal disease, and which is due to an inability to coördinate the muscles of the lower extremities properly. This latter form of ataxia is much more regular than the former, each step being insecure and unsteady, but without the violent and sudden reeling, after two or three steady steps, which occurs in the cerebellar form.

In young infants a tumor may cause a protuberance of some part of the skull by pushing one of the bones outward, as was seen in a case (Case 303), eight months old, of teratoma which was operated upon by Dr. Lovett at the City Hospital, and which is one of the few instances of this form of tumor on record.

DIAGNOSIS.—The diagnosis of tumors of the brain must in the great majority of cases be made by elimination. The variety of tumor can be determined most readily by considering the history of the case, as to whether it is tubercular, syphilitic, or otherwise. The diagnosis of a tumor can often be made by the slow and gradual development of the disease. When severe headache and vomiting exist, followed by paralysis, either monoplegic or hemiplegic, especially if this paralysis develops slowly, we should suspect the presence of some form of intra-cranial growth. This suspicion is much strengthened by the presence of optic neuritis or optic atrophy. The presence of localized convulsions in such cases tends to confirm the diagnosis, while if marked ataxia exists we are justified in suspecting cerebellar disease. A normal or only slightly elevated temperature with these symptoms which I have just mentioned also points to the diagnosis of a cerebral tumor.

PROGNOSIS.—The prognosis of tumors in early life is very unfavorable, no matter what the variety of the tumor may be. Although the patient may for a long time remain wholly unaffected by the morbid growth, he eventually, except in rare cases, succumbs to the disease.

TREATMENT.—Surgical interference in children, as in adults, proves on the whole to be the most valuable means at our command for lengthening life in cases of cerebral tumors. There is no other treatment which is of any especial benefit in either retarding the growth or curing this class of cases. Even where the exceedingly rare form of syphilitic gumma exists, iodide of potassium and other drugs have not apparently proved to be of much value.

In regard to what I have said concerning the latency of tumors of the rain, the case which I showed you in the wards some days ago exemplifies he extent to which this latency can exist where the tumor is tubercular.

I have to-day the opportunity of presenting to your inspection the results f the autopsy on this case.

You may remember my telling you when I was examining this infant (Case 304) while live that I could detect nothing abnormal except a moderately raised temperature by hich I could distinguish it from the case of infantile atrophy in the next bed, which had n almost identical temperature and similar symptoms.

The infant was thirteen months old, had never had any especial disease, and entered the ospital weak and emaciated. Its mind was clear. Its pulse was weak but regular, and either slow nor quick for its age. Its temperature was at times somewhat raised, varying from 37.2°–38.4° C. (99°–101° F.). There were no convulsions, and no paralysis or contractures, but merely progressive loss in weight, and finally death.

An examination of the brain of this infant shows miliary tubercle of the pia mater at the base of the brain without acute inflammation, which accounts for the lack of acute cerebral symptoms. Of especial interest, however, in the case are the patches of solitary tubercle, 1.2 cm. (½ inch) in diameter, which you see in the left temporal and occipital lobes and in the right frontal lobe of the cerebrum, and also in the lower left cerebellum. There is also caseous tubercle of the post-bronchial glands, tubercle of the lungs with a slight amount of broncho-pneumonia, miliary tubercle of the pleura, liver, and spleen, and caseous tubercle of the mesenteric glands.

Through the kindness of Dr. Bullard I am enabled to show you the result of the post-mortem examinations in some cases of cerebral tumors which have just occurred in his practice.

A boy (Case 305), four years old, of healthy parentage, but with a history of tuberculosis in his grandmother and an aunt, was perfectly well until he was ten months old. At hat time he had an attack of general tonic convulsions followed by paralysis of the right ower leg. After that the right leg slowly improved, but never entirely recovered. He egan to walk when he was fourteen months old. After this first attack he remained perfectly well until two months before his death, when he was found to have ptosis and acute onjunctivitis on the left side. Three weeks before his death he began to lose in weight nd to be very sleepy and stupid; he was feverish and lost the power of walking; he also ost his appetite and his bowels were very constipated. There was no history of his ever aving had any disease of the ears.

When examined by Dr. Bullard the head was not retracted, and no tenderness was ound anywhere over the cranium. When the left eyelid was raised the eye was found to e turned upward and outward. There was some swelling of the eyelids. Both eyes reacted to light. The tongue was protruded straight. The heart and lungs were normal. othing abnormal was found in the abdomen or spine. There was a flaccid paralysis of the ght lower extremity, with foot-drop. Nothing abnormal was found in the urine. A few ays later there was found to be some loss of power in the left upper extremity and left toe-rop. The knee-jerks were present.

He was treated with iodide of potassium, and his general condition improved somewhat. he drowsy condition, however, returned, and, although for a time improvement took place regard to the movements of his limbs, he gradually became more stupid, and finally was a torpid condition. He swallowed with great difficulty. He had strabismus of the left e. Nothing abnormal was found in the urine, but it was passed, as well as the fæces, involuntarily. His temperature varied from 37.2° to 37.7° C. (99° to 100° F.), and his pulse as between 80 and 90.

An examination during the latter part of his life showed that the thoracic, epigastric.

cremasteric, and plantar reflexes were excellent; the triceps reflexes were good. The knee-jerks were good, the right less than the left, the latter being exaggerated. Nothing else abnormal was detected.

An examination of the eyes by Dr. Standish, thirteen days before his death, showed marked choroiditis in the right eye, with large tortuous veins and arteries nearly obliterated. There was indistinctness of outline in the disk in the left eye, with the veins large in proportion to the size of the arteries. At this time he had deep sighing respirations with intervals of a minute or more.

One week before his death the right arm was rigid at the elbow and the hand and fingers were flaccid. At times an erythema would be seen on his arms and body. Turning his head evidently caused pain. The upper part of the head was cyanotic. He was much emaciated. The right pupil was much larger than the left, and neither pupil reacted to light. The pulse increased in frequency, and at times was between 158 and 160. The abdomen was retracted. He remained in a stupid state until his death.

The post-mortem examination made by Dr. Bullard showed rigor mortis present in a moderate degree. The abdomen was retracted. The head was larger than normal in proportion to the size of the body. Nothing else abnormal was noticed on physical examination.

The pleura and pericardium, with their cavities, and the heart were found to be perfectly normal. Behind and to the right of the trachea, at or just above its bifurcation, two nodules about 2.5 cm. (1 inch) in diameter were found; they were apparently enlarged lymph-glands. On section they were found to be composed of yellowish-white cheesy material. Nothing abnormal was detected in the right lung. In the left lung, about the centre of the upper lobe, was a cavity about 2.5 cm. (1 inch) in the longest and 1.2 cm. (½ inch) in the shortest diameter. This was filled with cheesy material, friable, and easily removed. The liver, spleen, intestines, gall-bladder, and bladder presented nothing abnormal.

On examining the head there was nothing abnormal noticed externally. The longitudinal and lateral sinuses contained a very small amount of blood, clotted and liquid. The dura mater everywhere seemed normal, and was not unusually adherent to the cranium. The pia mater seemed normal everywhere except in the neighborhood of the Sylvian artery. Here it was more adherent than elsewhere, small pieces of the brain coming away with it when it was torn off. Both lateral ventricles were enlarged.

On the superior surface of the cerebellum there was a projection in the median line of part of a mass which occupied the anterior portion of the central lobe. On section it was found to be yellowish-green and much firmer than the rest of the cerebellum. Nothing else abnormal was detected macroscopically.

The tumor was examined by Dr. Dunham, who reported that it appeared to have occupied the anterior middle portions of the cerebellum, and to be about 5 cm. (2 inches) broad, 3.5 cm. (1⅜ inches) from in front backward, and 2.5 cm. (1 inch) from above downward. It was circumscribed, and its substance was more consistent than that of the cerebellum. It had two globular projections 1.2 cm. (½ inch) in diameter, one on each side, extending forward, probably one towards each side of the upper part of the fourth ventricle or beginning of the aqueduct of Sylvius, but not far enough to invade the pons. It did not extend farther back than the limits of the quadrate lobes. The cerebellar peduncles were not involved. The amygdalæ, which were almost directly below and in front of the tumor, were not affected.

Histologically the tumor was a sarcoma. In parts the structure was gliomatous; in others the cellular elements were so abundant that the microscopic picture was like that of a small, round-celled sarcoma. There were many blood-vessels in the substance of the tumor. Although the lung was not much affected, several of the peribronchial glands had undergone cheesy degeneration. The kidneys were small, but their tissues showed nothing unusual. Nothing abnormal was found in the other organs.

There was no evidence of tubercle in the brain or its meninges.

The next case is that of a little girl (Case 306), eleven years old, a patient at the Children's Hospital. Her parents were healthy, and there was no history of any disease affect-he nervous system in the family on either side. There was no history of phthisis. The l was born after a natural labor, with a head presentation, and without the aid of for- . When she was eighteen months old she had an attack of pneumonia: she is said ive had some "head trouble" at that time, and was never well afterwards. Up to the of six years she had earache, accompanied with a discharge from the ear. According r. Bullard, there was some evidence of hydrocephalus at or before this time. She was r as strong as other children. She did not walk until she was twenty-seven months and she was more liable to fall than other children. She was always of a nervous perament, restless, and unable to sleep well. She could never bear any excitement. en she was seven years old she had another attack of pneumonia, with a complicating ussis.

Three years ago she had a severe illness, of which the most prominent symptom was in the head. This pain was intense in the temples, especially in the left one, and would hold the back of her head with both hands. There was much severe vomiting his time. The temperature was stated to be about normal, and the pulse natural. re was also pain in the neck and in all the limbs, but it was slight in the right ex- nities and more severe in the left extremities. This illness lasted ten weeks, and she er completely recovered from it. She, however, became well enough to go to school.

A little later she was found to be blind in the left eye, and three weeks later the right also became blind. The blindness was supposed to have come on gradually.

When she was between eight and nine years old she had another very severe illness, racterized by pain in the head and vomiting. At this time she was first noticed to have nentary "spasms," in which she would scream with pain and would then lose con- usness, but without convulsions or rigidity. There was no heightening of the tempera- during this illness. During this attack she could not move any of her limbs.

A few months later she began to improve, and a month after this was able to walk ie. After this there was gradual improvement.

When she was ten years old the headaches became worse, and she had a third severe ck, with vomiting and pain in the head, lasting four weeks. Since that time she has been able to walk alone.

On entering the hospital she was found to be totally blind. There were paresis and in- rdination of both lower extremities. There was considerable incoördination of the left d, while coördination of the right hand seemed normal. There was no atrophy any- ere. The sensation was unimpaired. The knee-jerks were alike and normal. For two ks she was unable to go to sleep easily, on account of pain and restlessness. While in hospital she would have nausea and vomiting at times, and headache would occur four five times a week, but not so severe as to make her scream. She was unable to walk hout assistance. When some one held her hand she walked with the feet quite straight, king the ground first with the heels, and tilting the pelvis more than normal. Her etite was good. At times she would have constipation, followed by diarrhœa, with in- intary dejections. The vomiting and headache continued. While she was in the hos- l she was for a time quite comfortable. Her temperature ranged from 36.9° to 37.7° C. .5° to 100° F.). There were no other symptoms worthy of note. Examination of the ne showed it to be normal.

After leaving the hospital, when she was eleven years old, she had less headache for a rt time, but then became worse. She had several severe attacks, reported by the family 'fainting-spells," in which there was loss of consciousness without convulsions, and she l quietly in one of these to-day.

I have here the result of the examination of the head and a statement of the patho- cal conditions which were found.

On removal of the external tissues the cranium presented a translucent appearance, gesting extreme thinness of the cranial bones, and large white bands 2.5 cm. to 3.7 cm. (1 ½ inches) broad lay in the position of the larger cranial sutures, as though these sutures long been held open by intra-cranial pressure. The bones of the cranium were unusually , those forming the calvaria being not much more than 0.6 cm. (¼ inch) in thickness. calvaria was very elastic, could be readily compressed, and when the pressure was usly. When the dilatation reaches an advanced stage a thinning of the erial walls results, which may lead to rupture of the blood-vessels or to norrhage. More common than the hemorrhage, however, is the occlusion the blood-vessels, which cuts off the blood-supply and acts in the same y as in other cases of thrombosis of the arteries, causing more or less tening and disintegration of the cerebral tissues supplied by them. The eries of the base of the brain are the ones that are most frequently ected, and there are secondary lesions of the parts of the brain supplied them.

SYMPTOMS.—The symptoms dependent on these lesions vary in accord-ce with the pathological condition.

In syphilitic meningitis the principal symptoms are severe headache in rious parts of the head, more or less constant, lasting for many days or en weeks, and frequently accompanied after a time by paralysis of some f the intra-cranial nerves, especially of the third or of the seventh. As in ther cases of meningitis, the optic nerves may also be affected, and the child hows the general symptoms of a severe intra-cranial affection, such as omiting and dulness.

The localized tumors or gummata present essentially the same symptoms s do the other forms of tumors of the brain in children which I have just lescribed.

The symptoms produced by syphilitic endarteritis are the direct result f either the local dilatation or the local occlusion of the blood-vessels, which I have just mentioned. The symptoms vary according to the areas f the brain affected, but the most common ones are the various forms of aralysis of the extremities and sensory disturbances.

DIAGNOSIS.—In regard to the diagnosis of intra-cranial syphilis in hildren, the symptoms differ greatly in different cases. The most charac-eristic group of symptoms, and one which is exceedingly suggestive of ntra-cranial syphilis, includes attacks of organic paralysis, central in origin, ccurring at intervals of days or months without known cause, and without narked symptoms of either tumor or tuberculosis.

The diagnosis of cerebral meningitis may be made from the occurrence of evere headaches, followed by paralysis of one or more of the motor cranial erves, and occurring without marked rise of temperature.

Gummata present no symptoms sufficient in themselves to distinguish nem from other intra-cranial tumors, so that their existence can only be uspected.

The presence of syphilitic lesions elsewhere is our principal ground for aking the diagnosis.

Syphilitic endarteritis may be suspected when an acute affection in the ighborhood of the pons or medulla not produced by traumatism occurs a syphilitic subject, or where acute symptoms suggestive of hemorrhage embolism occur, and where no other probable cause can be shown, such cardiac or renal disease.

PROGNOSIS.—The prognosis of intra-cranial syphilis is said to be moderately favorable. The early stages of syphilitic meningitis, and sometimes even gummata, may be favorably influenced, or even cured. Of this, however, we have no decided proof, and in the more advanced cases, or where endarteritis exists, the prognosis is unfavorable, as no known remedies appear to have much influence on the secondary changes in the arteries.

TREATMENT.—The treatment should be with large doses of iodide of potassium, usually combined in the beginning with mercury. For a child two or three years old the initial dose of the iodide may be 0.3 gramme (5 grains) three times daily, gradually increased to 0.6 gramme (10 grains) unless gastric disturbance occurs.

IDIOCY.—By the term idiocy is meant a condition of marked mental deficiency. This mental deficiency may be of different grades.

PATHOLOGY.—Idiocy is, as a rule, the result of imperfect or impeded brain development, or it may be caused by actual destruction of portions of the brain. This condition may be produced by (1) traumatism, (2) non-traumatic inflammation, and (3) mechanical pressure.

(1) Traumatism acts usually by causing hemorrhage or destruction of the cerebral tissue in other ways.

(2) The most common form of inflammation causing idiocy is a more or less diffuse encephalitis, which ends in sclerosis and meningo-encephalitis.

(3) Hydrocephalus appears to cause or to accompany certain cases of idiocy. In some of these cases the distended ventricles cause atrophy of the cerebral tissue by pressure, while probably the distention of the ventricles is sometimes secondary. How far the degenerative conditions are primary and how far they follow pre-existing inflammations is at present unsettled.

The result of these pathological conditions is usually atrophy. This atrophy may be of intra- or extra-uterine origin, and may be local or general.

SYMPTOMS.—The symptoms of idiocy vary according as the individual represents a high or a low grade of this condition. An idiot may have a large head from hydrocephalus, or he may have a small head from cerebral non-development or from cerebral atrophy. Again, idiots may have normally developed crania both as to size and as to shape. In the lower grades there is often some physical malformation in connection with the mental impairment. In the more severe cases of idiocy there is considerable incoördination of the limbs, and the movements of the child are awkward and irregular. In many cases the speech is almost unintelligible. The idiot does not take notice of surrounding objects as does the normal child, and even when the sight and hearing are perfectly normal the impressions made on the senses are deadened. Epileptiform convulsions very commonly accompany idiocy, and play a most important part in the general condition of the patient.

The symptoms which are usually met with, and which enable us to gnosticate a pronounced case of idiocy, are the vacant expression, the casional presence of strabismus, the drooping head, the drooling, and the k of all idea of cleanliness. The teeth are usually decayed. Sometimes e child is so limp that he is unable to bear his weight at all, or will stand ld by his parent's hands, with his feet far apart, his knees bent, and his unk leaning forward. The whole body sways to and fro with an oscil-ting movement and absence of equilibrium. When able to walk alone he lks in a staggering, uncertain way, and falls easily. In many cases, wever, the child cannot even sit up alone. The muscles of the neck are ten so weak that the head falls over on one shoulder or forward on his est. The vertebral column fails to support the trunk and bends to a arked degree, and all the muscles are feeble and comparatively useless. ack of the power of attention and lack of memory exist in all cases, and n the higher grades are often the most prominent symptoms.

DIAGNOSIS.—We should be careful in very young children not to confuse slow or retarded mental development with idiocy. There is so much variation in the time at which children walk and talk, that a delayed development of these functions must not be considered to represent a condition of mental impairment. Some children develop so slowly, both bodily and mentally, that they appear very backward in comparison with others of the same age. Children in the first year of their lives may be so seriously affected by some grave disease that their development is prevented from advancing normally, and in comparison with other children of the same age they may be far below the usual grade of intelligence. If, however, we xamine this class of cases carefully, we see that, although they are very ackward in their development, they are gradually developing, and that hey do not represent the condition of complete arrest of development which xists in idiots.

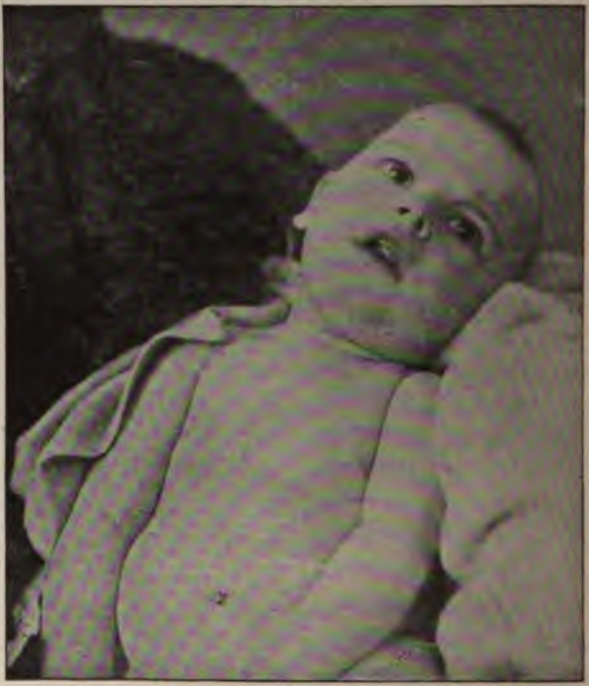
It is well to remember that in rhachitis we are apt to have not only tarded mental development but a weakness of the extremities simulating aralysis. When both these conditions occur, such cases may sometimes be istaken for idiots.

TREATMENT.—The treatment of idiots is essentially comprised under e question of their education. The education of this class of cases should begun early, usually from the fourth to the sixth year. Much can be ne to improve the various defects which exist in each individual. He can ually be taught to coördinate his movements, and by attending to his neral health his physical condition can often be much improved. In any cases if convulsions are present they can be more or less controlled. alformations or paralyses can be treated with benefit by apparatus or by eration. The best results in these cases will be attained by placing the ildren in institutions devoted to the training of idiots. Parents can be ld that the association of their children with others who are feeble-minded not a disadvantage, while it is often a great disadvantage for the children sound mind in a family to be associated with one who is idiotic. In the large majority of cases, however, they will always have to be supervised during their lives, and, in most instances, after they have advanced to a certain point they are liable to retrograde.

I have here a feeble-minded or idiotic child (Case 307), three years old.

I shall first call your attention to the child's peculiar vacant expression, and to the fact that it behaves more like an infant than like a child. Its mental does not correspond to its physical development, for it is able to walk and to use its arms and hands freely. This child, however, was not able to support its head alone during the first year of its life, and did not learn to walk until very lately. You see that there is no especially unnatural shape to its head, which has the circumference which would be normal for a child of this age.

CASE 307.



Idiocy.

This child presents the usual variations in temper which are so common in idiots. In the very severe grades the temper is apt to be happy and quiet, while in this grade, where the physical development has not been so much interfered with, we find that explosions of temper are quite frequent. The child is not able to feed itself, and, although it will probably develop into an individual of fair strength, we can have but little hope of any improvement in its mental condition. You will notice that it drools continuously.

I shall not attempt to describe the various forms of idiocy, such as are produced by hydrocephalus, cretinism, epilepsy, syphilis, acute febrile diseases, traumata, and other causes, but shall simply mention a peculiar class which is represented by microcephalus.

MICROCEPHALUS.—When the head is under a certain size it is called microcephalic. The size which is usually accepted as representing a microephalic head is from 40.5 to 43 cm. (16 to 17 inches). According to Broca, icrocephalus exists where the brain weighs 1049 grammes (35 ounces) in he male, and 907 grammes (30 ounces) in the female. It is generally conidered that this microcephalic condition is due to a lack of intra-cranial ressure. Together with the lack of development of the cranial bones here exists in these cases a lack of development or atrophy of the brain, hich may be considered either as the cause of the lack of intra-cranial ressure or, as is still believed by some writers, as the result of the external ressure caused by a premature synostosis. Microcephalic children are eeble-minded and usually present the symptoms of a somewhat low grade f idiocy. They not infrequently show signs of want of power of the imbs. This child which I have here is an instance of this kind.

She (Case 308) is three and a half years old, and is the eldest of three children. Her parents are healthy, as are the other children. She has never spoken. She can feed herself, and she walked when she was two and a half years old. She has incontinence of urine. She has never learned anything, has a violent temper, and sometimes has nervous attacks, which are probably of an epileptiform nature. The cranium is normal in shape, except that the forehead is very narrow, with a median vertical broad ridge. The fontanelles are closed and show no depression. There are no marked prominences about the skull. She is decidedly feeble-minded, and her attention cannot be attracted or fixed readily. The eyes are apparently normal, and her teeth are in good condition. There is a condition of paresis and incoördination, but the sensation is normal. The chest measures 49.5 cm. (19½ inches), and the head 43 cm. (17 inches).

I show her to you merely on account of the small size of the head in comparison with the hydrocephalic heads of which I have already spoken.

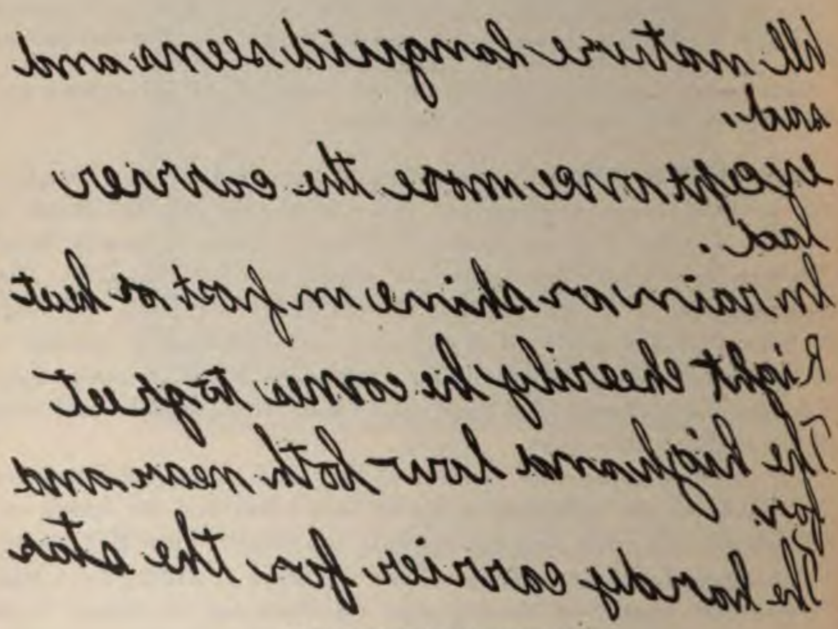
MIRROR WRITING.—An unusual and somewhat striking symptom which at times occurs in severe and, as a rule, chronic cerebral disease is one which is called "mirror writing." This symptom is usually found where there is cerebral degeneration or among the feeble-minded. The actual pathology of the affection has not yet been determined. Through the kindness of Dr. Acker, of Washington, I am enabled to describe to you two cases (Cases 309, 310) of this kind which I had an opportunity of examining with him, and I shall quote freely from what he said after carefully studying these cases.

The condition represented by cases of this kind is designated "mirror writing" because the individual writes in such a way that the letters can be deciphered only when they are reflected in a mirror, when they assume the appearance of ordinary writing. These specimens of writing are similar to those which appear on blotting-paper on which the impression of an ordinary specimen of writing has been taken. The affection is usually found among left-handed children and in adults after right-handed paralysis. There seems to be a physiological tendency for left-handed children to fall into the habit of "mirror writing." The tendency of the left hand to write n this way is, according to Erlenmeyer, due to the fact that it is easier to se the arms in a centrifugal direction, the left from the right and the right rom the left. Leonardo da Vinci was a noticeable example of this affection. The earliest recorded case of "mirror writing" was in 1688, in an epileptic girl twenty-one years of age.

Dr. Acker's first case (Case 309) was a mulatto boy, ten years of age. He was born prematurely at about the eighth month. His father is a nervous man, and does not talk plainly, but is well educated. His mother has tuberculosis of the lungs. One maternal uncle was insane.

For the first few weeks of his life he was in a very feeble condition, but finally he became healthy and strong. Whenever he was slightly sick he would have convulsions. When he was two and a half years old he fell a distance of 420 cm. (14 feet) upon a bed of concrete. A deep wound in the frontal region was caused by the accident, but there was no fracture. He did not lose consciousness, and immediately after the fall responded intelligently to any questions that were put to him, but he did not cry even when the stitches were put in the cut. From the time of the accident the convulsions became more severe and more frequent. Three years ago he began to have chorea. His intelligence is about the same as that of the average child. At one time it seemed as though he would develop into a kleptomaniac, but at present he shows this disposition at intervals only. He is of a mild and docile temperament, has very little to say, and responds usually by a nod of the head. He is naturally left-handed, and his first attempts at writing resulted in this form of mirror writing. He has also been taught to use his right hand, and he now writes with equal dexterity in two ways with each hand.

FIG. 96.

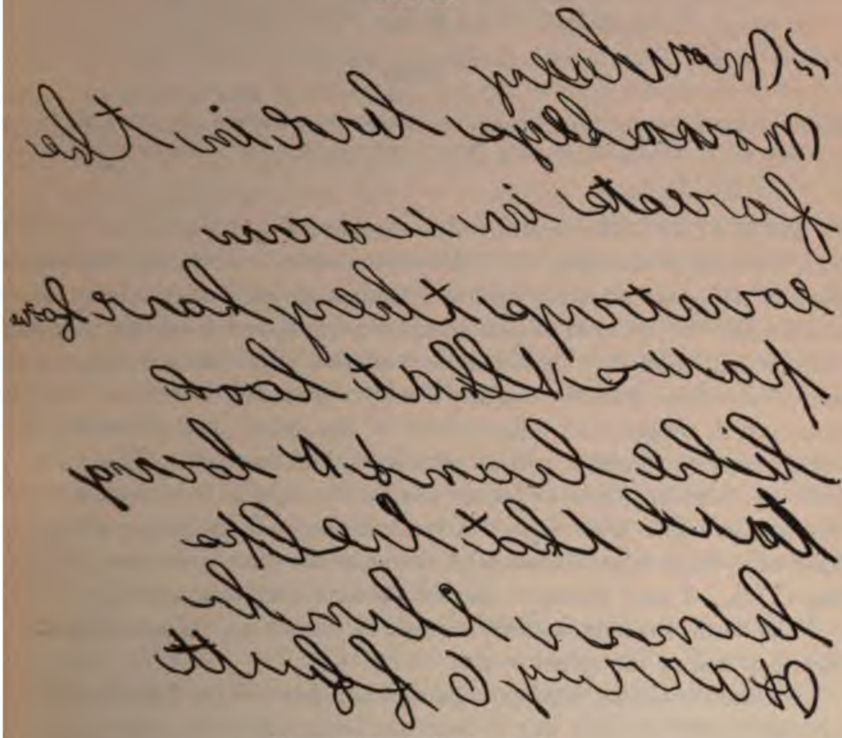


Mirror writing of a boy 10 years old.

Here is a specimen (Fig. 96) of this boy's writing, and if you will hold it in front of the mirror you will see that it represents a child's writing, the upper line being "All nature languid seems and sad."

The next case (Case 310) was a colored boy, nine years old. His father and mother were healthy, but of a low order of intelligence. He had two sisters who were fairly intelligent, and a brother eighteen years old who was idiotic. The boy himself was not bright, and his mother could not trust him away from home. He did not talk plainly. He had onvulsions during the first year of his life, but was considered to be in fair health. He had lways been left-handed, and writes "mirror writing" only.

FIG. 97.



Mirror writing of a boy 9 years old.

Here is a specimen (Fig. 97) which represents some very poor writing of this boy's. The upper lines are "Monkeys live in the forests in warm countries."

LECTURE XXXII.

CORD.

MYELITIS.—POLIOMYELITIS ANTERIOR.—PARALYSIS CAUSED BY CARIES OF THE SPINE.—HEREDITARY ATAXIA (FRIEDREICH'S DISEASE).—LOCOMOTOR ATAXIA.—SYRINGOMYELIA.

MYELITIS.—The term myelitis denotes an inflammation of the spinal cord, whether of the gray or of the white matter. *Acute myelitis* has been used to designate an acute diffuse inflammation of both the gray and the white matter of the cord of non-traumatic origin, and is an affection almost unknown in children. Considerable confusion still exists in regard to the use of the term *transverse myelitis*, which from its derivation should be employed to designate an inflammation of the spinal cord extending transversely over the greater portion of a section of the cord. This term has, however, been employed to denote the results arising from compression of the cord, whether from injury or from caries or from tumor, although in these cases there exists considerable doubt as to whether any true inflammation exists. I shall therefore discard the term transverse myelitis.

The term *meningo-myelitis* is used to denote an inflammation of the meninges and of the spinal cord.

As *acute myelitis*, *meningo-myelitis*, and *hemorrhage* into the cord are extremely rare in early life, it does not come within my province to discuss them. I shall therefore begin by speaking of the form of myelitis represented by poliomyelitis anterior.

POLIOMYELITIS ANTERIOR.—The most frequent and therefore the most important disease which affects the spinal cord with a resulting paralysis in infancy and early childhood is called poliomyelitis anterior. This disease occurs most commonly in the first three years of life. It is rare in the first six months of life. It may occur in later childhood, and, very rarely, in adults. It is met with more commonly than cerebral paralysis.

The disease may be primary, in which case it is without any known cause; or it may be apparently secondary to other diseases, such as the acute exanthemata and erysipelas. Traumatism appears to be occasionally a cause of the disease. Most of the cases occur during the summer months.

PATHOLOGY.—The pathological condition which occurs in poliomyelitis anterior is now considered to be an acute inflammation of the cells of the anterior cornua of the spinal cord, with a resulting degeneration and atrophy of these cells. This condition may be confined to the anterior cornua, but in some cases it may involve the spinal meninges somewhat. So few postmortem examinations of the early lesions connected with this disease have been made that we are dependent for our knowledge of it mostly on cases hich have been examined a number of months or years after the produc- on of the initial lesion. These later pathological conditions are, however, uite characteristic. The circumference of the limb grows small in com- arison with that of the opposite one, the result of an active muscular asting and of retarded growth. The bones of the affected limbs are often orter than those of the other side, sometimes even to the extent of several iches. In certain cases, however, the lengths of the bones seem to be but ttle affected, though the atrophy of the muscles may be very marked. The nterior cornua of the region affected, which is usually in either the cervical r the lumbar enlargement, are found to be greatly atrophied and many of he large motor cells to have been destroyed. According to Osler, the ffected half of the cord may be considerably smaller than the other, and he anterior lateral column may show slight sclerotic changes, chiefly in the yramidal tract. Accompanying this condition the corresponding anterior erve-roots are found to be atrophied, and the muscles connected with the egion of the cord which is affected atrophy and gradually undergo a fatty nd sclerotic change.

SYMPTOMS.—The onset of the disease in the great majority of cases is cute. Its course is chronic. In the acute form the onset may be preceded or some days by fever and restlessness, but it is very apt to appear sud- lenly, with, at times, convulsions which, as a rule, are of a milder type than hose which occur in cerebral paralysis.

In addition to the cases which are manifestly acute in their origin there ave been mentioned certain subacute and chronic cases. There is some loubt, however, whether these cases do not originate in the same manner as hose which are called acute. The probability is that in most of the cases which appear not to have had an acute onset and in which the paralysis eems to develop slowly, the early acute onset has been overlooked. This ubacute variety of poliomyelitis anterior does not differ from the acute cases n any way in its symptoms, prognosis, diagnosis, and treatment.

Following the acute onset there are at times unconsciousness, lasting ometimes for a number of days, vomiting, general nervous disturbance of he bladder and intestines, and a variety of symptoms of nervous irritability which may represent the prodromata of a number of diseases. The temper- ture is seldom very high, 38.3° to 38.7° C. (101° to 102° F.); it may, how- ver, in certain cases be higher. At times there are no prodromata, but the aralysis is noticed in the morning after a night's rest, although on the vening before the child was seemingly perfectly well. The severity and ength of the prodromal symptoms are no indications of the gravity of the esion or of the prognosis as to recovery. Pain in the paralyzed limb is not n uncommon symptom, but occurs only very early in the disease. The isease is primarily a motor disturbance, sensation remaining intact. Cere- ral symptoms, if present, pass off rapidly with the appearance of the paral- sis. The paralysis is usually apt to affect more than one limb in the begin- ing, but, as a rule, soon becomes monoplegic. The leg is more frequently

It is always difficult to diagnosticate poliomyelitis in the initial stage of e disease. At that time pain and sensitiveness of the affected limb may present, and may lead us to suspect that the disease is rheumatism. The nvulsions and unconsciousness which may appear at this stage are so ten present in other diseases that they are not of much aid in making the agnosis of poliomyelitis anterior.

The most reliable test at our command for making the diagnosis of liomyelitis anterior is the electrical reaction, and when this can be obtained clearly characterizes the disease. The normal muscles react to both the radic and the galvanic current. In applying the galvanic current a quick uscular contraction is noticed both on the opening and on the closing the negative (cathodal) and of the positive (anodal) pole, but is greater hen the cathodal pole is closed. When the galvanic current is applied the muscles affected by poliomyelitis anterior, the contractions continue, ut are slower and less sharp, and the reverse of what takes place in normal uscles occurs. Thus, the anodal closure causes a contraction equal to r greater than that caused by the cathodal closure (reaction of degenertion). As the muscles recover there is first a return to the normal galvanic eaction and later to their normal faradic excitability. The diagnosis in oung children, however, by means of the galvanic current is practically seless except in the hands of an expert. The faradic excitability begins to liminish within a few days after the onset of the paralysis, and disappears ntirely from those muscles which are severely affected.

DIFFERENTIAL DIAGNOSIS.—Poliomyelitis anterior is most apt to be mistaken for cerebral paralysis, and can be best differentiated from that disease by means of the symptoms which I have already described, and which are represented in this table (Table 104).

TABLE 104.

	Cerebral Paralysis.	Poliomyelitis Anterior.
Pathology	Hemorrhage. Embolus. Thrombosis. Sclerosis. Atrophy. Porencephalia.	Inflammation of anterior cornua of cord.
Age	Under three years.	Under three years.
Onset	Acute febrile.	Acute febrile.
Motor disturbance	Paralysis. Most common form hemiplegia. Spastic rigidity. Spastic gait. All the muscles of a limb affected.	Paralysis. Most common form monoplegia. Flaccid. Groups of muscles in a limb affected, usually the extensors.
Contractures	Of all the muscles, especially the flexors and adductors.	Of the flexors in the calf.

TABLE 104.—*Continued.*

	Cerebral Paralysis.	Poliomyelitis Anterior.
Spasmodic movements .	Athetosis. Post-paralytic chorea. Epileptiform convulsions.	Absent. Convulsions may occur at the onset of the disease.
Sensation	Unaffected.	Unaffected.
Nutrition	Arrest of growth.	Tendency to extreme atrophy coming on early in the paralyzed limb.
Electrical reaction . . .	Normal.	Reaction of degeneration.
Tendon reflex	Exaggerated on the paralyzed side.	Absent.
Speech	Liable to be impaired.	Unimpaired.
Intelligence	Often impaired.	Normal.

Other affections which may be mistaken for poliomyelitis anterior are (1) the paralysis following multiple neuritis; (2) progressive muscular atrophy; (3) pseudo-hypertrophic muscular paralysis; (4) rhachitic pseudoparalysis; (5) scorbutus.

(1) The principal points by which multiple neuritis is to be distinguished from poliomyelitis anterior are (*a*) the symmetrical affection of the limbs in the former and tenderness over the nerve-trunks; (*b*) the atrophy in multiple neuritis is not so severe as in cases of poliomyelitis anterior; (*c*) the course of the disease is different, cases of multiple neuritis almost invariably recovering, while severe cases of poliomyelitis do not recover.

(2) Progressive muscular atrophy, to which I shall refer in a later lecture (page 763), is so rare an affection among children that it need only be alluded to. There have been a few cases, however, where this disease began in children in the legs, and the paralysis is to be distinguished from that of poliomyelitis by its gradual onset, by the galvanic reaction continuing normal, and by the faradic excitability usually remaining as long as there is any muscular substance left. In this disease, also, the reflexes are not lost until the muscular substance has disappeared.

(3) Pseudo-hypertrophic muscular paralysis in its early stage is not likely to be mistaken for poliomyelitis, for the absence of abnormal electrical reaction, the increase in the size of the muscles, and its gradual onset are distinguishing points; although in the later stages of this disease atrophy may occur, the history will then clearly differentiate the condition from poliomyelitis.

(4) In certain cases of rhachitis the power of using the legs is so much affected that the mistake is quite commonly made of supposing that these

ldren are affected by poliomyelitis anterior. The condition in rhachitic ldren is one of weakness and not of paralysis, and can be distinguished the normal electrical reaction of the muscles and the lack of pronounced phy.

(5) The pseudo-paralysis which is commonly seen in cases of scor- us is often mistaken for some organic disease of the central nervous tem, with its resulting paralysis. The differential diagnosis from polio- elitis anterior, however, is not difficult to make, for the involvement other joints in addition to those of the legs, the presence of pain tenderness to such a degree that the child cries whenever the limbs are hed, and the normal temperature of the skin clearly distinguish this dition from poliomyelitis, in which disease normal sensation, freedom n pain, and a cold feeling of the limb affected are found.

PROGNOSIS.—So far as a fatal issue is concerned, the prognosis is very orable. If death occurs it usually takes place at the end of one or two ks, and is the result of interference with respiration, which may be caused ere the paralysis is extensive. Where in the initial stage of the attack ebral symptoms are prominent and continue for some time, the prognosis rave.

A second attack of the disease is very rare, and when it occurs it usually nes a few days after the original attack, and manifests itself by an in- ase of the existing paralysis. The paralysis, as a rule, will not increase en it has been stationary for twenty-four hours. The tendency of liomyelitis is for a time to improve. Some of the limbs affected recover the first few days, but in those which remain affected longer perfect overy is rare. When there is no improvement after six or eight months probability is that entire recovery will never take place, though under per treatment a slight improvement may go on for years.

We must remember that, even when untreated, a case of poliomyelitis is y apt to improve for one or two months quite rapidly, then rather slowly two or three months, and then, after a stationary period, contractions, seness of the joints, and malpositions may begin to be evident and may rease indefinitely.

When proper treatment is carried out, the prognosis is much more favor- le, and the period of possible improvement can be extended for some ars. According to Bradford and Lovett, there is certainly no leg, how- r wasted and contracted, that is not amenable to some improvement by erative or mechanical treatment.

TREATMENT.—The treatment of poliomyelitis by means of drugs has duced such unsatisfactory results that it may be said to be useless. The y multiplicity of the remedies which have been experimented with proves ir inefficiency. It is doubtful whether any treatment by drugs can be neficial to a central lesion of this character. Although a number of reme- s have been recommended to be given in the onset of the attack, it is bable that none of them are of any especial benefit; though it is wise to see that the bowels are freely moved, and, if the attack has been ushered in by convulsions, to treat these symptomatically if they continue.

Although we know of no rational means of treating the primary lesion of poliomyelitis anterior, we know that the results of this lesion, as shown by paralysis of the muscles, are such that the paralysis should be treated at once. The indication is to combat the rapid atrophy which is part of the disease, and to prevent its increase, and its later results from proceeding to a degree which would interfere with subsequent repair. To accomplish this, the affected limb should be supported in a normal position and carefully guarded against the stretching of joints, ligaments, and muscles. In addition to this, gentle massage and the faradic current applied five or ten minutes at a time at least four or five times a week are indicated to keep the affected muscles in the best possible condition and to combat the atrophy which to a greater or less degree occurs. The regular application of heat is also found to be useful where the limb is cold. According to Bradford and Lovett, muscles are much less likely to contract and deformities thus less apt to result in properly supported limbs.

The later manifestations of club-foot and other deformities should be dealt with by the orthopædic surgeon.

It may be well to mention that I have adopted the name poliomyelitis anterior as best representing the disease as we now know it. It has been called with less reason by various names, such as *infantile paralysis*, *essential paralysis of children*, *acute atrophic spinal paralysis*, *myelitis of the anterior horns*, *myogenic paralysis*, *dental paralysis*, and *poliomyelitis anterior acuta.*

I have some cases here such as you will be likely to meet with in your practice, and I shall now examine them before you.

This little girl (Case 311, page 683) is nine years old.

She was perfectly well up to the time of an attack, which came on suddenly and without known cause. She is said to have fallen while she was playing, but no injury of the leg could be detected, though she was carefully examined under ether. The exact date of the attack is not known, but it was some time ago. Her general health is reported to have been very good, and she seems to be bright mentally. She is, as you see, well developed, and has a good color. Nothing abnormal has been detected on physical examination of the lungs, thorax, abdomen, or other organs. The pulse is regular and of good strength. The left leg shows considerable atrophy, being 4.37 cm. (1¾ inches) smaller than the right in the calf and 2.5 cm. (1 inch) in the thigh. The leg is somewhat cyanotic, and is cold to the touch. There is marked weakness of the muscles below the knee, especially the extensors of the foot and toes. When she is lying in bed the movements of the thigh are performed with some strength. On walking she rotates the leg outward, so that the foot is at right angles with the line of motion, and she drags the toes. The joints are freely movable. Nothing abnormal has been detected in connection with the spine, which presents the condition of a movable lateral curvature, due to the shortening of the affected leg.

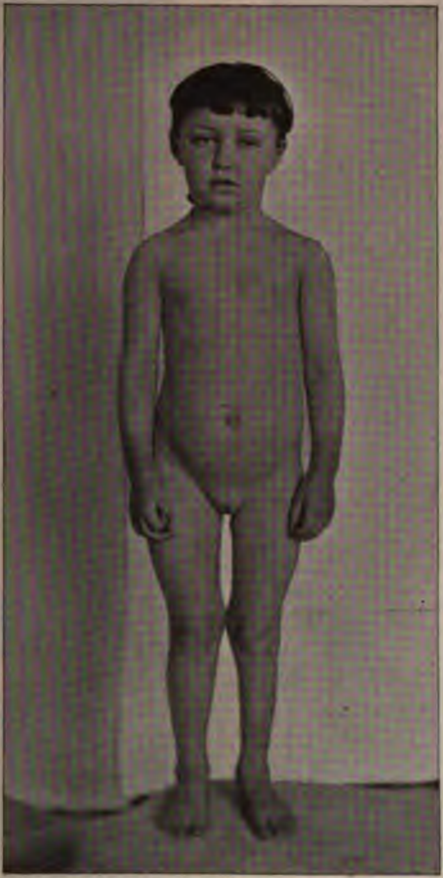
She is being treated by massage, electricity, applications of hot cloths twice daily for half an hour, and by apparatus.

She represents the condition of poliomyelitis anterior of the left leg, and, although she may receive some slight benefit from treatment, the probability is that she will always be lame.

is boy (Case 312) is twelve years old.

is said to have had rheumatic fever when he was thirteen months old. It was that he dragged his right leg when creeping, and this leg has evidently been affected ice he began to walk. The leg is atrophied, and there is a condition of valgus in t. There is slight permanent flexion in the knee, and the hip is also slightly flexed.

CASE 311.



Poliomyelitis anterior. Left leg. Female, 9 years old.

movements of the limb are otherwise good. The adductors are in good condition. bduction is chiefly accomplished by means of the tensor vaginæ femoris.

his case is evidently one of poliomyelitis anterior, with valgus of the right foot.

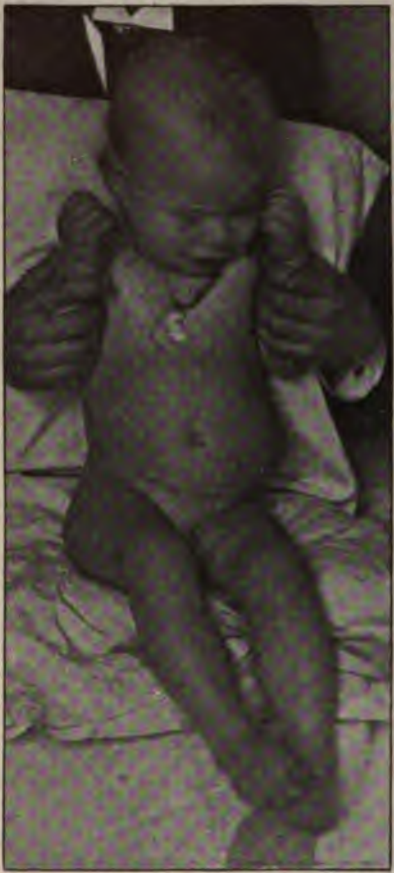
his infant (Case 313, page 684), twenty months old, is an unusual and interesting f infantile paralysis of the abdominal muscles.

e is stated to have always been healthy until five weeks ago, when on coming into the he vomited and two days later limped a little. When the infant cries you see that bdominal walls bulge, especially on the left side. The motion of the left leg is very ut slightly flaccid. The patellar reflex is absent, and he sits up very feebly.

his little girl (Case 314, page 684) is two and one-half years old, and presents the condition as the previous case.

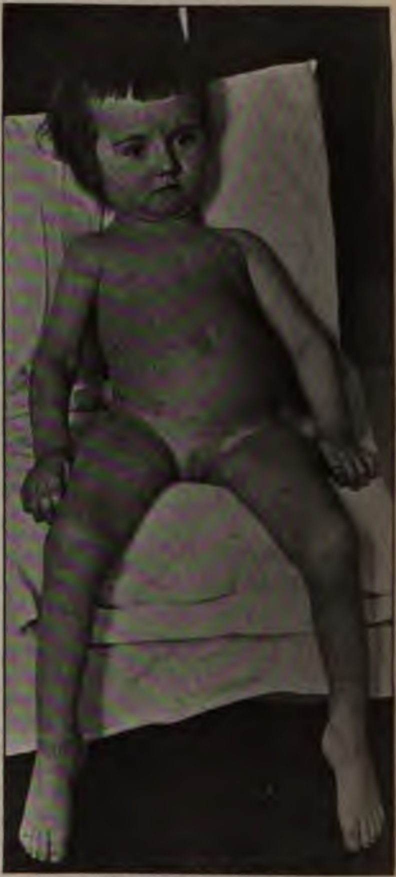
he has a sister who is said to have had an attack of poliomyelitis anterior when she n months old. No other history has been obtained about this case, except that she ell and strong until this attack, which occurred six weeks ago. The onset of the disease was sudden, and was accompanied by high fever, followed in three days by complete paralysis of the muscles of the upper and lower extremities of the body and of the head. The arms and head soon recovered. She is unable to sit up alone, and the abdominal muscles are paralyzed to such an extent on the left side that, as you see, they are flaccid, bulge out, and do not react normally. The left leg is perfectly flaccid. The knee-jerks are absent. The surface temperature is diminished, and there is atrophy of the legs.

CASE 313.



Poliomyelitis anterior. Abdominal muscles, left side. Male, 20 months old.

CASE 314.



Poliomyelitis anterior. Abdominal muscles, left side. Female, 2½ years old.

(Subsequent history.) Under treatment with electricity and massage, complete recovery took place.

The next case (Case 315, page 685) is that of a boy (I.), six and one-half years old, who was apparently healthy at birth, and who has never had any illness.

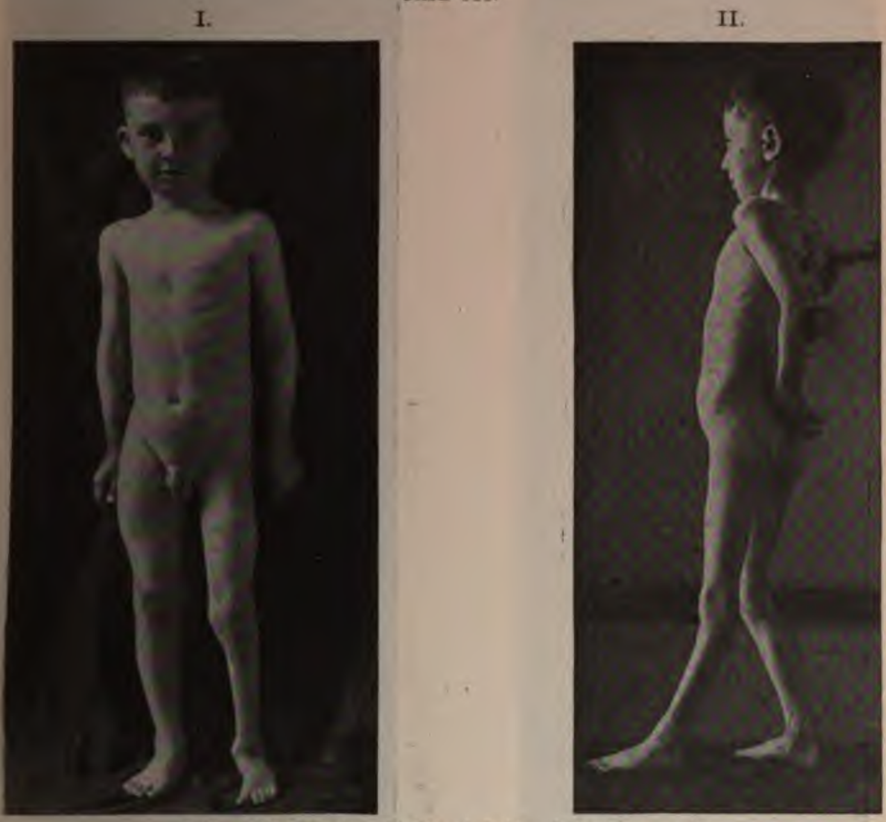
When he was one year old he was noticed to have some motor disturbance of the left leg. On examination of the leg the surface temperature is found to be diminished, the knee-jerk is absent, and there is an atrophy of 5 cm. (2 inches) of the thigh and 6.5 cm. (2½ inches) of the calf. There is also 3.7 cm. (1½ inches) shortening in the leg. The child walks, as you see (II.), with a marked limp of the left leg, and there is the condition of flail-joint in his left knee and ankle.

These symptoms, without any further history of the case, justify us in making a diagnosis of disease of the spinal cord rather than of the brain. This is a typical case of the appearances presented in the advanced stages of a severe case of poliomyelitis anterior.

This girl (Case 316, page 686), sixteen years old, represents very well the results which may occur from an attack of poliomyelitis anterior.

She is said to have had some disturbance in her left leg following a fall from a high chair when she was seven months old. She did not walk until she was eight years old, and has been lame ever since. She came under my observation at the hospital when she was thirteen years old, and at that time presented the evidences of a long-standing paralysis of spinal origin. The knee-jerk was absent. The left leg was cold and atrophied, and the foot was in the valgus position. She has, you see, a lateral curvature, due to paralysis of the muscles of one side of the back. She has shown only slight improvement under treatment for the last three years.

CASE 315.



Poliomyelitis anterior. Flail leg, left side. Male, 6½ years old.

Here is a little girl (Case 317, page 686), five years old, who, as you see, has paralysis of both legs.

She was well and strong until about her third year, when she had an attack of whooping-cough. During this attack she also had some other illness, which was characterized by fever and pain in the back. The loss of power of her legs dates from this time, and is said to have been gradual. She is fairly well developed, and the paralysis has affected both legs and thighs as well as the psoas and iliac muscles. There is marked atrophy, and the reflexes are absent.

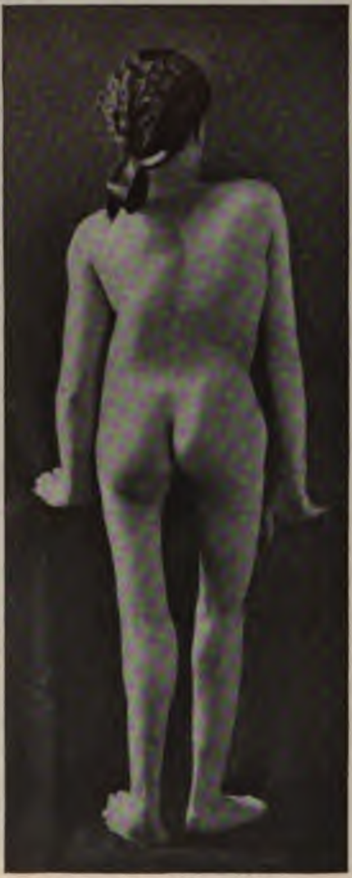
You notice in this case that the limbs are held apart and are flaccid. If the case were one of cerebral paralysis there would be in place of this flaccid condition a contraction of the adductors of the thigh, which would have been apt to hold the limbs closely together.

At times this contraction would perhaps be so strong as to prevent the limbs from being drawn apart.

The prognosis for complete recovery in this case is unfavorable.

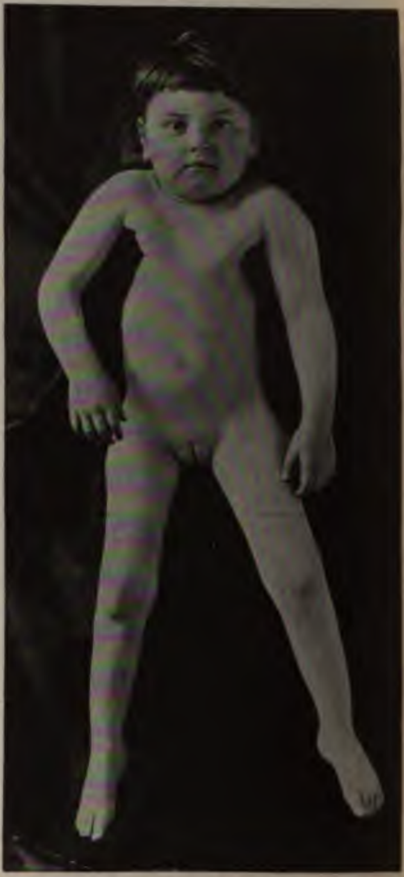
The treatment will be of a general nature, such as I have already explained to you should be adopted in cases of this class.

CASE 316.



Poliomyelitis anterior. Talipes varus. Lateral curvature. Female, 16 years old.

CASE 317.



Poliomyelitis anterior. Paralysis of both legs. Female, 5 years old.

This little girl (Case 318, page 687), five years old, is a case of poliomyelitis anterior which has affected the right leg.

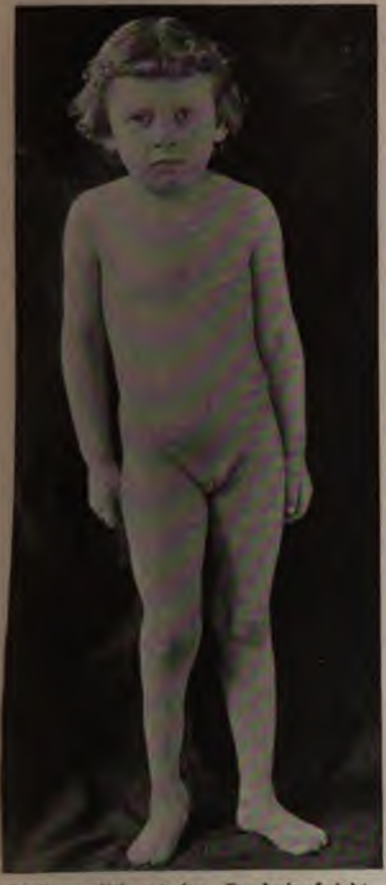
When she was three years old she fell from a step, and was seized with a sudden attack of paralysis of the right leg. A month later she walked with toe-drop of the right foot and slightly of the left. The skin of the limb is not especially cold or blue. The right thigh measures 24.1 cm. (about 9¼ inches), the left thigh 24.8 cm. (9¾ inches). The right calf measures 16.2 cm. (6½ inches), the left 17.5 cm. (7 inches). The patellar reflex is absent on the right side and very slight on the left. The right leg is 1.2 cm. (½ inch) shorter than the left.

Under the application of massage and the use of various mechanical apparatus there has been slight improvement.

This boy (Case 319, page 687), eleven and one-half years old, has a good family history, and is said to have been swung about by his feet when he was seven months old, to which the family attribute the present condition of his right foot.

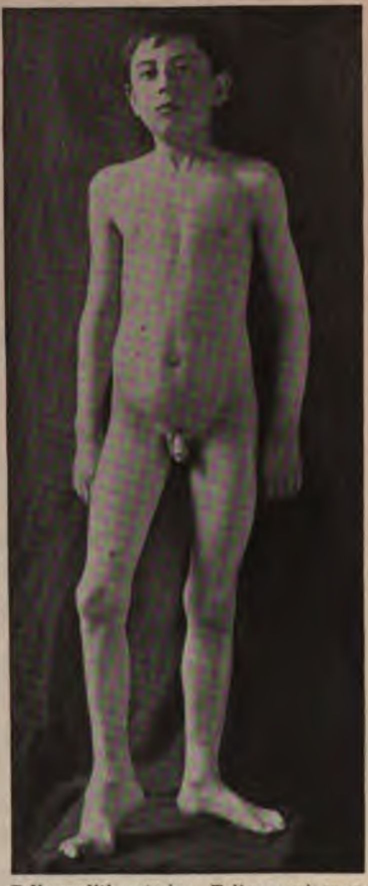
The anterior portion of the foot is flexed, as you see, at a sharp angle at the medio-tarsal joint. The foot can be easily bent to a right angle, but not beyond. Tense bands of plantar fascia can be felt when the foot is straightened out, but it can be brought into position by the use of considerable force. The length of the legs is equal. There is 1 cm. (about $\frac{3}{8}$ inch) atrophy in the right calf and 0.6 cm. ($\frac{1}{4}$ inch) of the right thigh.

CASE 318.



Poliomyelitis anterior. Paralysis of right leg. Female, 5 years old.

CASE 319.



Poliomyelitis anterior. Talipes equinus on right side. Male, 11½ years old.

He represents the condition of talipes equinus, the result of a contraction of the flexor muscles following an attack of infantile paralysis.

I have also here a boy (Case 320), twelve years old, who illustrates a case of poliomyelitis anterior secondary to erysipelas.

He had an attack of erysipelas when he was fourteen months old. The erysipelas lasted for about one month, and was followed by an attack of diarrhœa which lasted for six weeks. It was noticed that the infant was weak and had little power in the left leg about one week after the beginning of the erysipelas. After recovering from the diarrhœa he began to walk a little, but with a limp, which he has had ever since. The leg has since been growing smaller, and he has lately shown no improvement whatever. There has never been any pain in the leg. He walks with a decided limp, and the foot is brought to the floor with a slap. The knee bends backward beyond its proper position. The leg is much atrophied, the right thigh being 11.2 cm. (4½ inches) less than the left, and the leg 7.5 cm. (3 inches) less than the left leg. The leg and foot of the affected limb are slightly colder to the touch than those of the other. On raising the foot of the affected limb it is seen that hyperextension can be produced to an angle of 140°.

In this case I shall advise apparatus to prevent the further formation of flail-joint at the knee, which is evidently now present.

PARALYSIS CAUSED BY CARIES OF THE SPINE.—In cases of paralysis caused by caries of the spine the lesion is essentially a compression of the cord: this is usually slow in its progress, and it is doubtful whether in it a true inflammation occurs even in the beginning. The condition resulting from compression occurring in the course of caries of the spine may be found in any part of the cord. It is most frequently met with in disease of the dorsal region, though it may occur in the cervical and lumbar regions. In caries of the spine the compression of the cord is not often the result of pressure from the vertebræ, but usually is caused either by an abscess in the vicinity of the diseased vertebræ, or more commonly by a thickening of the meninges.

When the lesions of the cord are of any considerable extent, ascending and descending secondary degenerations follow after a time. If the process ceases, it leaves a certain amount of sclerosis of the cord at the seat of the disease. This may be very slight, or the cord may be considerably reduced in size and yet may transmit normal nervous influences.

SYMPTOMS.—The onset of the disease is sometimes quite sudden, but more frequently it is rather gradual. The symptoms vary according to the part of the cord which is affected.

When the lesion is in the dorsal or the lumbar region the onset is represented by numbness and weakness in the legs. This is quickly followed by a paralysis which may become complete in a short time.

If the lesion is below the level of the sixth dorsal vertebra, the legs alone are affected; if on a level with this point, the abdominal muscles are involved. Sensation up to nearly the level of the lesion may be diminished, or even lost entirely. In regions above the lumbar enlargement the reflex reactions are exaggerated and ankle-clonus soon appears.

When the disease affects the cervical enlargement, or any portion of the cord above, all the extremities are apt to be paralyzed. In severe cases there will be retention of urine, with subsequent incontinence. The bowels are usually constipated, but incontinence of fæces is sometimes present.

In lesions of the lumbar enlargement the knee-jerks will be lost. Trophic changes in the limbs are not marked, but the muscles are somewhat wasted, and rigidity may develop. Bed-sores are apt to form. The reaction of degeneration is not present.

The characteristic picture of lesions in the dorsal region caused by caries of the spine is paraplegia.

DIAGNOSIS.—The disease is to be differentiated from poliomyelitis anterior, in which disease monoplegia is more common than paraplegia, and in hich the reflexes are lost and the action of degeneration is present. In dition to this means of making a differential diagnosis, the absence of itial fever and prodromata, of disturbances of sensibility, of paralysis f the sphincters, and of a tendency to bed-sores in poliomyelitis anterior of great aid in differentiating it from the results of caries of the spine, here rigidity of the limbs, increased reflexes, and contractures are promi- ent symptoms.

The differential diagnosis from cerebral paralysis is more difficult, as e condition of the limbs is similar in both. The diagnosis is made by he absence of all cerebral symptoms, and by the presence of such special ymptoms as rigidity and prominence of the vertebræ in caries of the spine.

PROGNOSIS.—The prognosis in these cases is in general favorable. A ertain number of cases remain uncured, but nearly all recover under treat- ent, although the condition may persist for months.

TREATMENT.—The treatment of these cases is, as a rule, to be directed o the caries, and consists essentially in perfect rest on a rectangular bed- rame. Massage and electricity are sometimes of assistance when applied o the paralyzed limbs. Where no improvement occurs after several months, aminectomy must be considered; and there has been a case (Case 321) at he Children's Hospital where improvement followed this operation. In his instance an abscess was pressing upon the cord, and on its being dis- overed and emptied recovery took place. The operation was performed by r. H. L. Burrell.

HEREDITARY ATAXIA (Friedreich's Disease).—Hereditary ataxia a very rare disease. It is an organic affection of the spinal cord, usually ccurring in several members of a family and developing in later childhood. he names family ataxia and generic ataxia have also been used.

PATHOLOGY.—The pathology of the affection is a slow, progressive egeneration of the posterior and lateral columns of the cord.

SYMPTOMS.—The characteristics of this disease are its slow develop- ent, staggering gait, loss of muscular power, nystagmus, sometimes loss f knee-jerk, frequent disturbance of speech, and finally complete helpless- ess with mental impairment.

PROGNOSIS AND TREATMENT.—The prognosis of hereditary ataxia is ways unfavorable, and there is no known remedy which is of benefit.

LOCOMOTOR ATAXIA.—In connection with this degeneration of e posterior and lateral columns of the cord which occurs in hereditary axia, I shall merely mention the degeneration of the posterior columns of e cord (locomotor ataxia), as this disease is almost unknown in childhood. he disease as it occurs in children usually involves the lateral as well as e posterior columns of the cord, and is thus closely related to Friedreich's isease.

Locomotor ataxia is to be distinguished from multiple neuritis, which sometimes closely resembles, the pain, ataxia, and loss of knee-jerk often curring in both. The diagnosis from multiple neuritis is to be made

(1) the fact that it is not of hereditary origin, (2) the absenc
and of mental symptoms, and (3) the ataxic and shuffling ga

SYRINGOMYELIA.—As defined by Osler, syringom
matous new formation about the central canal of the spi
cavity formation. This disease is so rare in children that
state that it is now regarded as a gliosis, a development
neurogliar tissue in which hemorrhage or degeneration ta
the formation of cavities.

In this disease we usually find a diminution of sensati
cold, according to the site of the lesion, which is commonly
upper dorsal or the lower cervical region. There is apt to b
one or both arms, accompanied by marked wasting. There
some weakness in the legs. The reflexes are increased, an
dition is likely to result. These symptoms are usually a
marked lateral scoliosis.

In typical cases the diagnosis is easily made where the
trophic paralysis of one or both of the upper extremities, wi
tactile sensation and loss of thermic and painful sensation b
region, and a weakness of the lower extremities, with a ten
rigidity.

Syringomyelia is an incurable disease, and the treatme
usually limited to correcting, if possible, the lateral curvat
quently accompanies it.

LECTURE XXXIII.

BRAIN AND CORD.

ultiple Cerebro-Spinal Sclerosis.—Cerebro-Spinal Meningitis.

LTIPLE CEREBRO-SPINAL SCLEROSIS.—By multiple scle-
the brain and cord we mean a disease in which the nerve-elements of
areas in the brain and cord are more or less replaced by connective
The sclerosis which occurs in these cases, however, is not a distinc-
ion of multiple cerebro-spinal sclerosis, as it is the same that occurs
r sclerotic conditions of the nervous system. It is simply the
e distribution of these areas which is pathognomonic of the disease.
ease has also been described under the name of disseminated sclero-
ular sclerosis, focal sclerosis, herdsklerose, and sclérose en plaques.

OLOGY.—The etiology of the disease is obscure ; but heredity appears
ne of the causes of multiple sclerosis, and traumatism, shock, and
acute diseases, especially those of an infectious character, have an
cal significance.

HOLOGY.—Only a small number of autopsies of this disease have
ade in children.

characteristic feature of the disease by which it is distinguished from
lerotic diseases of the brain and cord is the erratic and multiple dis-
n of the sclerosis. The sclerotic patches may occur in the brain or
ord, or in both, and they are perfectly irregular as to the parts of
bro-spinal system which they involve. According to Osler, there is
ase in connective tissue of the sclerosed patches, and their fibres are
nd firmer than normal. The gradual growth destroys the medulla
erves, but the axis cylinders persist in a remarkable way.

PTOMS.—The onset of the disease may be rapid or slow, but is
kely to be rapid. According to Pritchard, the child is noticed, after
a blow on the head, or a shock or fright, or without any apparent
tremble. In some cases the disease may be ushered in by a convul-
In connection with the tremor, nystagmus may appear as an early
n, but, as a rule, it is a later one. The gait is usually affected early,
ements being clumsy or staggering. Among other early symptoms
nus and diplopia may be mentioned. Headache and vertigo are
y not infrequent, although in young children it is somewhat difficult
mine the presence of these symptoms. Exaggeration of the reflexes
lepend upon the location of the lesion is an early symptom in some
nd may be associated with ankle-clonus. The later symptoms are
nce of speech, mental weakness, slow muscular wasting, and paralysis
xtremities.

DIAGNOSIS.—The differential diagnosis of multiple cerebro-spinal sclerosis is to be made chiefly from hereditary ataxia, as there is no other disease of the nervous system occurring in children which especially simulates it. Although in both diseases ataxia, nystagmus, and defects of speech occur, and although tremor is a common symptom of both, yet these symptoms differ somewhat in their form.

According to Pritchard, *tremor* is a common symptom in multiple cerebro-spinal sclerosis in children, while in hereditary ataxia it occurs in only a certain proportion of cases; in the former disease it is of the voluntary type, in the latter it is of the choreiform variety. Again, in hereditary ataxia the affection of the *speech* occurs, as a rule, later than in sclerosis. On the other hand, *ataxia* of the extremities is less constant in sclerosis than in hereditary ataxia, and the *inability to stand* with the feet together and the eyes closed, while common in hereditary ataxia, is rarely observed in sclerosis. Various paræsthesiæ which not infrequently occur in hereditary ataxia, especially the *girdle sensation*, are not common in children affected with sclerosis.

In addition to these other clinical differences there are three symptoms which afford a marked contrast in the two diseases. These are (1) the condition of the reflexes, especially that of the patellar tendon; (2) the mental state; and (3) the tendency to convulsive seizures. In multiple sclerosis the knee-jerk is commonly exaggerated and rarely abolished, while in hereditary ataxia it is often abolished. The mental condition is commonly dulled at some stage of the disease in multiple sclerosis, and is usually in the form of a simple dementia. In hereditary ataxia, on the contrary, the intellect is unimpaired, mental weakness being exceptional. Convulsions are quite common in sclerosis and are rare in hereditary ataxia.

A differential diagnosis should also be made from chorea, which can be eliminated readily by the absence of tremor, by the presence of incoördinate movements, and by the absence of nystagmus and of true ataxia.

PROGNOSIS.—The prognosis in multiple cerebro-spinal sclerosis for permanent recovery is very unfavorable. The disease may be arrested temporarily, but improvement in the general condition of the child, as a rule, merely marks a remission. The child rapidly becomes so helpless that there is a corresponding liability to complications and to death.

TREATMENT.—There is no drug which appears to have any effect upon the disease, the treatment being wholly symptomatic. The general health of the child should be carefully attended to, and in this way the inroads of the disease on the nervous system can be combated.

CEREBRO-SPINAL MENINGITIS.—By cerebro-spinal meningitis we mean an acute infectious disease characterized by a leptomeningitis of the brain and spinal cord.

Although this disease is usually classed under the infectious fevers, it seems to me to be associated more naturally with diseases of the nervous system, because the main pathological lesions are found in the brain and in spinal cord. However closely it may in the future be proved to be ociated with other diseases, such as pneumonia, and however firmly we y believe that its cause is a microbe as in the other diseases of the infec- s class, still the salient symptoms by which we can make our diagnosis those produced by central organic nervous lesions. The disease does not ear to be contagious. It may be acute or chronic. It may occur as a mary disease or in connection with some other infectious disease, such as te lobar pneumonia.

ETIOLOGY.—Cerebro-spinal meningitis at times occurs in wide-spread lemics. It is also met with in a sporadic form. Although there has not been made a sufficient study of the epidemic form of the disease to allow to state much that is definite about its causation, it is probable that it is same as in the sporadic form. From the sporadic cases which have n carefully studied it is evident that certain bacteria are the cause of the ease. The most common organism which has been found is the pneu- coccus of Fraenkel, but the streptococcus and staphylococcus pyogenes reus have also been found in a few cases. No distinction except a bac- iological one can be made between the cases in which these bacteria are nd; nor can any be made between the epidemic and the sporadic cases, they have the same symptoms. Although there is supposed to be a se connection between cerebro-spinal meningitis and pneumonia, yet the rmer disease is frequently found without the lesions of pneumonia being esent.

PATHOLOGY.—The pathological lesions which represent this microbic rm of cerebro-spinal disease are chiefly an inflammation of the pia mater, ith its accompanying serous, fibrinous, or purulent exudation. The brain d cord may be involved. Foci of hemorrhage and of encephalitis are metimes found. The prominent primary lesion which produces the pical, uncomplicated picture of the acute variety of cerebro-spinal menin- tis in its early stage is a leptomeningitis, and the disease can well be oked upon as a *microbic leptomeningitis*.

As has been well stated by Delafield and Prudden, the degree of the sion in the brain varies greatly, depending upon the period at which death curs. At times, when death occurs early in the disease, no macroscopic ange will be evident. Microscopic examination in these cases, however, ows a moderate degree of extravasation of leucocytes in the vicinity of e vessels. In well-marked cases the pia mater and the cord are more or s densely infiltrated with serum, fibrin, and pus. This pathological con- tion may be found over the convexity and base of the brain, and is fre- ently most marked in the latter situation. In the cord the infiltration y occur over the anterior and posterior surface, and in some cases, prob- ly owing to the recumbent position of the patient, it is most marked on posterior surface. The ventricles of the brain and the central canal of cord may contain turbid serum mingled with pus-cells and sometimes od-cells. The membranes and underlying nervous tissue may be hyperæmic and the seat of capillary hemorrhages. In protracted cases the ventricles may be dilated with serum.

In addition to these characteristic lesions of the disease, there are a number of secondary changes in different parts of the body, which are not constant, but which occur with sufficient frequency to warrant their mention. Thus, there may be subserous punctate hemorrhage in the endocardium; petechiæ in the skin; hyaline and granular degeneration in the voluntary striated muscle; occasional multiple abscesses in various parts of the body; suppurative inflammation of the joints; parenchymatous degeneration of the heart, liver, and kidneys; swelling of the gastro-enteric lymphatic system, and metastatic choroiditis.

The lesions are essentially the same in the epidemic and sporadic cases of acute cerebro-spinal meningitis.

In the form which from its length may be called chronic the pathology has not been determined, as a sufficient number of autopsies of this variety has not yet been obtained. It is, however, possible that the various later symptoms of organic central disease which occur in some of these cases, and especially in those which do not recover, may be produced by the lesions of hydrocephalus and cerebral abscess.

SYMPTOMS.—The disease is usually sudden in its onset, attacking at times perfectly healthy children. The prominent symptoms are intense headache, photophobia, and at times convulsions, pain, hyperæsthesia, vomiting, delirium, and, later, coma; also sensitiveness to sound and to touch. Tenderness on pressure over some portion of the vertebral column is found not uncommonly. The temperature in the more severe cases is high, 40.6° to 41.1° C. (105° to 106° F.); usually, however, it is 38.3° to 38.9° C. (101° to 102° F.). There is no regular temperature curve; in fact, the symptoms, temperature, pulse, and respiration vary in different cases. The pulse is usually quick; the respirations are rhythmical, but somewhat quickened. The bowels are usually constipated.

Strabismus is a common symptom, and rigidity and retraction of the neck and back (opisthotonos) are soon noticed. The knees are usually drawn up. The child emaciates rapidly. The pupils are altered. It is not uncommon to find metastatic choroiditis with exudation of pus into the vitreous (Wadsworth). There is often bilateral loss of hearing. Remissions in the symptoms are frequent. A *tache cérébrale* can at times be found. The spleen, especially in acute cases, is enlarged. If the brain and cord are also decidedly involved, symptoms corresponding to the locality and degree of the lesion appear. This is especially noticeable in the chronic form, where the disease has lasted for some months. Pneumonia, arthritis, pleuritis, and pericarditis may arise as complications.

DIAGNOSIS.—The prominent symptoms on which you must rely in differentiating cerebro-spinal meningitis from tubercular meningitis, for which it would be most likely to be mistaken, are the sudden onset, extreme headache and hyperæsthesia, opisthotonos, herpes, and regular pulse in the bro-spinal disease as compared with the usually slower progress and der symptoms of the tubercular. In some cases the onset is not so den, and difficulties have arisen in the differentiation from typhoid fever pneumonia; but, except in the rather rare meningeal types of these latter ases, the diagnosis will in a few days become clear.

In young infants the symptoms of cerebro-spinal meningitis may be ely a heightened temperature with clonic convulsions, so that the diag- is cannot be made during life from the various forms of reflex convul- ns which may occur at this age, and cerebro-spinal meningitis can only suspected. A case illustrating this fact was seen by me in consultation h Dr. W. L. Richardson.

A male infant (Case 322), healthy at birth, was suddenly attacked when it was six s old with general clonic convulsions, accompanied by a temperature of 40° C. (104° F.) the first twelve hours, and afterwards to the time of its death by a temperature of 8° C. (102° F.). The attack followed a period of nervous excitement in the mother, o was nursing it, and who in other respects showed no abnormal symptoms. There were symptoms of cerebro-spinal meningitis, such as retraction of the head or opisthotonos, d in the intervals between the convulsions, which occurred about every hour, the infant med perfectly well. It did not vomit, and did not have any abnormal symptoms con- cted with the eyes. The convulsions, which constituted the only symptom, continued, d on the second day of the attack the infant died suddenly.

The report of the autopsy, made by Dr. Whitney eighteen hours after death, was as lows:

Rigor mortis well marked. Lividity of the dependent parts of the body, and in small parated patches over the arms, legs, and chest.

The calvaria presented nothing abnormal. The inner surface of the dura was covered th opaque yellowish patches of lymph, especially marked over the base of the skull. The sels of the pia mater were markedly injected, and its meshes were filled with an opaque enish-yellow exudation. This exudation extended over the entire brain and into the inal canal. Microscopic examination showed the presence of large micrococci, usually ociated in pairs, two of which were sometimes united with a chain of four (pneumococcus).

The heart was normal, and its cavities were filled with dark fluid blood.

The lungs were not fully retracted, and were engorged with dark blood, which was so undant as to suggest extravasation into the alveoli. The pleural surfaces were perfectly ooth.

The abdominal organs—spleen, liver, and kidneys—were markedly injected with blood, t were otherwise normal.

The stomach and intestines presented nothing abnormal.

The pathological diagnosis was, acute purulent cerebro-spinal meningitis, and general nous congestion.

PROGNOSIS.—The prognosis, where the child is young and the onset is olent, with high temperature and continuous convulsions, is very serious; it, even in the apparently fatal cases where coma has intervened, a change ay take place and the child recover. The first two weeks are usually the itical periods, so far as the acute form of the disease is concerned. The sease varies in duration, sometimes lasting for only a few days, in other ses for a number of weeks; but in some cases it lasts for months, when it nstitutes the chronic form, which is apt to prove fatal, both from exhaus- n and from the development of more serious central nervous lesions.

TREATMENT.—The treatment of cerebro-spinal meningitis varies according to the severity of the symptoms. In most cases sedatives, such as the bromides, are indicated, and where the pain is severe opium in considerable doses is often needed. The ice-bag or Leiter's coil applied to the head, and absolute quiet in a darkened room, are important adjuvants to the treatment. In many cases the pulse becomes so weak and the prostration so marked that stimulants are needed until convalescence is established, when they can usually be replaced by tonics. In some cases the hyperæsthesia and general sensitiveness to noise, light, and motion in the room are so extreme and so characteristic that the attendants should be cautioned not to touch the child or the bed unnecessarily, and absolute quiet should be enforced in the room and throughout the house.

I have already told you that, as a rule, cerebro-spinal meningitis in children is a disease which is characterized by acute onset. This case, which I take from my notes, is illustrative of this fact:

A boy (Case 323), thirteen years old, had never had any especial diseases, but had been rather delicate for a number of months. He went to a Christmas party on December 25, and on returning from the party complained of the motion of the sleigh in which he rode home. On the following day, in the afternoon, he was found to be listless, to have his tongue coated but not dry, and to have a temperature of 40.5° C. (105° F.) and a pulse of 140. He complained of tenderness and pain in the back of his neck; there was also tenderness in the abdomen. He appeared to be somewhat dull.

On the following day the temperature in the morning was 39.4° C. (103° F.), and the pulse was 120. He was much more dull and apathetic than on the previous day, and in the afternoon became delirious. In the evening he had involuntary passages of urine and loose discharges from the bowels. His temperature was 40° C. (104° F.).

On the following day his temperature was 39.4° C. (103° F.), and the respirations varied from 40 to 80 and were regular. He was unconscious. Subsultus tendinum was present. There was retraction of the head. The pupils did not respond to light, but were equal in size. A *tache cérébrale* was present.

On the evening of the following day, four days from the onset of the disease, he died.

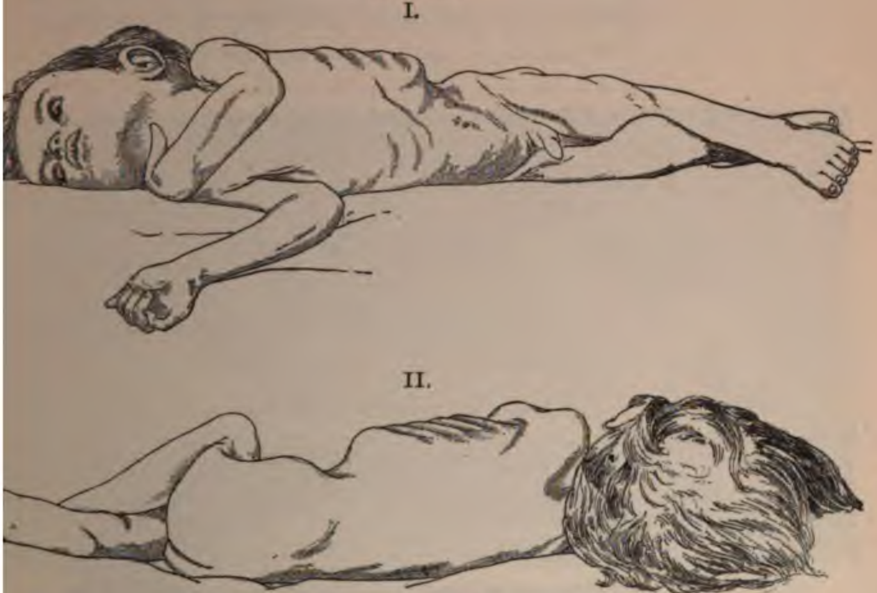
The autopsy made by Dr. Gannett showed the convex surface of the entire brain and cord to be covered with a thick exudation of pus, the spleen to be enlarged, and the case to be one of acute cerebro-spinal meningitis.

I have here in the wards a child (Case 324, page 697), two years old, who was brought to the hospital on the 21st of the month with the history that it had been showing symptoms of malaise for six weeks. Two weeks previous to entering the hospital it had a convulsion, and the indefinite and general symptoms had become more pronounced. There had been loss of appetite, with constipation; at times vomiting, slight cough, and a heightened temperature.

You see on examining the child the position which it assumes in bed. The head is retracted, and the muscles of the neck are rigid. The eyes are staring, but the pupils react to light. There is at times, though not now present, slight opisthotonos. On examining the child in front (I.), you see that the abdomen is retracted. Looking at it from behind (II.), you see that the occiput touches the back of the neck, and that the emaciation is extreme, so that the vertebræ and ribs have become quite prominent. The child is apparently unconscious, and does not notice objects which are brought before its eyes, although the eyes are open. It moans at times, and sometimes the legs are drawn up. No efflorescence has been detected anywhere on the skin. Although, as I have already told you, the onset of cerebro-spinal meningitis may be acute, yet in certain cases the prodromal symptoms are of a subacute character and somewhat prolonged, as has occurred in this case, which seems be one of this disease. It has been in the hospital for seven days, which would make time since it was first noticed to be sick seven weeks. Since entering the hospital temperature has varied from 36.6° to 38° C. (98° to 100.5° F.). At intervals it has mited and has apparently been unconscious. Sometimes it has cried out sharply, as ugh in pain. A *tache cérébrale* has been found at times, and the retraction of the head been almost continuous.

The continuous retraction of the head, with at times opisthotonos and unconsciousness thout the serious cerebral symptoms which after the fourth or fifth week would accomny an attack of tubercular meningitis, and the absence of any symptoms which point

Case 324.



Cerebro-spinal meningitis. Male, 2 years old.

wards disease of the thoracic or abdominal organs, lead me to make the provisional diagsis in this case of cerebro-spinal meningitis. From what I have told you in a previous ture in speaking of tubercular meningitis, especially of the recurrent form, an instance ase 272) of which I showed to you at that time, you will understand that the diagnosis ust be somewhat uncertain in a sporadic case of this kind until the disease has been under servation a still longer time.

The treatment of this case is simply the frequent administration of milk, with the addion of stimulants when indicated by the weakness of the pulse. The child has been in apathetic a condition that the use of any drug has been unnecessary. Although at imes it has cried out as if in severe pain, yet these attacks have not been sufficiently long indicate their control by an opiate.

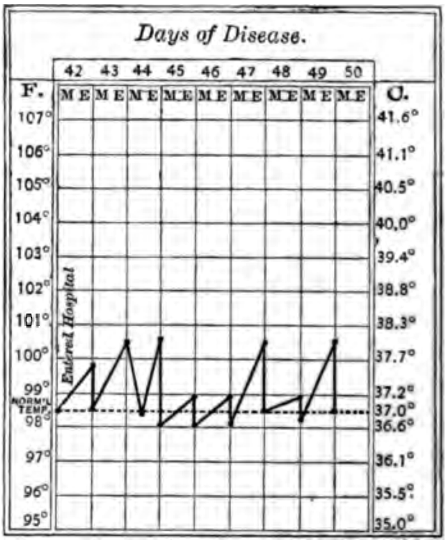
(Subsequent history.) During the following month the child remained very much the me as described above. The head was retracted at times, and the emaciation became treme, the abdomen being very much sunken (boat-shaped). In the next two weeks the ourishment was taken more readily, the head was less retracted, and he began to notice bjects around him, but he vomited once or twice nearly every day. The temperature at is time became normal.

This chart (Chart 26, page 698) marks the temperature from the day when the child tered the hospital, in the sixth week of his illness, until the temperature became normal, ne days afterwards.

One month later, which was two months from the time when the child entered the hospital, he was able to sit up without help. There was no retraction of the head, but the muscles of the neck were very rigid, and the head showed a tendency to fall back.

During the following month the child continued to improve slowly, increased in weight, recovered his appetite, and when seen one month later was found on physical examination to be in a normal condition. Here is a picture (III.) of the child, which was taken after an examination made by me which showed him to be in a normal condition in every respect.

CHART 26.



Case 324.

III.

Cerebro-spinal meningitis. Recovery after 4½ months.

This next child whom I have to show you is a girl (Case 325), eight years old, who apparently represents that form of cerebro-spinal meningitis which is designated chronic, and only a few cases of which have been reported.

The child entered the hospital two days ago. Her parents are said to have been healthy, and there is no evidence of tuberculosis or syphilis in the family. A brother whom I saw in consultation died of cerebro-spinal meningitis. With the exception of an attack of measles and of whooping-cough, the child has not had any other diseases. The present illness began four and a half months ago. The child had not been entirely well since the attack of pertussis which occurred one year ago.

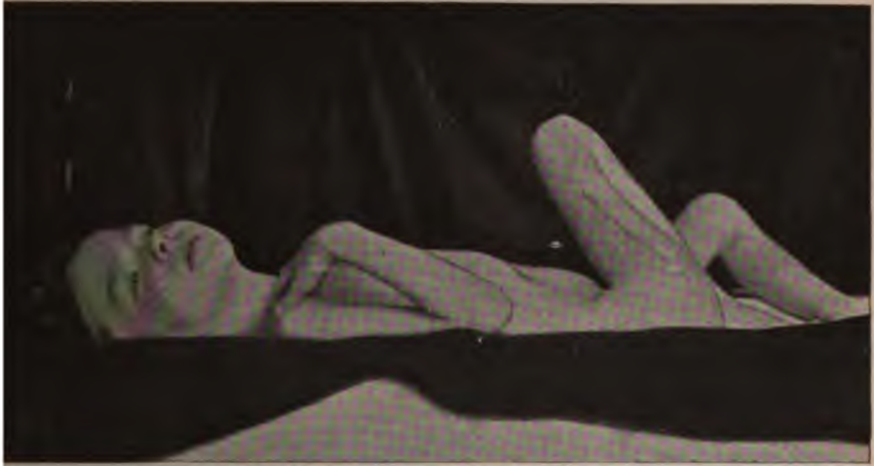
The onset of this attack was sudden. She went to bed in fairly good condition, but woke up in the night delirious, screaming, and apparently not recognizing her parents. These symptoms continued until the following week. There were no convulsions. A week later vomiting occurred every two or three days. This was not dependent upon food, and it has occurred at intervals up to the present time. The bowels were constipated. There has been more or less opisthotonos from the beginning of the illness, and also in the beginning there was decided retraction of the head. The stiffness of the neck has gradually diminished, but at times has been present since entering the hospital two days ago. Up to he present time the child is said to have had constantly a heightened temperature, varying rom 37.7° to 39.4° C. (100° to 103° F.), with a rapid pulse and quick respirations. Nothng abnormal has been found in the urine. There has been no efflorescence on the skin.

About four weeks ago the child was noticed to be blind. This has occurred suddenly. The child has had constant headache, and shortly after the beginning of the attack showed loss of power of motion in both legs. At times there has been incontinence of fæces and f urine. An examination of the urine showed it to have a specific gravity of 1015, to be ormal in color, to have an acid reaction, and not to contain albumin or sugar. No evience of syphilis was detected. She sometimes showed improvement in her general sympoms and became conscious, but she has not been able to sit up or to walk.

On physical examination you see (I.) that she is somewhat emaciated.

CASE 325.

I.

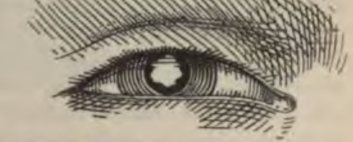


Chronic intermittent cerebro-spinal meningitis. Tache cérébrale showing on right thigh. Female, 8 years old.

There is extreme hyperæsthesia of the body and extremities. The slightest motion of he bed seems to cause discomfort and pain. An examination of the thoracic and abdominal rgans shows that they are normal. The pulse is 80 and regular, the respirations are natural, he temperature is 37.7° C. (100° F.). This morning she had an attack which was characerized by spasmodic contractions of all the muscles f the body, lasting for about thirty seconds. At his time there was no loss of consciousness, and the hild screamed for some time afterwards as though n pain. During the attack the pulse grew feeble nd intermittent, the respirations slow and supericial, and the extremities cold. Brandy was given ubcutaneously, and reaction took place, so that she now comparatively comfortable.

CASE 325.

II.



Metastatic choroiditis occurring in cerebro-spinal meningitis.

On examining the eyes (II.) you will see that lthough the pupils react and the retina is evidently ensitive to light, yet apparently she is blind.

You will notice in the middle of the eye a ellowish mass with an irregular border. Dr. Jack's eport of the examination of the eyes is as follows:

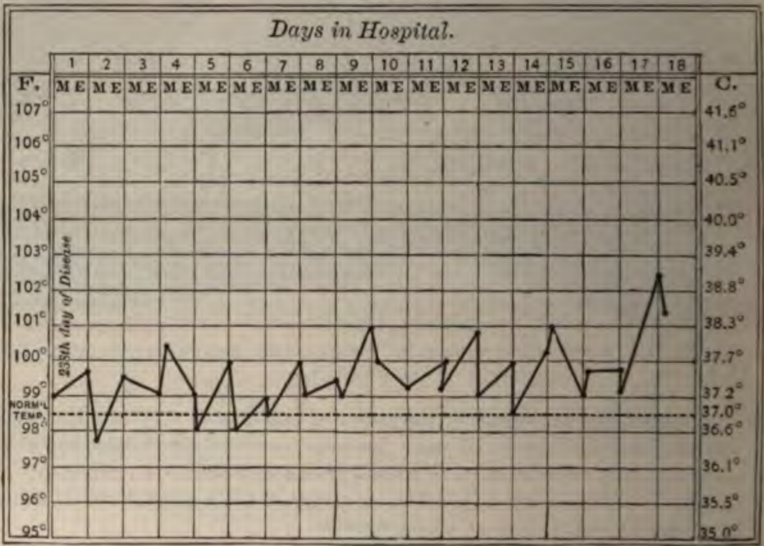
"There is a very slight hyperæmia in the ciliary region. The iris seems slightly pushed orward, and its pupillary edge is a little uneven. A yellowish or yellowish-white reflex appears from the fundus of the eye even without the use of the ophthalmoscopic mirror, and it is easy to distinguish that this reflex does not come from the level of the lens, but that it is situated deeper. The tension of the eyeball is very much reduced, and there is very little tenderness on pressure."

These yellowish appearances in the pupils are sometimes called pus emboli. The disease is due to embolism, and is called metastatic choroiditis with an exudation of pus in the vitreous. It occurs quite frequently in cerebro-spinal meningitis. It is to be differentiated from glioma. Sometimes this yellowish mass fills the vitreous entirely, sometimes only in part. It may have blood-vessels on its surface.

You see on drawing the finger over the right thigh that a decided *tache cérébrale* is produced, which lasts from ten to fifteen minutes.

This is only the third case of this form of cerebro-spinal meningitis which has come under my observation. In both the other cases the children eventually died from a prolonged sickness of many months, during which they at times seemed to be recovering. Cases have been reported by others, as by Henoch of Berlin, to have recovered, so that in this especial case we are not able to give a more definite prognosis. At present there is no lesion which I have detected that would prevent the child from recovering, although she will always be blind. On the other hand, she may eventually die from exhaustion.

CHART 27.



Chronic cerebro-spinal meningitis.

(Subsequent history.) After the above report the child remained in about the same condition. At times she screamed as though in pain, but she took her nourishment fairly well. She had one slight convulsive attack, which involved mainly the upper extremities, the lower extremities being only slightly contracted. During this attack her thumbs were turned in, her fingers clinched over them, and her arms, which were usually extended at her sides, were flexed at the elbows. Her face showed no sign of spasm, and during the attack the radial pulse was full, soft, and regular. After a few seconds the muscles again became relaxed, and there was no further tendency to contraction. The usual position in which she lay during the following weeks was with the thighs slightly flexed and abducted and the legs flexed at the knee, with the heels almost touching each other. About two weeks after entering the hospital the right leg became flexed on the thigh to such an extent that the knee almost touched the chin and the heel rested on the vulva. Any attempt to extend the leg made the child cry out as though in pain, the left leg being naturally extended in bed. This condition of the right leg continued for several days and then disappeared.

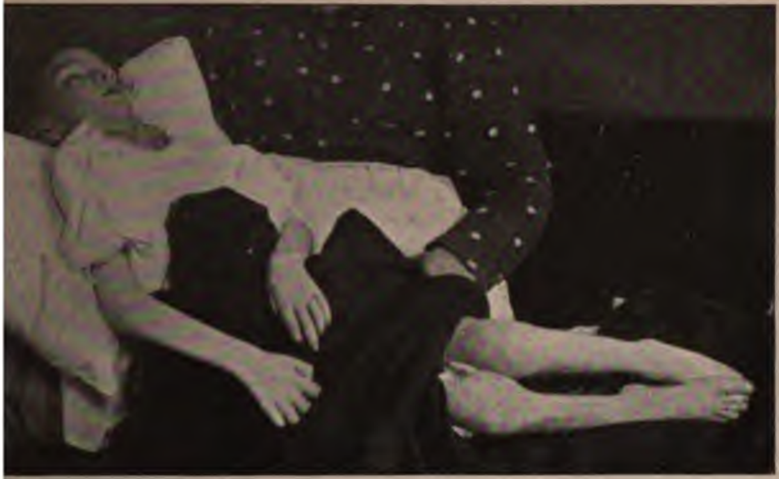
lays later a slight convulsive attack took place, which seemed to affect the right side han the left.

his chart (Chart 27, page 700) shows the temperature during the eighteen days when ild was in the hospital. The pulse during this time varied from 68 to 100; the respi- sometimes varied from 34 to 52, but were usually about 28.

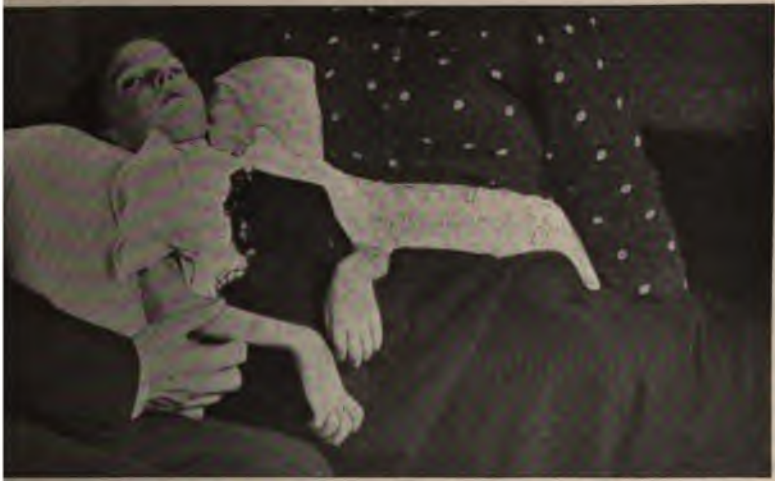
he fingers were flexed most of the time, and there was so much rigidity of the limbs e reflexes could not be satisfactorily determined. The pus embolus in the right eye

CASE 325.

III.



IV.



cerebro-spinal meningitis. Spastic condition of extremities 5½ months after onset of the disease.

to be farther back from the plane of the iris than at the previous examination. The of the left eye remained in about the same plane with the iris. The head was held any position in which it was placed, and she cried when it was moved. The pulse , weak and compressible, the respirations were rapid, 42, the alæ nasi moved some- nd there was apparently a slight degree of dyspnœa. The temperature in the axilla 6° C. (101.5° F.). There was slight cyanosis of the cheeks and lips, and an eruption of milia on the chest, apparently arising from her continually perspiring day and night. She lay in a stupor all the time, except when she was moved, when she would cry out. She showed no signs of understanding anything that was said to her. Sometimes she would be seized with an attack of rapid breathing lasting several hours. The bowels had been constipated up to within two days, when diarrhœa occurred. There was incontinence of fæces and urine, but no vomiting. During the last few days previous to this examination the teeth were kept closed, and had to be forced apart when she was fed. She was reported to have had one week previous to this examination a convulsion, in which the head was drawn back, the body and extremities were rigid, and the eyes rolled up. The child cried out sharply just before the convulsion. A physical examination made at this time showed nothing abnormal in the chest or abdomen.

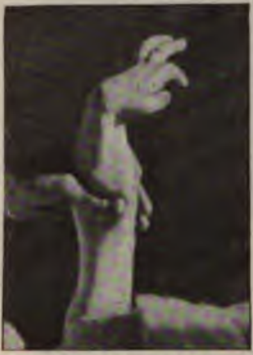
Eighteen days after entering the hospital the child was taken to her home, so that the daily record could not be obtained.

An examination made two weeks after she left the hospital showed a spastic condition of the extremities and neck, as seen in these illustrations (Case 325, III. and IV., page 701).

When seen by Dr. Bullard at this time the child took no notice of her surroundings, and her eyes when opened had a vacant expression, due largely to the mental condition, although at this time she was undoubtedly blind. The extremities were much wasted, and were all in a condition of spastic rigidity. There was slight flexion of the thighs on the body and of the legs on the thighs, while the feet were extended in nearly a straight line with the legs.

CASE 325.

V.



Chronic cerebro-spinal meningitis. Spastic condition of hand 5½ months after onset of the disease.

The hand, as you see in this illustration (V.), is flexed almost at right angles to the wrist. The proximal phalanges of the fingers are hyperextended, while the other phalanges are flexed. The thumb is strongly adducted, and its distal phalanx is flexed.

This is a position of the hand frequently found in the later stages of spastic paralysis, and is due to the preponderant contraction of the flexors of the wrist and weakness of the interossei and lumbricales.

(The child gradually grew weaker, and died of exhaustion a few weeks later.)

I will also report to you another of these rare cases of chronic cerebro-spinal meningitis, which I saw in consultation with Dr. Townsend.

The child (Case 326), a boy, four and a half years old, had been previously well, with the exception of an attack of measles when he was one year old.

On May 9 he was suddenly attacked with vomiting, which continued at intervals for two days. From the beginning of the attack he complained of severe pain in the head and abdomen. On the second day of the attack there was much contraction of the head, and he was slightly delirious, although rational most of the time. The temperature was raised from the beginning of the attack. There were no convulsions. The bowels were not moved during the first week of the disease. When first seen by Dr. Townsend the pulse was 124 and regular, the temperature 38.8° C. (102° F.), and the respirations 20 and regular. There was slight opisthotonos. There were no contractions of the muscles of the limbs. The symptom of Kernig was present. There was no tenderness along the spine. The cutaneous sensibility was everywhere normal. There were no cutaneous efflorescences or ecchymoses. The pupils were regular and reacted normally to light. There was no strabismus or photophobia. Nothing abnormal was detected on physical examination. The patient was apparently in great pain, cried out a great deal, and moaned continually. The suffering during the next few days was so great that morphine in doses of 0.002 gramme ($\frac{1}{30}$ grain) had to be given. This dose had to be increased so frequently that it was found that the child took 0.015 gramme ($\frac{1}{4}$ grain) before relief was obtained.

tion of ice to the head and spine gave no relief, and
mber of days later it was found that there was needed
rol the restlessness and pain 0.01 to 0.02 gramme
grain) of morphine during the twenty-four hours.

the twenty-fourth day of the disease the tempera-
hich had varied from 37.7° to 39.4° C. (100° to 103°
ame normal, remaining so until the forty-seventh
During this time the head was only slightly retracted,
child seemed free from pain, but remained in a
tless condition, not speaking, and taking but little
ment or stimulants. He became emaciated, passed
e and fæces involuntarily, and occasionally vomited.
ve enemata were not retained, but on the forty-first
the disease peptonized milk was retained, and on the
xth day he was able to take gruel, and at that time
and laughed.

the forty-seventh day of the disease a relapse oc-
the temperature rising to 39.7° C. (103.6° F.). The
as rigidly drawn back, the eyes were staring, and the
turned. The symptom of Kernig, which had never
ared entirely, again became well marked. At this
saw the child with Dr. Townsend. On the sixty-
ay of the disease the convulsive movements of the
n and leg, with turning in of the left eye, occurred.
days previous to this relapse a number of sudamina
ed on the neck and trunk, and an evanescent ery-
ous eruption on the neck and face, lasting only a
urs. From the sixty-first to the sixty-sixth day of
ease his body was covered with a macular effloresc-
the macules varying in size. Ecchymoses were at
e seen, and repeated examinations of the chest and
en showed nothing abnormal.

om the seventy-first day to the seventy-third day
nperature was again normal, the child took his food
he neck was straight, and his general appearance was
aging.

the seventy-fourth day he again had convulsive
ents, most marked on the left side of the body. The
as drawn back, and at noon the next day his tem-
re was 39.4° C. (103° F.). The pulse, which during
tire illness ranged from 120 to 140 and had previ-
een regular, was now at times irregular and inter-
t. The bowels were constipated at this time.

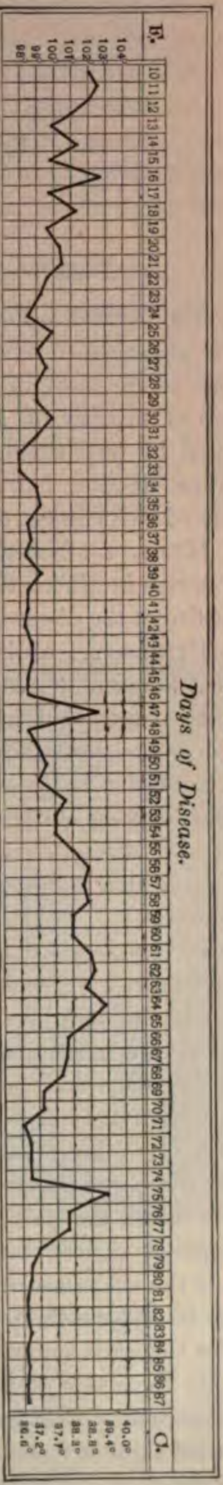
fter this, although the temperature became normal,
ld failed rapidly, and there was so much emaciation
e finger and thumb could easily encircle his thigh.

died quietly on the eighty-seventh day from the
the onset of the disease.

rough the kindness of Dr. Townsend I am enabled
you his temperature chart (Chart 28) from the tenth
the disease.

was very difficult, indeed impossible, to give a
sis in this case, as at times it seemed as though he
recover, and then the temperature would rise again
e unfavorable symptoms would return.

CHART 28.



Chronic intermittent cerebro-spinal meningitis.

LECTURE XXXIV.

PERIPHERAL NERVES.

NEURITIS.—PARALYSIS OF THE NEW-BORN.—NEURALGIA.

NEURITIS.—Neuritis is an inflammation of the peripheral nerves. It is accompanied by pain and tenderness in the affected regions, and in the more severe cases by paralysis and atrophy. I shall not dwell upon the cases of neuritis of a single nerve-trunk or its branches, which may be caused by traumatism, cold, or pressure, or may occur in the course of various diseases, but shall merely say a few words concerning a definite form of this disease, called *multiple neuritis*.

MULTIPLE NEURITIS.—In certain constitutional conditions a number of nerves in different parts of the body are affected with neuritis, and this constitutes the disease *multiple neuritis*.

ETIOLOGY.—Multiple neuritis usually occurs in the course of or subsequent to one of the infectious diseases. Of these diseases diphtheria is the most common, but it is said to follow scarlet fever and measles. A mild form sometimes occurs after typhoid fever. At times multiple neuritis is produced by drugs, such as lead, arsenic, or alcohol. It is not a common disease among children. The epidemic form of the disease has long been prevalent among the Japanese, and is known by the terms kakke and beri-beri, but it is quite rare in this country, and I have never met with it in children.

PATHOLOGY.—The pathological condition in multiple neuritis is an interstitial or parenchymatous inflammation of the nerves. A few nerves may be affected, or the distribution may be general. The nerves of the special senses, however, are rarely affected, and the nerves of the head and face are not usually involved.

SYMPTOMS.—The onset of the disease may be acute or subacute. It may at the beginning present severe symptoms, such as extreme pain, tenderness over the nerve-trunks, and fever with an accompanying paralysis. On the other hand, the pain in the beginning may be very slight, and the first symptoms noticed may be a gradually increasing weakness of the limbs, while the tenderness may be found only when especially sought for. There may be hyperæsthesia, anæsthesia, numbness, and loss of muscular power. After the acute symptoms have passed away the faradic irritability is diminished: the action of the nerves to the galvanic current is diminished, and the reaction of degeneration is present. When the extensors of the leg are affected there is foot-drop, and when those of the forearm are affected there is wrist-drop. The course of the disease is apt to be a long one, and in the later stages atrophy occurs, while the early hyperæsthesia may give place to a more or less marked anæsthesia, and numbness and various other paræsthesiæ may occur. In mild cases, where only pain and tenderness exist, the knee-jerks are not diminished, and may be even slightly increased, but in the more typical cases of the disease they are absent. Contractures and spasmodic conditions are absent, the paralysis being flaccid. The temperature is apt to be somewhat raised, and is decidedly so at the onset when the disease is acute.

Diagnosis.—The diagnosis is to be made chiefly from poliomyelitis anterior, which may simulate multiple neuritis in certain cases; but in the former disease the absence of pain except during the first few days, with the more limited distribution of the paralysis, and the absence of tenderness, will serve to distinguish it from the latter.

Prognosis.—The prognosis of multiple neuritis is favorable even where the disease begins with an acute onset accompanied by delirium and high fever, and, although the paralysis may last for many months, the cases usually recover.

Treatment.—The treatment is at first by absolute rest in bed, and later with electricity, massage, and strychnine.

Iodide of potassium is indicated in those cases which are caused by lead or arsenic.

In the subacute cases electricity and massage are indicated from the very beginning.

It is safer to wait until the pain and marked tenderness have disappeared before beginning the administration of strychnine.

Case 327.

Multiple neuritis. Female, 11 years old.

This little girl (Case 327), eleven years old, represents a case of multiple neuritis produced by doses of 1 gramme (15 minims) of Fowler's solution given three times a day for some weeks during an attack of chorea.

The first symptoms which were noticed while she was taking the arsenic were that she vomited several times, but this was not supposed to have been caused by the arsenic, and the drug was therefore continued. It was next noticed that the child was unable to walk. Her limbs appeared to be very weak, and there was absence of knee-jerks and ankle-clonus. The sensation of the limbs was normal. A few days later she was found to have tender points over various parts of the legs. The legs then became atrophied. About a month later tender points developed in the arms, and she soon lost the power of using her arms, to such a degree that she had to be fed. At this time, although the arsenic had been omitted for several days, a large quantity of it was found by Professor Wood in the urine.

You see to-day that she has no remains of the choreic movements, but that she is rather stupid, and that there is tenderness on deep pressure over certain points in the calves of the legs. She has no headache and no other abnormal symptoms. She is being treated with the faradic current daily and with 0.18 gramme (3 grains) of iodide of potassium three times a day. Since this treatment was begun, three weeks ago, the power of grasping has returned, and the arms react somewhat better to the faradic current.

(Subsequent history.) One month later it was found that she could almost support herself without assistance. A little later she walked with crutches, and a month later she could walk without assistance, but with difficulty. The knee-jerks were still absent. She continued to improve, and finally after a number of months recovered entirely.

This boy (Case 328), who has been brought to the hospital this morning, is an interesting case of multiple neuritis.

He is nine years old. He was perfectly well until he was six years old, when he had an attack of measles. He was sick for two weeks, and then recovered apparently entirely. A week later it was noticed that he became fatigued on going up-stairs, and finally he lost the use of his limbs. For a year he walked with the help of a chair. There was simply loss of power of motion, but no pain. The appetite was not lost. He at times had slight headache. After the paralysis appeared it was noticed that the cervical glands swelled at times. The bowels were regular, and there was no trouble with the urine. The limbs were somewhat tender on pressure. His temperament was changed, so that he was rather fretful. Somewhat later he lost the use of his legs entirely, so that he had to be carried.

He then left his home and went to Florida, and after a few months recovered the use of his limbs entirely and became perfectly well. He returned to his home and went to school for a month. At the end of that time the symptoms of the previous attack began slowly to return, and he finally had to stop going to school.

On examination he is found to protrude the tongue straight. He has no symptoms referable to the head. There are red exanthematous patches on the elbows and knees. There is no especial atrophy of the legs, but there is a good deal of emaciation of the arms. The arms cannot be raised beyond the level of the shoulder. There is some pain in the shoulders when the arms are raised for him. There is tenderness on pressure of the shoulders. The flexion of the arms is good; the grasp of both hands is weak, but there is no loss of movement. The patellar reflexes are absent, and he walks with a peculiar tilt of the pelvis. He can stand well with his eyes shut. There is no disturbance of the kidneys and bladder, and no proof that the symptoms result from masturbation. No irritation is noticed about the prepuce, which, however, is tight. He cannot get up from a sitting posture or when lying down. He apparently has lost the power of pushing with his arms. The vertebral column is straight, and there is no apparent tenderness. He has never had diphtheria nor any of the eruptive diseases except measles.

The history of this patient and the examination lead me to eliminate hereditary ataxia and locomotor ataxia. The rapid improvement which took place in this instance when the child was taken away from his home for some months, and the recurrence of the symptoms within a month after his return, justify me in suspecting that the cause of the disease is a local one connected with the child's home. Of such toxic influences, that from lead is the most common and probable.

PARALYSIS OF THE NEW-BORN.—By paralysis of the new-born is meant that form of peripheral paralysis which occurs during the delivery, and which, as a rule, affects the face or one of the extremities. In this sense it is to be separated from injuries to the brain and spinal cord which are produced during the delivery,—in fact, from any paralysis of central origin which may occur in intra-uterine life, either before or at the time of delivery.

ETIOLOGY.—The cause of this form of peripheral paralysis is most often traction made upon the head of the child during delivery, thus producing a direct injury to the nerves, or dislocation or fracture of one of the bones, resulting in pressure on the nerves. Although this form of paralysis has

known in a number of cases to result from pressure by the forceps
g the delivery, yet it has also been met with after an apparently
al delivery, where the pressure did not seem to be especially severe
longed.

ATHOLOGY.—When the nerves of the face are affected, the resulting
is supposed to be from an injury of the facial nerve; and when the
s affected, the lesion is supposed to be an injury of the brachial plexus
the nerves in the lower part of the neck. When the limbs are affected,
arms have been known to be paralyzed; but, as a rule, the lesion is of
rm.

YMPTOMS.—A paralysis of this form becomes apparent immediately
birth. This is a very important fact to remember, as in this way we
ifferentiate the disease from a paralysis resulting from poliomyelitis
or, which is exceedingly rare in the early months of life, the youngest
n record being twelve days old. Where the face is affected, it is due,
rule, to an injury of the seventh nerve, thus producing a peripheral
paralysis. The peripheral form of facial paralysis is distinguished
the central in that in the former all three branches of the seventh
are apt to be affected, while in the latter form only the lower two
hes are involved. In the peripheral form, therefore, the eye on the
ed side cannot be closed entirely, causing the condition known as *lag-
almia*, and there is inability to wrinkle the muscles of the forehead
e affected side. In facial paralysis of central origin the muscles of
orehead are not affected, and the ability to close the eye is but little
ased.

here the paralysis affects an arm it hangs lifeless by the side, with the
turned backward and the fingers often flexed. The fingers and fore-
nay be moved, but the movement of the upper arm to any extent is

IAGNOSIS.—This form of paralysis is to be diagnosticated from cerebral
ysis by the absence of increased reflex irritability and by the distri-
n of the paralysis. In the cerebral form all the muscles are affected;
e peripheral form, only individual muscles. It is doubtful whether
yses of spinal origin occur in the early days of life.

ases of paralysis of the arm in the new-born should also be diag-
ated from surgical injuries represented by fractures, dislocations, and
ation of the epiphyses. These are eliminated only by a careful exami-
n of the head of the humerus on the affected side, showing the absence
epitus, abnormal mobility, callus, or deformity.

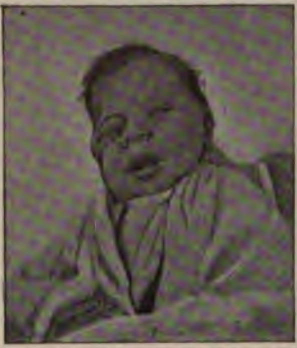
ROGNOSIS.—The prognosis in cases where the face is affected is very
as the paralysis in these instances lasts but a short time. We must,
ver, be somewhat guarded in the opinion which we give concerning
, as in some instances the paralysis does not disappear and the muscles
e face are left irreparably injured.

n regard to the paralysis of the arm, the prognosis is generally unfavorable, especially if marked improvement does not soon occur, and ordinarily when improvement takes place it is very slow. Most of these cases never recover, and partial recovery should not be expected for a number of years. Shortening of the arm is marked in the later history of the severer cases.

We can therefore state that peripheral paralysis of the new-born when it affects a limb is much more serious in its prognosis for complete recovery than when it affects the face.

TREATMENT.—Electricity and massage continuously applied for a number of years is a very important part of the treatment of these cases, and obviates the atrophy of the muscles from disuse, which must necessarily take place to a greater or less extent.

CASE 329.



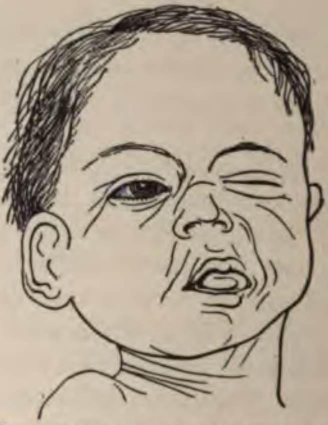
Peripheral paralysis of the new-born. Paralysis of right side of face. Forceps delivery. Infant, 2 hours old.

Here is an infant (Case 329), two hours old, who has a peripheral paralysis of the right side of the face, caused by pressure of the forceps on the seventh nerve.

In this case the closure which you notice of the right eye is produced by the swelling of the face and eyelid. You see that the entire right side of the face is affected.

I have here another infant (Case 330), one year old, who presents the condition of peripheral paralysis of the right side of the face.

CASE 330.



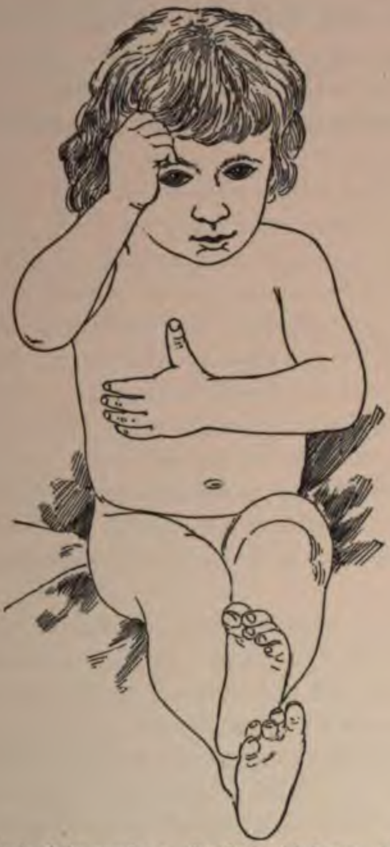
Peripheral paralysis of the new-born. Paralysis of right side of face. Infant crying. Male, 1 year old.

When the infant cries you see that the lines on the right or paralyzed side of the face are somewhat obliterated, and that the right eye cannot be closed (lagophthalmia). The lines of the left or non-paralyzed side of the face, on the contrary, are deepened, and the left eye can be closed. The mouth is drawn to the left.

The prognosis of this case is bad for complete recovery, and treatment of any kind will probably be of no avail, owing to the length of time for which the lesion of the seventh nerve has existed.

little boy (Case 331) is two years old. He was healthy at birth, but the labor was ntal. When he was three days old it was found that his left arm was swollen. irst seen at the hospital when he was seven weeks old. At that time he was able his fingers and wrists, but held his arm with the elbow straight to the side and the nated. He is now, as you see, able to make slight movements of flexion of the

CASE 331.



Peripheral paralysis of the new-born. Paralysis of left arm. Male, 2 years old.

id slight contractions of the deltoid. Under the use of electricity he has been gradual improvement. He can grasp objects fairly well with his left hand, and the elbow completely, and raise his hand and forearm as far as the nipple. You n raise his right arm with ease to his head, but cannot raise the left hand farther lower part of the chest.

evidently a case of paralysis of peripheral origin caused by trauma.

prognosis in this class of cases is often grave for complete recovery, but, as you iderable improvement has taken place in this child.

URALGIA.—In contradistinction to the affection of the nerves I have just described as neuritis is a functional affection of the fibres of the peripheral nerves, represented by pain and called ia.

ralgia is so rare in infancy and early childhood that I shall not do more than refer to it. When neuralgia occurs it may affect very different localities, and may be represented by intercostal neuralgia or the various milder forms of flitting pains in different parts of the body which so commonly occur in children.

I have found in most cases of neuralgia that temporary relief from the pain can be obtained by the use of phenacetine, and I have never seen any contra-indications to using this drug. It can be given in doses of 0.06 gramme (1 grain) for every year of the child's life up to 0.6 gramme (10 grains). I am in the habit of guarding against any possible bad effects by giving the phenacetine in a little brandy-and-water.

LECTURE XXXV.

NERVOUS DISEASES PRESUMABLY ORGANIC.

Chorea.—Epilepsy.—Insanity.

ing of the next class of nervous diseases, which I have called
organic," it may perhaps be well to explain why I have
this term. It is because we cannot help feeling that in true
ue epilepsy there must be some organic lesion, and that it
ins for future investigation to show what the lesion is. When
as been determined we can relegate the disease to the organic
ibly it may be decided that it belongs to the functional diseases.
ere can be but a slight pathological distinction between these
those which I have called functional, but their chronic course
rious nature ally them clinically so much more closely to the
nown organic origin than to the indefinite functional class that,
y in teaching, I have decided to separate them from the latter.

A.—Chorea is a disease characterized by irregular and invol-
ular movements without loss of consciousness, and affecting the
olition.

ase is rare in infancy, but may occur in the early months of
lom begins after puberty. It is most apt to begin and is most
its symptoms during the period of the second dentition,—that
e period of active growth, from six years to puberty. The
ber of cases is found among the female sex and among those
receive sufficiently nutritious food. It will be well for you
d clearly that a sharp distinction should be made between the
ea, with its characteristic choreiform symptoms, and the same
ymptoms resulting from various diseases, sometimes represented
ervous lesions, sometimes by purely reflex causes. It will save
seless reading of the literature of chorea and much profitless
to its etiology and pathology if you will bear this distinction
liminating those forms of chorea which are due to gross lesions
us system, such as the post-hemiplegic and congenital forms,
nce very materially reduce the cases of true chorea. In like
hould separate from true chorea those cases of peripheral irri-
ich the partial choreiform symptoms are evidently reflex and
by removal of the cause. Examples of these reflex choreiform
re the facial chorea from naso-pharyngeal irritation and the
iform movements occasionally arising from errors of refraction
insufficiency. The consideration of these anomalous forms of

chorea belongs with the diseases in which they occur, and they should be spoken of in connection with the other symptoms of these diseases.

ETIOLOGY.—Chorea can be precipitated by other diseases, such as measles, though this, in my experience, rarely occurs except among the poorly cared-for. A certain number of cases have so directly followed intense fright that we must acknowledge acute mental conditions as a cause. The disease which is most frequently associated with chorea is rheumatism. The percentage of cases, however, in which this association takes place is difficult to determine. This difficulty arises from the want of uniformity in the reported cases of different observers, due to their different ideas as to what constitutes rheumatism. If only the cases of acute articular rheumatism are to be classified under rheumatism, very few cases of associated chorea will be spoken of; while if all the flitting aches and pains of childhood are considered to be rheumatism, the number of choreic cases caused by rheumatism rises to fifty per cent., or possibly more. The truth will in the future probably be found to lie in some intermediate number, for that in certain cases a close connection exists between chorea and rheumatism is very evident. The difficulty becomes still greater when we examine the relationship between chorea and endocarditis. Of course where there is a rheumatic element in the case we should expect a cardiac lesion to arise, and to be dependent on the rheumatism. In certain cases, however, we find chorea with endocarditis entirely irrespective of rheumatism. This occurs to such an extent that in our cases of chorea we should watch for cardiac lesions just as carefully as in our rheumatic cases. Heart-murmurs of a hæmic nature may occur in chorea as in any other disease of a debilitating nature. They should, however, always be looked upon seriously, as possibly indicating an insidious form of organic endocarditis, which, instead of being evanescent and passing off entirely with the recovery of the chorea, may either seriously disable the heart or lead to a fatal issue. A special microbic cause for chorea, as for rheumatism, must be thought of, but as yet has not been proved to exist. An hereditary tendency to nervous explosions of a choreic type has long held a prominent place in the etiology of chorea. In my experience, however, it is not very common, unless the children are poorly nourished, badly cared for, or exposed to nervous excitement during their school life.

Overtaxing of the central nervous system during the school year has so often been shown to result in an attack of chorea in the spring and in a recurrence in the autumn on returning to school, that it should be recognized in considering the etiology of the disease. Strain of the ocular muscles has been considered an exciting cause of chorea.

PATHOLOGY.—There are a large number of cases of chorea in which the disease is found to have no apparent pathological lesion. Its symptoms, however, show us that the morbid process is located in some part of the central nervous system. The lesion, however produced and whatever it is, is represented by a profound excitement of the motor centres, presumably to their inanition, and is accompanied by a temporary inability of these tres to recover themselves. Many lesions have been described as occur- g in chorea, but in the pure cases (Sydenham's chorea) which I have just ribed, and which really represent the disease, there is no lesion which a our present knowledge we can say is characteristic.

SYMPTOMS.—Chorea may be in its distribution general or partial; in its rse acute, subacute, or chronic. In many cases the disease is exceedingly d in its symptoms and of a benign type; in others it assumes a severity ch seems to threaten life. I shall presently show you examples of both es of the disease. The beginning, though at times sudden, as from ht, is, as a rule, gradual, at first a few muscles only being affected. The ld becomes fretful and impatient, and we must carefully differentiate se symptoms from those resulting from bad temper, for which they are to be mistaken by the family. The clinical picture of the disease is a ky, irregular, involuntary contraction and relaxation of the muscles, apt begin in the fingers, hands, and face. There is an irregular, uncertain ion of the part affected, and efforts of the will only partly control the vements. As the disease progresses, the voluntary control of the muscles ninishes more and more, and at times disappears entirely.

The movements ordinarily cease during sleep, but in severe cases they tinue during and even interfere with it. At times the child is unable to lk, on account of weakness. The speech may become slow and indistinct, m the affection of the muscles of the tongue and of the larynx, and even stication and deglutition may become difficult. In very severe cases the ficulty in speech may be enhanced by the mental condition, which may ome impaired, and which is represented by dulness and apathy. The don reflexes are apt to be lessened in severe cases. The muscles grow ak and soft, and there is considerable emaciation. There is usually loss appetite, and the bowels are often constipated. The urine and its urea ve been found to be increased during the course of the disease. The namometer usually shows impaired muscular power. In certain cases the scles of the extremities on one side of the body are principally or alone ected (hemichorea). These cases do not differ from the ordinary bilateral es in any way except in this respect.

In very severe cases there may be involuntary evacuations of the fæces l of the urine. The disease is distinct from epilepsy, and there is little nger of the patient becoming epileptic unless the disease happens to develop an individual who is predisposed to that condition.

PROGNOSIS.—Chorea is very apt to show relapses and to recur every r for some years. Though often obstinate in the persistency of its nptoms, yet it may be said to be self-limited, and, as a rule, to recover, vided no complications, such as from cardiac disease, arise. The time ich elapses before complete recovery is very variable, but well-marked es usually extend over a period of three or four months. Although, you will notice, in speaking of the prognosis of chorea I consider it, as a rule, a benign disease, yet we must always look upon it as a serious disturbance until we are sure that we are dealing with the usual mild form of the affection. As an illustration of how careful we should be to give a guarded prognosis in the early stages of acute chorea, I shall mention the following case:

A girl (Case 332), nine years old (Cook and Beale), began to have choreic movements, which constantly became worse. Delirium developed, with a slight fever, a rapid and feeble pulse, and a quick and interrupted respiration. Death suddenly occurred one hundred and thirty hours after the onset of the disease. The autopsy revealed extreme anæmia of the pons and medulla, but no other changes of note in other parts of the body.

We must allow that even uncomplicated chorea is a varying disease as to the severity of its symptoms and their persistence for a longer or a shorter time. We also know that there is a marked tendency to relapse, and that the number of relapses varies to a great degree. The length of the attack and the response to treatment may differ much. Bearing these facts in mind, you will comprehend the rapidity with which certain individuals are attacked or the quickness with which they recover. You will meet with some cases which recover rapidly under only hygienic treatment, and with others which are apparently unaffected by any drug whatever. Where heart-murmurs, evidently representing organic disease, appear, you will often find cause for wonder in the comparatively slight discomfort which the cardiac lesions entail. At times, again, you will be surprised at the rapidly fatal course of some cases complicated by cardiac disease, and at their uncontrollability by any treatment whatever.

TREATMENT.—The disease is variable in its duration whether treated by drugs or not. There cannot be said to be any specific treatment with drugs for chorea, but of the many drugs that have been used in this disease arsenic has, in my experience, been the most beneficial. Arsenic should, however, be used with care, and on the appearance of any evidence of the physiological action of the drug, such as nausea or œdema of the eyelids, it should at once be discontinued. It should not, as a rule, be given in very large doses, as cases have occurred in which it has produced a multiple neuritis of many months' duration. Where any special cause can be found for the attack, such as rheumatism, appropriate treatment directed to that cause should be employed. It is manifest, however, in the uncomplicated cases that our main reliance must be placed on hygiene and food. Fresh air, nutritious food, tonics to control the anæmia and general prostration, kindness, seclusion to secure mental quiet, stimulants if there is much resulting weakness, and the bromides for insomnia and over-excitement, are the means which I have found most valuable in managing this disease. I have seen well-marked cases get well in from sixty to seventy days where good food and a small amount of stimulant constituted the entire treatment.

If the attack is very severe, skilled nursing is a very important adjunct in the treatment. The child should be protected from harming itself by ms of the padded bed, and light but well-padded splints to control the vements during sleep are indicated occasionally.

I have a number of cases of chorea to show you. It is one of the most mon diseases that are seen in the hospital.

Here is a little girl (Case 333), six years old, who represents one of the milder forms horea. There is no history of nervous or cardiac disease or rheumatism in the family, the child herself has never been sick before.

Three weeks ago she complained of pain in her left hand and arm, and later the scles of the arm began to twitch. Soon after, the whole body was affected in the e way. Somewhat later it was found that the child could not talk plainly, and it with some difficulty that she could feed herself. She seemed nervous and peevish, showed constant irregular incoördinate movements, chiefly of the face, mouth, and er extremities. The legs were slightly affected, and sometimes the muscles of the nk also. There has been no paralysis of the muscles. The eyes have been normal in ir reaction.

Since entering the hospital she has been treated chiefly without drugs, and especial ntion has been paid to giving her a nourishing diet, baths, gentle massage, and rest in in a quiet corner of the ward.

On entering the hospital, three weeks ago, a physical examination showed nothing ormal in connection with the heart or other organs. An examination of the urine wed it to be normal.

You see to-day that she is looking very well, and that the incoördinate movements have sed entirely. Marked improvement was shown after she had been in the hospital for weeks, and for the last few days, about the forty-second day from the onset of the ase, I have considered her cured and ready to return to her home.

Here is a little girl (Case 334, page 716), eight years old, whose symptoms are so racteristic that we can at once make a diagnosis of chorea.

I have not been able to ascertain anything concerning the history of this child, except she has been subject to attacks of this nature for some time. There is no history of umatism, nor of any other disease. The child seems to be physically well and strong, on examination nothing abnormal is found in any of the organs, with the exception slight systolic souffle heard distinctly at the apex of the heart and transmitted through axilla into the posterior scapular region. The area of cardiac dulness is not increased. patellar reflexes are increased. There are marked choreic movements of the hands, , and head. On inspection you see that the limbs are flexed and extended, with irreg- incoördinate movements, and that there are from time to time the same muscular tractions in the face. She shows a certain amount of mental disturbance, characterized etimes by peevishness and sometimes by slyness; she is dull rather than bright.

She represents the disease chorea of a moderately severe type, which from its constant rrence and chronic course will probably prove to be very intractable. The prognosis o her mental condition is especially serious, as the probability is that the mental im- ment will increase rather than decrease.

She has been treated with a number of drugs, such as arsenic, quinine, iron, and others, of which seem to be of any benefit.

In regard to the mitral systolic souffle to which I have just referred, it is possible that wholly a functional manifestation. You must always bear in mind, however, that is great liability in cases of chorea of organic cardiac disease developing, and that l all signs of cardiac disturbance have disappeared a very guarded prognosis should be n as to whether the cardiac disturbance is of functional or of organic origin.

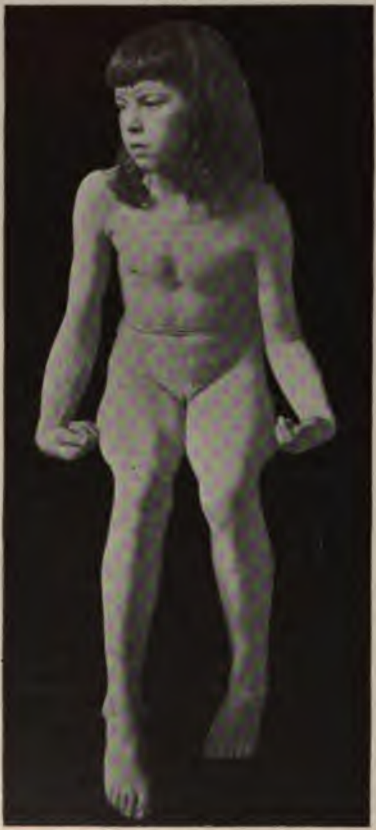
In a case of this kind, instead of the gradual diminution of the murmur, which would n likely to occur, judging from the very slight evidence of cardiac disturbance present, cially as the murmur could be well accounted for by the choreic functional disturbance, possible that an endocarditis with valvular disease may exist and later produce more ous symptoms.

I shall now show you this little girl (Case 335), thirteen years old, who represents one of the milder forms of recurrent chorea.

She had a number of diseases preceding her first attack of chorea. When she was two years old she had an attack of diphtheria, when five years old one of measles, when seven years old one of scarlet fever, and when eight years old one of rheumatism.

When nine years old she had her first attack of chorea, which occurred in the spring of the year and lasted for a number of months. This was followed in the spring of the next year by a second attack. In the spring of the following year she had a third attack of the disease; at this time the incoördinate movements were not so marked as in the previous attacks, but the debility was greater. When she was examined during this attack, it was found that the heart, although weak and somewhat irregular, presented no evidence of murmurs. The pulse was 84, the temperature was normal, and there were no signs of any other disease. She was treated at the hospital, and recovered in a few months. In the spring of the next year she had a fourth attack of chorea. At that time nothing abnormal beyond the choreic movements was noticed. She was treated with from 0.18 to 0.36 gramme (3 to 6 minims) of Fowler's solution three times a day, and in a few months left the hospital apparently well.

CASE 334.



Chorea. Female, 8 years old.

CASE 335.

Recurrent chorea, fifth attack. Female, 13 years old.

In the spring of the present year, one year from the beginning of the fourth attack, she entered the hospital with a fifth attack, for which she is now being treated. In this attack, after using Fowler's solution for a few weeks and not obtaining any especial benefit, I have given her iron and nux vomica. She is gradually improving under this treatment, and, as you see, has a fairly good color, has a good appetite, and seems quite strong. There are some remains of the incoördinate movements, which especially appear when she is embarrassed by the observation of the people who are around her. You see she now has a slight twitching of the face and hands, and occasionally the hands, and especially the thumbs, are drawn inward with an arhythmical movement.

The prognosis in this case is good. Although she has had five attacks of chorea, no organic lesion of the heart, nor any other abnormal condition, has resulted from them, and she will probably recover entirely, and will not continue to have attacks of the disease when she is a little older.

This little boy (Case 336), eight years old, is a case of chorea which I wish merely to how you as one in which the treatment by quinine was found to be followed by an increase n the choreic movements and to be entirely without benefit.

The child has always been of a nervous temperament and very studious, and has grown rather more rapidly than other children of the same age. He was taken sick one month ago, and has been in the hospital two weeks.

On entering the hospital he had the usual symptoms of chorea, incoördination on using his muscles, and difficulty with his speech. His mind was clear, and there was nothing bnormal detected about him except a cardiac murmur, apparently hæmic, and considerble general weakness, so that he walked with difficulty. He was at once treated with bsolute rest and quiet in a room separate from the rest of the patients in the hospital, and mall doses of iron were administered. His general condition improved gradually but lowly during this week, when it was thought advisable to endeavor to hasten his recovery by giving him quinine. Sulphate of quinine was administered in doses of 0.12 gramme 2 grains) three times a day, with orders to have it gradually increased to 1.2 grammes 20 grains) in the twenty-four hours. This treatment was continued during the last week ntil the amount of quinine taken in the twenty-four hours amounted to 1 gramme (15 grains).

Under this treatment he has grown steadily worse. The disturbance in speech has greatly increased, and he has lost the power of using his arms and legs. His mind is clear. There is no vomiting, but he has a certain amount of tinnitus aurium and a slight headache. As the deglutition is also beginning to be affected, I have considered it wise to mit the quinine.

(Subsequent history.) After the quinine had been omitted for twenty-four hours the child's general condition was decidedly improved. The treatment with iron was renewed, and he gradually recovered, leaving the hospital one month later in apparently good health; the cardiac murmur had disappeared, there were no incoördinate movements, and he could speak and use his arms and legs normally.

This next boy (Case 337), nine years old, has been subject to attacks of chorea for nearly four years. The attacks usually come on in the spring with considerable severity, and continue for nearly six months, gradually diminishing in intensity until the symptoms are scarcely noticeable. The child has a history of rheumatism, not, however, of a high grade. The attack from which he is now suffering began four months ago, and has been a quite severe one, so that he has been unable to control the movements of his hands and face during the day; they are, however, quiet at night.

On entering the hospital a physical examination showed a marked systolic murmur, heard most distinctly at the apex and transmitted to the axilla. The area of cardiac dulness was not especially enlarged. The urine was normal, and nothing abnormal was detected about the child.

He was at first treated with Fowler's solution, 0.12 gramme (2 minims), three times a day. After four days the choreic movements became less marked and the cardiac souffle less distinct. Two days later, however, the Fowler's solution had to be omitted, as it caused nausea and vomiting. At this time there was a double souffle, heard most distinctly over the left third interspace, close to the sternum. A few days later Fowler's solution was renewed, but, as it caused gastric disturbance, again had to be omitted, and it was found that it could not be given for more than two days at a time without causing puffiness of the face.

A month later the choreic movements had decidedly lessened and the cardiac murmurs disappeared.

To-day, two months from the time when he entered the hospital, he appears to be free from the disease. His muscular movements are natural, though his legs are slightly weak; he has a good appetite, and there are no abnormal symptoms connected with the heart.

I show you this case as representing one of recurrent chorea in which arsenic is not tolerated, and in which the indications for treatment are chiefly rest, good food, bathing, and massage in the beginning, followed later by the administration of some mild form of iron, such as the tartrate of iron and potassium. The cardiac disturbance in this case was in all probability functional rather than organic, as not only were the cardiac murmurs most distinct when the child was weak and anæmic, gradually growing less as he grew stronger and the anæmia disappeared, but also on the most careful physical examination I can now find no evidence of organic disease. You can therefore consider it a case of recurrent chorea with accompanying functional cardiac disturbance.

This little girl (Case 338), four and one-half years old, in this bed in a quiet corner of the ward, is an exaggerated case of chorea.

Until this attack she had been a healthy, bright, strong child. She had an attack of pertussis when she was two and one-half years old, and one of measles when she was three and one-half years old. She has never had rheumatism. She began to have choreic movements of a rather subacute type one year previous to this attack for which she has been brought to the hospital. The onset of this second attack was while she was going to school, and when she was in fairly good health. The muscles of the mouth and face were first affected, and she seemed to get very much excited when at school, without any apparent cause. The symptoms rapidly increased in severity, and she was brought to the hospital a few days ago.

I shall not give you the details of this case, as they differ very little from those which I have already described to you. I show her to you as representing one of the exaggerated forms of chorea.

The symptoms have increased since entering the hospital, and the muscular movements are so prominent, even at times occurring when she is asleep, that she has to be kept in a padded bed. She is apparently unconscious. She sleeps with the greatest difficulty, and she has been unable to speak since she entered the hospital. At times the head has been slightly retracted, but there has not seemed to be any especial rigidity of the muscles of the neck. The area of cardiac dulness is not enlarged. There is a slight cardiac murmur, heard most distinctly at the base of the heart. She is being treated with stimulants and as much milk as it is possible to make her swallow, but at present she is taking only about 473.11 c.c. (1 pint) in the twenty-four hours.

(Subsequent history.) The choreic symptoms lasted for some weeks, but gradually grew less violent, and the child finally recovered entirely.

An examination of the heart two years later showed that organic disease was present, as indicated by the enlargement of the cardiac area of dulness, a mitral systolic murmur at the apex, and an accentuated second pulmonic sound. At the time of this examination the child was found to be weak and delicate, and was evidently suffering from the effects of organic cardiac disease.)

In connection with the previous case I shall report to you a case which I saw in consultation with Dr. Boardman.

A boy (Case 339), ten years old, had always been delicate, but had had no special disease, such as rheumatism, until six weeks previous to the time when I saw him, when he was said to have had an attack of epidemic influenza. He recovered completely from the disease in ten days, and seemed as well as ever. Three or four days later he began to show symptoms of chorea. These symptoms gradually increased in severity, and finally were continuous, except when he was asleep. After he had had the chorea for one week he was unable to articulate, and began to have trouble with deglutition. He soon lost the power of controlling his limbs, grew very weak, and was confined to his bed. There was considerable insomnia. In the second week of the attack the choreic movements became so violent as to endanger his falling out of bed. The temperature up to the time when I saw him, in the fourth week of the attack, was normal. The pulse varied from 140 to 150, and the respirations from 35 to 40.

When I saw him, at the end of the fourth week from the beginning of the chorea, his mind was perfectly clear; he had a little pain in the hands and shoulders, apparently from the continual movements. He was unable to articulate clearly. There was difficulty in swallowing, and he was considerably emaciated. Nothing abnormal was found in the lungs. The heart was beating tumultuously. The area of cardiac dulness was very slightly enlarged, but there were no cardiac murmurs.

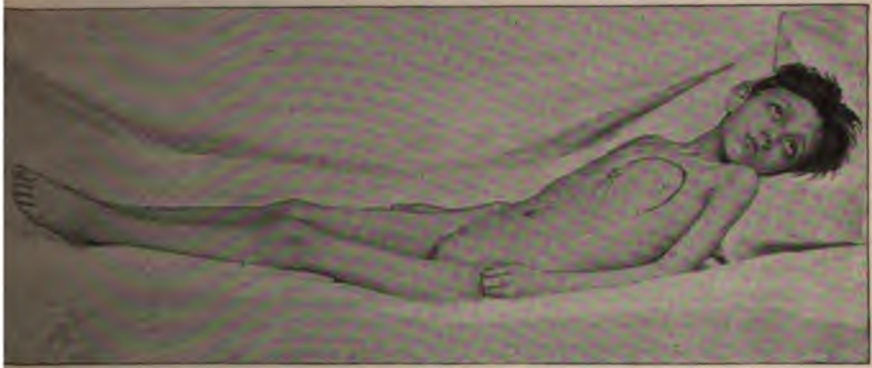
The case was apparently one of primary acute chorea without complications. Although in many of these severe cases of chorea no evidence of cardiac disease can be obtained on physical examination beyond a slight dilatation of the left ventricle, yet some disease of the endocardium or valves may often be found at the autopsy. In these cases, however, the temperature is, as a rule, raised. In this case the continuous normal temperature and the absence of any signs of cardiac disease beyond a slight dilatation from the apparent weakness of the ventricular muscles seemed to indicate that it was a case of simple chorea without disease of any of the organs.

Although the child was carefully nursed and remedies of various kinds were employed to strengthen the action of the heart and to support his general strength, he failed rapidly, and died of exhaustion a few days after I saw him.

I will now show you this little girl (Case 340), eight years old, whom I have had placed in a warm room so that she can be examined naked without harm.

CASE 340.

I.



Rheumatic arthritis. Endocarditis. Cardiac enlargement. Chorea. Female, 8 years old.

The history given to me when the child entered the hospital was that the mother had been subject to attacks of rheumatism. This child has not had any especial diseases, with the exception of an attack of measles when she was three or four years old, until she had an attack of rheumatic arthritis six months ago. At that time she was confined to bed with fever, and with pain, tenderness, and swelling in all her joints, especially of the knees and fingers. Although she recovered from the acute symptoms of the rheumatism, she has since then never been able to use her arms and hands, nor has she been able to walk much. I have no record of the condition of her heart during the attack of rheumatism, but so far as I can ascertain there was no evidence of cardiac disease prior to the rheumatism. During the course of the rheumatism there were no other especial symptoms noticed, except that her disposition was evidently much changed and she became peevish and fretful.

One week before entering the hospital she began to have choreic movements. They were moderate in degree, but incessant. A few days later the incoördination of the muscles was also noticed when she endeavored to speak or to swallow. There were continual choreic movements of the eyes, face, and fingers, and, although seemingly she could understand what was said, she was unable to speak clearly. She has been in the hospital ten days, and is, as you see, much emaciated. You will notice the incoördinate movements of all the muscles of the face, eyes, head, neck, body, and extremities. The peculiar look which occurs in these cases, and which can be expressed by embarrassment, is clearly shown here. The child feels that she cannot control her muscular movements, and cannot even fix her gaze on any object steadily. Although the case is a severe one, the mind is not affected beyond a slight degree of hebetude. On physical examination I find that the lungs are normal. On examining the cardiac region you notice that the impulse of the heart is outside of the mammary line and in about the sixth interspace. On palpation the contractions of the heart are found to be of an irritable nature, clearly felt, but not so strong as normal. At times there is a feeling as though the ventricular contractions were hesitating, and they are of an irregular form, which suggests that the incoördination of the other muscles is participated in by those of the heart. On percussion there is normal resonance to the right of the sternum and under its upper part as far as the third right interspace. There is dulness under the sternum, beginning at the second left interspace, extending across to the third right interspace, and involving the lower part of the sternum. I have marked the area of cardiac dulness in black. It extends upward to the left of the sternum as far as the second rib, then to the left and downward outside of the mammary line until it reaches the impulse of the heart in the sixth left interspace. On auscultation a murmur is heard most distinctly with the first sound at the apex of the heart, and is transmitted to the axilla and to both sides of the back. This murmur is transmitted to the base, but gradually lessens as the area of the large vessels at the base of the heart is reached. Nothing else abnormal is detected on a further physical examination of other parts.

This is evidently a case where during the course of a rheumatic attack an endocarditis in all probability developed. This endocarditis has been followed by enlargement, mostly represented by dilatation of the left ventricle. During the course of the rheumatism and of the cardiac complication the chorea has developed.

The prognosis in a case like this must be very guarded. In some instances the disease, or rather combination of diseases, grows rapidly worse, and the child dies seemingly from exhaustion. In cases of a milder form the child gradually recovers from its chorea and from its rheumatism, but is left with an organic disease of the heart from which it never recovers. The cardiac disease, however, can in most cases be much benefited by careful treatment, especially by rest in bed. In these cases the dilatation grows decidedly less, while the heart becomes stronger, and, as the chorea passes away, shows a normal area of dulness.

During the course of a case of this kind we must at any time expect in place of gradual improvement a decided increase in the severity of the symptoms. The valvular lesion of the heart may become much more extensive, assuming the ulcerative form which is usually so fatal. The pericardium may become affected, and broncho-pneumonia may occur as a complication. I shall therefore have to tell the parents of this child that she is in an extremely critical condition, and that for some days or weeks it will be impossible to say whether she will live or not.

The treatment of the case is with milk and stimulants. It is impossible for her to take solid food, and the milk is with the greatest difficulty introduced into her mouth. I will now have the nurse feed her (II., page 721), so that you can see how almost impossible it is for her to take the milk in her mouth or to swallow it, although she evidently is desirous of doing so.

I have tried various methods of administering the milk and stimulants in this case, but have found that the jaws close so spasmodically whenever a spoon is introduced between the teeth that the milk is usually spilled before she receives it. The method which I have found to be most successful is by this feeding-cup with a rubber nipple fitted to the neck of the cup. The rubber nipple is perforated with a large hole. The soft rubber does not incite the choreic movements of the jaw to the degree that anything hard would do. You see that by a little effort of sucking she takes the milk fairly well, though the difficulty in swallowing continues. The amount of milk which we endeavor to give her in e twenty-four hours, and which I think is sufficient to support her strength until the cute stage of the disease shall have passed by, is 1419.33 c.c. (3 pints). She is also taking bout 60 c.c. (2 ounces) of port wine in the twenty-four hours.

(Subsequent history.) The child remained in about the same condition for the next ree or four days, when the temperature rose to 38.8° C. (102° F.), and on the following ay to 40° C. (104° F.), and she complained of pain in the precordial region. On examiation, in addition to the cardiac murmurs a friction-sound was heard all over the cardiac rea, but especially in the neighborhood of the left nipple. The area of percussion dulness mained the same, and did not extend to the right of the sternum. The child moaned conderably during the day, and was very restless. The choreiform movements becoming more xaggerated, the port wine was increased in amount and 3.75 c.c. (1 drachm) of infusion f digitalis was given once every eight hours. Under this treatment the pulse grew a ittle stronger and the child's restlessness became less. The temperature also fell to 38.6° C. (101.5° F.), and on the following day to 38° C. (100.5° F.). The pain continued uring the next few days, and there was a certain amount of diarrhœa. The stimulant was ncreased to 120 c.c. (4 ounces) in the twenty-four hours. The child then became less estless, took more nourishment, and slept better. The diarrhœa ceased on the seventeenth ay from the time when she entered the hospital, and at this time she began to swallow etter and to speak more distinctly. During the next few days her general condition was mproved, and she seemed brighter. An examination of the knee-jerks at this time showed hat the tendon reflex was absent. There was great atrophy of the muscles. The liver as found to be somewhat enlarged, and there was slight dulness under the left clavicle, ut nothing definite was detected on auscultation. The lymph-glands were enlarged in oth axillæ. The temperature at this time varied from 37.5° to 38° C. (99.5° to 100.5° F.), he thirty-first day from the child's entrance into the hospital, and continued at this height or the next week. During this time the child remained in about the same condition, but rew much weaker and showed more hebetude.

CASE 340.

II.

Administration of milk in a severe case of chorea.

From the thirty-seventh day from the time when the chorea began the child grew uch weaker, had incontinence of urine, refused to take her nourishment, and on some ort wine being given to her vomited. She then was attacked with dyspnœa, which aused her to cry out loudly. On examination, dulness and diminished vocal resonance ere found in various parts of the chest, and a few hours later she died.

These charts (Charts 29 and 30) show the child's temperature from the time when she entered the hospital until her death.

CHART 29.

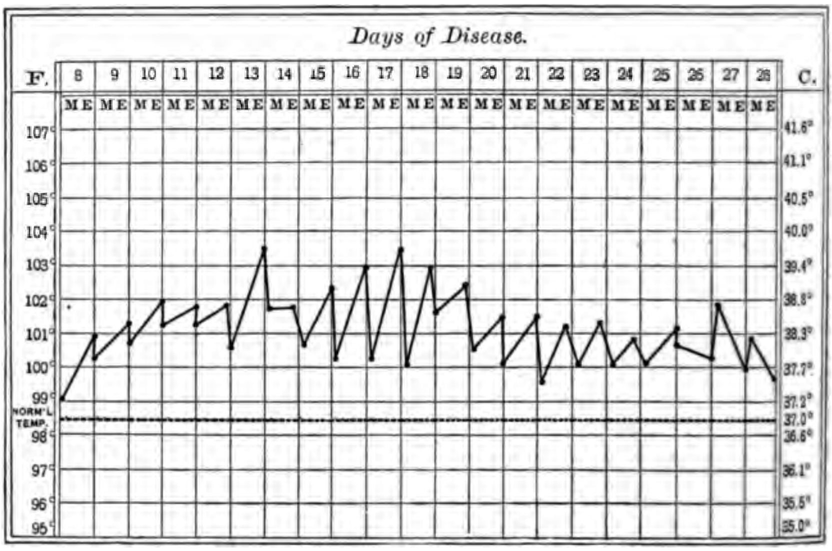
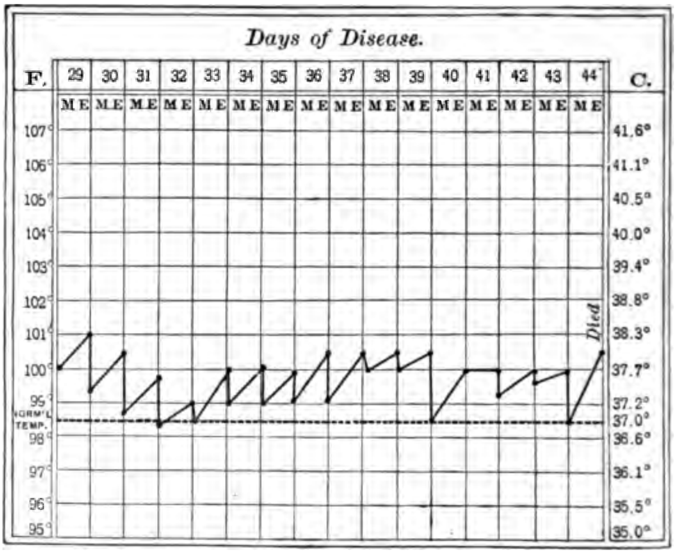


CHART 30.



The autopsy was made by Professor Councilman.

The head was not opened.

The peritoneum was normal.

The liver was enlarged, extending 4 c.m. (1½ inches) below the margin of the ribs.

In both pleural cavities there was a considerable accumulation of blood-stained fluid. The anterior mediastinum was thickened.

The pericardium at the apex of the heart was adherent to the left pleura, and about this area the tissues were thickened and œdematous. The right lung was slightly adherent pleura by comparatively late adhesions. The pleura of the lung was smooth, with xception of the adhesions just spoken of. The lymphatics over the surface of the a were greatly dilated. The upper left lobe of the lung was congested and gave a tion of small nodular masses in it. On section there was a distinct lobular consolida-throughout the upper lobe. The left lung was of a dark-red color, comparatively th on section, and somewhat solid. Muco-purulent matter could be squeezed out of ronchi. The chief characteristic of the lung was the extreme dilatation of the inter-ar spaces. The lymphatics all through the lung were visible. In the bronchi there onsiderable œdematous fluid. The blood-vessels at the base of the lung were free. ronchial glands were enlarged and reddened.

he left lung was not so adherent as the right. Over the posterior portion of the a there was a slight fresh pneumonia. The lung was somewhat compressed by the tion of blood, otherwise it was in about the same condition as the right lung. The rdial cavity was obliterated. The adhesions were easily broken down, except at the where the pericardium was greatly thickened. Its surface was covered by a thick of fibrin and exudation.

he heart was enlarged. Over its surface was a dense layer of fibrin. At the apex of eft ventricle, at a point corresponding to the adhesion of the pericardium, the myo-um felt soft and had a whitish infiltration. Beside this, corresponding to the intra-icular septum, there was a line of rather firm, thick, whitish nodules. The interior e right side of the heart contained moderately firm fresh clots. The surface of the ardium on the right side of the heart was pale and soft. Along the free border of the ulo-ventricular valve there were a few fresh vegetations. The left side of the heart was d, and the ventricle was thickened. The edge of the mitral valve was thickened and d, and there was distinct loss of substance in the thickened portion of the valve, which rregular and eroded edges. The muscular substance of the heart was pale, with small sh spots beneath the endocardium. These spots were very slightly elevated, and were or less circumscribed. Similar spots could be seen in the cardiac muscles. The sur-f the left auricle was thickened, and beneath the thickened area were numerous small sh points. The aortic valves were intact, save for a few fibrinous deposits at the edge contact. The beaded row of elevations described on the surface of the pericardium ponded to the course of the descending branch of the left coronary artery, and was bly due to thrombi with suppuration around them along the course of the artery.

he spleen was enlarged to the size of 10 × 8 × 3½ cm. (4 × 3 × 1¼ inches), and was aratively soft. Over its surface were a few small adherent thrombus-masses. On n the Malpighian bodies were extremely prominent.

he mesenteric lymph-glands were enlarged and slightly softened.

he liver was large, the bile-ducts were free, and the gall-bladder was slightly dis-d. The portal vein was free. The surface of the liver was dark red. The lobules prominent, and on section were slightly congested.

he pancreas was apparently normal. The suprarenal glands were normal. Both ys were of the same general size and appearance; in both the cortex was extremely and the capsule was easily torn off. In the cortex there was a slight diffuse staining. lomeruli were pale, but otherwise showed no change. In the lower ileum there was a enlargement of the follicles and of Peyer's patches. The glands at the root of each were enlarged and swollen. The left jugular vein was filled by a rather firm, slightly ive thrombus-mass, which extended downward into the subclavian vein and across the superior cava, into which it projected, and on the end there were a few soft fresh

he anatomical diagnosis of this case was,—
. Chronic pericarditis and mediastinitis.
. Acute ulcerative endocarditis.
. Thrombosis of innominate and left jugular veins.
. Broncho-pneumonia.
. Passive congestion and œdema of lungs.
. Adhesions of pericardium.

7. Dilatation of interlobular lymphatics.
8. Acute pleurisy, right side.
9. Hydrothorax, both sides.
10. Acute spleen-tumor.
11. Occlusion of descending branch of left coronary artery.
12. Acute swelling of bronchial and mesenteric glands.

Cultures made from various organs showed the presence of streptococci, but not of pneumococci.

EPILEPSY.—Epilepsy is presumably an organic disease of the nervous system in which the pathological lesion has not yet been determined.

The characteristic symptoms are attacks of unconsciousness with or without convulsions, with a great liability to a recurrence of these attacks through a long period of time. The transient loss of consciousness without convulsions which occurs in epilepsy is called *petit mal,* while the loss of consciousness with general convulsive manifestations is called *grand mal.* Convulsions precisely similar to those occurring in true epilepsy may occur in organic cerebral disease as the result of external traumatism or from other causes; such convulsions have been termed *epileptiform.* The term *Jacksonian epilepsy* is applied to localized convulsions which are the result of organic cerebral affections. These latter forms must not be confounded with true epilepsy.

It is important that a sharp distinction should be made between the convulsions of true epilepsy and the many reflex convulsive attacks which come from a variety of causes and arise from the hypersensitive condition of the infant's nervous system. These reflex convulsions so closely resemble the convulsions which occur in epilepsy that the great importance of distinguishing between the two diseases can hardly be exaggerated. In the infant's rapidly growing brain the irritability of certain motor centres is physiologically far greater than in later childhood and in adult life. This irritability is the source of nervous explosions produced by many causes often slight in their nature, and it is impossible to differentiate these explosions by their clinical symptoms alone from the convulsive attacks of epilepsy.

ETIOLOGY.—It is usually granted that the initial lesion of true epilepsy lies somewhere in the cortical motor centres of the brain, and that the epileptiform convulsion is an irritation of these centres. True epilepsy may of course originate in early infancy, and does so in a large number of cases. Whether, however, infantile convulsions may be the cause of epilepsy is a very different question. The fact is that we do not as yet know what produces epilepsy. The various etiological factors which are usually cited, such as fright, injury, and dentition, probably have nothing more to do with the production of the disease than to precipitate its development in an individual who is already predisposed to it. Inheritance as a cause of epilepsy will presumably, in the future, hold a much less prominent place than has been granted to it in the past.

There is no good reason for believing that reflex convulsions in themselves ever lead to true epilepsy. It is of considerable importance that we should be able to allay the natural alarm of parents by telling them, after the convulsions have ceased for a sufficient time to allow us to say that they are not epileptic, that there is no chance of their having produced an epilepsy which will develop later.

SYMPTOMS.—Epilepsy may begin in infancy or at any time throughout childhood, but a frequent time for its development is at puberty.

The *petit mal* may exist in different degrees of severity. In the mildest form, which may often pass unnoticed unless the attendants are especially on the watch for it, the child stops for a moment in its occupation, whether speaking, eating, or playing, while its eyes become fixed and it assumes a vacant expression. This condition may last for only a few seconds, when the child assumes its former occupation as though it had never been interrupted, and usually is not aware that anything has happened. In other cases this condition lasts a little longer, and slight twitching of the lower part of the face and of the extremities may occur. In other cases, again, the attacks are more severe, the child complains of being dizzy, staggers, has slight convulsive movements and turns pale, this condition lasting for a minute or more, and being quite marked, but without any total loss of consciousness. Momentary attacks of staggering sometimes occur alone in place of the attacks above described. At times these attacks of petit mal are the only manifestations of the disease, but in severe cases they are apt to be accompanied by occasional attacks of grand mal. They may occur as often as twenty or thirty times a day, or, on the other hand, they may be noticed only once in four or five days, and sometimes they are absent for longer intervals.

In the *grand mal* the attacks are of much greater severity. They are sometimes preceded for several hours by a feeling of malaise or general discomfort, but this is not always present. Patients sometimes have notice of the sudden onset of the attack, and such notice immediately preceding the convulsions and forming part of the attack itself is called the *aura*. This aura may be of different kinds. It is most commonly a sense of fulness or oppression in the epigastrium, from which something seems to rise into the throat, and unconsciousness supervenes. It may be, however, a pain or a sensation of numbness, tingling, or other form of paræsthesia in various parts of the body. Sometimes tinnitus is the first symptom. Frequently the patient has no warning whatever of the attack, but falls unconscious with or without a cry. The face becomes congested, and the eyes usually turn upward so that only the whites can be seen. After this follows the stage of tonic convulsions, which is sometimes so short that it is overlooked. Then come the clonic convulsions, which in typical cases are general, although the limbs on one side of the body are sometimes more affected than those on the other side. The movements of the limbs are apt to be very violent, the hands are clinched, the thumbs being flexed on the palms and the fingers closed over them. In many cases the patients froth at the mouth. In the more severe cases the children bite their tongues and pass their urine involuntarily. The duration of such attacks is usually five or ten minutes, but one attack may succeed another with little or no intermission. When the attacks follow one another in this way for several hours the patient is said to be in the *epileptic status,* and his condition as regards life is very serious. After the convulsion ceases the child's breathing becomes stertorous and the limbs are relaxed. Later, and before consciousness fully returns, the child often falls into a deep sleep, and on waking has no recollection of the attack, but complains only of headache and of mental confusion. Attacks often occur in the night, and in this case may be overlooked, the only evidence of them being that the child has wet the bed. In certain cases where only nocturnal attacks have been present we often have reason to believe that the disease has existed for considerable periods before its presence was suspected. In some cases in connection with the attacks there is a desire to walk or to run, so that the patient must be closely watched. In this condition children may walk straight against an obstacle, though they are more apt to stop when something comes across their path. Sometimes they walk or run in circles.

The cases of paroxysmal running described by Bullard are at times the early manifestations of an epilepsy which will develop later, though they may also be only the symptoms of hysteria, chorea, and organic cerebral disease.

Epileptic children are liable to bursts of ungovernable anger and violence lasting for hours, in which they may tear and destroy things, bite the mother or nurse, and are apparently for a time under the influence of illusions and hallucinations.

The condition of patients between the attacks is in the lighter cases and in the beginning of the disease usually quite normal. As the disease progresses, however, there is a tendency to mental impairment, and in the more severe cases, in contrast to the lighter ones, we are apt to find some enfeeblement of intellect, which at times may go on to an advanced dementia.

It has been considered by some of the most acute observers that those cases in which petit mal exists in connection with the more severe attacks are more liable to mental impairment than those in which the grand mal exists alone.

DIAGNOSIS.—As the convulsive attacks occurring in epilepsy cannot be distinguished clinically from similar attacks due to other causes, we are forced to differentiate epilepsy from other diseases by carefully eliminating other causes for the convulsions. We must also wait to see whether the attacks will continue indefinitely, in which case they are more likely to be epilepsy. A very fair illustration of the difficulty which may arise in diagnosticating infantile epilepsy is represented by these two infants whom I have had brought here to show you.

This infant (Case 341) was attacked at the age of ten months with general clonic convulsions. Previous to that time it had been mentally bright. It was then cutting the four upper incisors. One month later it again had a convulsion, the incisors having come through the gums. It is now two and one-half years old, and the convulsions have ontinued, varying in intervals and in severity. The child is now somewhat impaired nentally, but there have been no other symptoms of cerebral disease.

The diagnosis of epilepsy can be made in this case, but this was not warranted at the ime of its first convulsion, nor indeed for some time afterwards.

This second case (Case 342), eight months old, is, opportunely for your instruction, aving a general convulsive attack. You see that it is unconscious; that the muscles of he face and of all the extremities are in active motion; and that the eyes are turned up. This is the third attack that it has had to-day. The lower middle incisors are almost hrough the gum; the gum is not swollen or tense, and shows no indication for lancing. We must therefore look further for the cause of this nervous explosion. There is no evilence of anything in the ear, and the normal temperature aids us in eliminating the prolromal convulsions of one of the acute diseases with high temperature. On coming out of the convulsion previous to this one there was no evidence from paralysis or stupor that ny central nervous lesion had occurred.

So far as the clinical picture is concerned, this may be the beginning of an epilepsy, out the chances in a case like this are always that it is not one of epilepsy. The mother now remembers that she gave the infant last evening two or three beans which he managed o swallow. The case is probably one of reflex convulsions from gastric irritation.

(An emetic was given, the stomach was relieved of the beans, and the infant had no nore convulsions.)

The diagnosis of epilepsy is made from a continuance of the attacks after a considerable period without evidence of any organic disease or marked irritation. When the child bites its tongue during the attack and goes to sleep after the convulsion, or when there is temporary mental impairment after the convulsion, we have good reason to state that the convulsions are due to true epilepsy, especially if no symptoms of organic brain disease coexist.

Epileptic convulsions are easily distinguished from hysterical ones by the presence of consciousness in the latter, at any rate to a considerable extent. Hysterical convulsions in children are not very common, and almost never exist without the presence of other symptoms of hysteria.

PROGNOSIS.—The prognosis of epilepsy for life is, on the whole, favorable, and epileptics may live for many years.

As regards cure, the prognosis in cases beginning in early infancy is very serious. When the disease begins at the age of ten years or later a certain number seem to recover, at least temporarily. Many authorities consider that true epilepsy is never cured, yet undoubted cases exist where no convulsions take place for years.

TREATMENT.—The child should be treated at once, in order to avoid continuous shocks to its nerve-centres. Much benefit results from early attention to general hygienic conditions, to diet, and to protection from nervous disturbances.

The management of these cases demands constant watchfulness and tact, so as to regulate the surroundings of the child in such a way as to avoid all source of irritation and nervous excitement. The diet must be regulated according to the especial indications for each patient. Slight gastric irritation apparently produces more serious consequences than irritation of any other part of the body. A vegetable diet is usually indicated, but where the child does not thrive well on this it is advisable to give a certain amount of meat. Eggs are usually well borne.

The bromides in some form are, in my experience, the most useful drugs. It is often advisable in giving the bromides to change from one bromide salt to another, a greater effect being thus produced than by the constant use of one of them. Efficacious medical treatment depends more on the graduation of the doses, on the selection of the time for changing them, and on the determination of the intervals for administering them, than upon anything else. The best results in using the bromides are obtained by diluting the dose with a large quantity of water, 120 c.c. (4 ounces). As a rule, bromide of potassium has been found to be the most efficient and active of the bromides in cases of epilepsy. In giving the bromides it is well to begin with small doses, 0.12 to 0.24 gramme (2 to 4 grains), three or four times in the twenty-four hours, for the first year, and to double this amount for the second year. The dose should be increased gradually until the physiological action of the drug is noticed.

This treatment, at intervals of one or two weeks, should be carried on for long periods, and from six months to a year after the convulsive attacks have seemingly ceased.

This little girl (Case 343), who has been brought to the clinic this morning, is four and one-half years old. She was apparently a healthy infant. When she was ten months old she began to have convulsions, which were of a clonic type and infrequent at first, but when she was fourteen months old they became more severe and frequent. Since that time the convulsions have continued, and at one time she had fifty-four convulsions in forty-eight hours.

She was treated with bromide of potassium, beginning with doses of 0.12 gramme (2 grains) and gradually increased to 0.3 gramme (5 grains) four or five times in the twenty-four hours. Under this treatment the convulsions have become less frequent and her general health has much improved within the last year.

No other symptoms of disease have at any time been detected about this child, and the affection is simply represented by convulsive attacks followed by unconsciousness. Although the child shows considerable improvement at present, yet the probability is that she will never be entirely free from the epilepsy, and that as she grows older, especially as puberty is approached, the convulsive attacks may occur more frequently, and under these circumstances her mind may become more or less affected.

This child (Case 344), whom I have had brought to the clinic to-day to show to you as illustrating an extreme case of epilepsy, is three years old. She was healthy at birth, and remained so until she was two months old. At that time she began to have slight convulsive attacks, the cause of which could not be accounted for on a careful examination. During the earlier attacks she looked as if she were frightened. She would then scream, and become rigid and unconscious for about fifteen minutes, after which she would sleep three or four hours. These attacks occurred at all hours of the day and of the night. They have continued at irregular intervals, but are not now so frequent as in the first year and a half. During the first year she seemed as bright as any infant of her age, and developed normally.

She has been treated with the bromides, and they seem to have been of some benefit, but have not produced a permanent cure.

During the last year her mental condition has been much affected, and she evidently has a permanent injury of the brain produced by her epilepsy. She has never been able to sit alone or to bear her weight on her feet. She cannot feed herself, and she understands that is said to her. The head is of about the normal size. The face and eyes ant expression, and she has to be taken care of as though she were an infant in nonths of life.

is case there is no history of epilepsy or of any especial nervous disorder in the r of traumatism or of any serious disease which could have produced this nervous e. We can only say, therefore, that it is a case of chronic epilepsy starting from iown cause and resulting in permanent idiocy.

oy (Case 345) is ten and one-half years old. There is no history of epilepsy or of ease in his family. He has never had any especial diseases, but for the last six and s he has been attacked with convulsions occurring about once every three weeks. st six months the attacks are said to have been more frequent and severe. He at had as many as five in one week, and some of the attacks have lasted fifteen min- re. He states that just before one of these convulsive attacks he feels frightened He then loses consciousness and falls to the ground. The expression of his face ll, as though the continued shocks to his nervous system were producing a certain mental disturbance. He answers questions, however, readily and intelligently. l examination shows nothing abnormal.

CASE 345.

Epilepsy, 6½ years' duration. Petit mal. Male, 10½ years old.

entering the hospital, two weeks ago, he has had a number of convulsions and losely watched, and there seems to be no doubt that the attacks are real and not The attacks have usually occurred at night, but sometimes also when he has nd about the ward. The attack is usually ushered in by a loud cry, and he is to be in a state of clonic convulsion. He froths at the mouth, but has not bit- gue. The attacks have lasted for about fifteen minutes, and have then been fol- oma and prolonged sleep for some hours. In addition to the other convulsive there has been much twitching of the face during the attack. The eyelids are f open, and the eyes rolled upward and inward. The pupils react only slightly attack, and the eyeballs are not sensitive to touch. As the convulsive seizure y, the reaction of the pupils gradually returns. During the attack there is no ensation produced by pricking with a pin. Some of the attacks are preceded by and an attempt to get out of bed, so that he has to be restrained, and sometimes

LECTURE XXXVI.

III. FUNCTIONAL NERVOUS DISEASES.

(Organic nature not yet shown.)

(1) PROBABLY CENTRAL.

HYSTERIA.—HYPNOTISM.—CATALEPSY.—SIMULATED DISEASES.—INSOLATION.—CONCUSSION.—TEMPORARY AMNESIA.—TEMPORARY APHASIA.—ARRESTED PSYCHICAL DEVELOPMENT.—RETARDED SPEECH.—HEADACHES.—VERTIGO.—SENSITIVE SPINE.—TETANY.—PAVOR NOCTURNUS (Central).

TO-DAY, gentlemen, I shall speak of a class of cases which you are liable to meet with interspersed among the patients with definite diseases whom you are called to see.

These cases are called functional, and are represented by either a temporary suspension of, or a perverted use of, the normal physiological functions of the nervous system. We have at present no sufficient evidence to justify us in classifying these diseases as organic. These functional nervous phenomena play a *rôle* of considerable importance in early life, as they occur much more frequently at this period than they do in adults. The various functions of the nervous system in early life are in the process of development, and are not so perfected as they are in the more mature subject; in fact, they are in a state of unstable equilibrium: hence shocks of various kinds easily cause temporary disturbances which, not being grossly organic, can pass away after a period of rest.

This class of functional disturbances may be divided into nervous phenomena apparently resulting (1) from some affection of the nervous centres, and (2) from some irritation of the peripheral nerves.

I shall first speak of those functional diseases which are supposed to be of central origin. Of these hysteria is perhaps the most difficult to differentiate correctly and to understand, and I shall therefore begin with that disease.

HYSTERIA.—Hysteria is a functional disturbance of the cerebral centres represented, according to Möbius, by a state in which ideas control the body and produce morbid changes in its functions. The name is a misnomer, but it has been adopted so generally that we must use it for the present.

We know very little about the etiology of hysteria. Well-marked instances of the disease occur in early life, usually in the middle and later periods of childhood.

An inherited nervous organization or highly exciting surroundings, combined with a lack of proper home discipline, appear to present as likely ld for the disease to develop in as any conditions, such as fright, which parently, at times, directly lead to it.

The mere presence of emotional or imaginative conditions in children es not constitute hysteria. For the existence of the disease it is necessary have definite symptoms, either a markedly disorganized mental state, ralysis, anæsthesia, or some serious loss of function (amaurosis, deafness, sphagia).

SYMPTOMS.—The symptoms in this most protean of diseases are innu- rable. Convulsions and paralysis are quite common, while dysphagia, aurosis, and anæsthesia are met with only in the very severe cases, and not often seen in America. Anæsthesia is especially interesting as rep- enting a pure type of the disease, and is usually on one side of the body. ildren perhaps only two or three years of age affected by hysteria will netimes allow themselves to be pricked on the anæsthetic side of the face thout wincing.

Hysteria in children as usually seen in America is marked by the emo- nal conditions of the child, and by the presence, in many cases, of a fixed a relating to its own physical condition. The child believes that it can- t perform certain actions or functions, and hence does not perform them. ere probably has often been in the beginning some real difficulty or dis- rbance of the performance of these functions, such as pain, which has ssed away or which is not sufficient to produce the present condition.

The most common symptoms, aside from the mental condition, are) convulsions, (2) paralysis, and (3) anæsthesia.

(1) The convulsions are distinguished from those of epilepsy by the sence of loss of consciousness. The patient never seriously injures him- f in falling, and does not bite his tongue. He does not sleep after the ack.

(2) The paralysis is often of the spastic form, and may be either hemi- gic or paraplegic. In this form the limbs are rigid and the knee-jerks exaggerated. It may, however, be of the flaccid variety, with the ee-jerks diminished or absent. It is distinguished from the organic rms of paralysis by the normal reaction of the muscles to electricity, by absence of atrophy, by the absence of any affection of the sphincters, d at times by the presence of anæsthesia.

(3) When anæsthesia occurs it is usually irregular in distribution, occur- g in patches, or else it has the same distribution as in cerebral organic sease. It is often variable, changing more or less from day to day.

Although almost any symptom may occur in hysteria, yet the lack of iformity in the grouping of the symptoms, and the combination of symp- ms which belong to entirely different diseases, are of great aid in making e differential diagnosis from these diseases.

We sometimes meet with an exaggerated hysteria in children. The tacks are represented by screaming, running, jumping, and a feeling of ing pulled about; they may last for hours, or for days; their duration, a general feeling of uneasiness appears to precede the attack. He has been in this condition this morning, and has therefore been undressed and put to bed with the clothes simply thrown over him. As you now see him, he is evidently about to have an attack.

He is very restless, and has thrown back the bedclothes. You see that he now attempts to rise from the bed, and that his eyes are somewhat vacant and staring. These premonitory symptoms may simply represent an attack of petit mal, as a number of times they have gone no further, or may be the forerunners of a general convulsive attack such as is represented by grand mal.

(This especial attack happened to be represented by the form petit mal, and soon passed off without a convulsion.)

His pulse has been regular, and has varied from 70 to 90. His respirations have been about 24 in the minute. His temperature has been normal.

The treatment of this boy has been with bromide of potassium, but has not been followed by marked benefit. He is probably a case of chronic epilepsy which can never be cured, and which, according to Dr. Bullard's opinion after carefully examining him, will have to be taken care of in an institution for feeble-minded children.

This strong, healthy-looking boy (Case 346), seven years old, I have had brought to the clinic to-day to show you as another form of epilepsy. There is no history of organic nervous disease in the family. He was born after a difficult delivery, and on the following day had a number of convulsions, which continued at intervals for several days. They were of a clonic general variety, and were apparently relieved by small doses of bromide of potassium. During the first year, although the convulsions did not return, he had from time to time slight attacks, in which he turned pale and became almost unconscious. These attacks, however, lasted for only a few minutes. It is reported that during the first six months, although his physical development was fairly normal, he did not notice anything and seemed almost blind. After that time, however, his mental condition improved, although he seemed a little backward in comparison with other infants of the same age. During the first year and a half of his life his left leg seemed smaller than the right and was a little shorter, but no especial paralysis was noticed, and by the time that he was three years old no difference in the size of the limbs was detected. When he was one year old he was able to sit alone. His teeth were cut at the usual time. When he was two years old the measurements of the head showed that it was of about the normal size and the anterior fontanelle was closed. Towards the end of the second year he began to talk. When he was two and a half years old he had cut all his teeth and was well and strong, had a good appetite, and could walk well. He had, however, shown signs of mental disturbance. He was fretful, was subject to explosions of temper, and had to be carefully looked after to see that he did not hurt himself or the other children in the family.

When he was four years old he began to have convulsions of a clonic general type, occurring at night and ushered in by a scream. During these attacks he frothed at the mouth, was unconscious, had stertorous breathing, and after five or ten minutes would fall into a deep sleep from which he could not be aroused for a number of hours. On the following day he would be somewhat dull and fretful, but these symptoms would then pass away, and the convulsions would not return sometimes for a number of months.

As you see him to-day he is physically well developed. His mental impairment is, however, very evident; his eyes are not bright, he has a rather vacant, idiotic expression, and, although he has learned to read, he does not show as much intelligence as his brother who is four years old. Physical examination shows nothing abnormal.

This case illustrates a cerebral injury taking place at birth. This injury has left its mark on the brain in such a way that entire recovery will probably never take place. The convulsions are evidently epileptiform,—that is, they are caused by an irritation of some of the motor centres produced by the original cerebral lesion.

In a case of this kind treatment by drugs is usually without benefit. The attacks seem to be somewhat controlled by the bromides. There has been at times much constipation; when the constipation is excessive the attacks are more likely to occur, and it has been found that if the bowels are carefully attended to and the constipation thus combated he is in a better condition. It is a case in which much benefit can be obtained by mental raining, and he should be placed in some institution devoted to the training of feeble-minded children or in the hands of some expert in this branch of psychology.

INSANITY.—Insanity in children is very rare. In the ordinary forms of insanity no definite pathological lesion has been found which would account for the symptoms presented. Such changes as have been detected come very late in the disease and seem to be secondary. In paretic dementia, however, we find a special form of cortical interstitial encephalitis.

Instances of mania and melancholia at times occur. Hallucinations, which are a common symptom in the insanity of adults, occur in children usually in connection with the delirium of fever, or more rarely with epilepsy, as I have already described. Insanity is met with in children at any age; it is extremely rare before puberty, but then becomes more frequent.

The prognosis of insanity in children varies according to its form. Acute mania and melancholia are said to recover generally. True paretic dementia is never known to recover.

LECTURE XXXVI.

III. FUNCTIONAL NERVOUS DISEASES.

(Organic nature not yet shown.)

(1) PROBABLY CENTRAL.

Hysteria.—Hypnotism.—Catalepsy.—Simulated Diseases.—Insolation.—Concussion.—Temporary Amnesia.—Temporary Aphasia.—Arrested Psychical Development.—Retarded Speech.—Headaches.—Vertigo.—Sensitive Spine—Tetany.—Pavor Nocturnus (Central).

To-day, gentlemen, I shall speak of a class of cases which you are liable to meet with interspersed among the patients with definite diseases whom you are called to see.

These cases are called functional, and are represented by either a temporary suspension of, or a perverted use of, the normal physiological functions of the nervous system. We have at present no sufficient evidence to justify us in classifying these diseases as organic. These functional nervous phenomena play a *rôle* of considerable importance in early life, as they occur much more frequently at this period than they do in adults. The various functions of the nervous system in early life are in the process of development, and are not so perfected as they are in the more mature subject; in fact, they are in a state of unstable equilibrium: hence shocks of various kinds easily cause temporary disturbances which, not being grossly organic, can pass away after a period of rest.

This class of functional disturbances may be divided into nervous phenomena apparently resulting (1) from some affection of the nervous centres, and (2) from some irritation of the peripheral nerves.

I shall first speak of those functional diseases which are supposed to be of central origin. Of these hysteria is perhaps the most difficult to differentiate correctly and to understand, and I shall therefore begin with that disease.

HYSTERIA.—Hysteria is a functional disturbance of the cerebral centres represented, according to Möbius, by a state in which ideas control the body and produce morbid changes in its functions. The name is a misnomer, but it has been adopted so generally that we must use it for the present.

We know very little about the etiology of hysteria. Well-marked instances of the disease occur in early life, usually in the middle and later periods of childhood.

An inherited nervous organization or highly exciting surroundings, combined with a lack of proper home discipline, appear to present as likely a ld for the disease to develop in as any conditions, such as fright, which parently, at times, directly lead to it.

The mere presence of emotional or imaginative conditions in children es not constitute hysteria. For the existence of the disease it is necessary have definite symptoms, either a markedly disorganized mental state, ralysis, anæsthesia, or some serious loss of function (amaurosis, deafness, sphagia).

Symptoms.—The symptoms in this most protean of diseases are innu-erable. Convulsions and paralysis are quite common, while dysphagia, aurosis, and anæsthesia are met with only in the very severe cases, and e not often seen in America. Anæsthesia is especially interesting as rep-enting a pure type of the disease, and is usually on one side of the body. ildren perhaps only two or three years of age affected by hysteria will metimes allow themselves to be pricked on the anæsthetic side of the face thout wincing.

Hysteria in children as usually seen in America is marked by the emo-nal conditions of the child, and by the presence, in many cases, of a fixed ea relating to its own physical condition. The child believes that it can-t perform certain actions or functions, and hence does not perform them. ere probably has often been in the beginning some real difficulty or dis-rbance of the performance of these functions, such as pain, which has ssed away or which is not sufficient to produce the present condition.

The most common symptoms, aside from the mental condition, are) convulsions, (2) paralysis, and (3) anæsthesia.

(1) The convulsions are distinguished from those of epilepsy by the sence of loss of consciousness. The patient never seriously injures him-lf in falling, and does not bite his tongue. He does not sleep after the tack.

(2) The paralysis is often of the spastic form, and may be either hemi-egic or paraplegic. In this form the limbs are rigid and the knee-jerks e exaggerated. It may, however, be of the flaccid variety, with the ee-jerks diminished or absent. It is distinguished from the organic rms of paralysis by the normal reaction of the muscles to electricity, by e absence of atrophy, by the absence of any affection of the sphincters, d at times by the presence of anæsthesia.

(3) When anæsthesia occurs it is usually irregular in distribution, occur-ng in patches, or else it has the same distribution as in cerebral organic sease. It is often variable, changing more or less from day to day.

Although almost any symptom may occur in hysteria, yet the lack of niformity in the grouping of the symptoms, and the combination of symp-ms which belong to entirely different diseases, are of great aid in making e differential diagnosis from these diseases.

We sometimes meet with an exaggerated hysteria in children. The tacks are represented by screaming, running, jumping, and a feeling of eing pulled about; they may last for hours, or for days; their duration, however, is usually long,—at times, with intervals, over a year. No signs of organic disease are found in these cases; they seldom injure themselves, and are finally cured by moral influence, change of scene, and good hygienic surroundings.

Hysteria occasionally causes children to present symptoms of serious disease of the spine and joints. This most often follows some slight injury, but may occur spontaneously.

PROGNOSIS.—The prognosis in cases of hysteria is, as a rule, favorable.

DIAGNOSIS.—Generally, the diagnosis is not so difficult as in adult life, because the child is not able to control its sensations of pain and fear so completely as is possible with adults. In surgical cases, however, where hysterical affections simulate most closely organic disease of the joints, the diagnosis is often attended by extreme difficulty. The application of strong currents of electricity will usually show that the anæsthesia is not real.

A complete differential diagnosis of hysteria would occupy more time than I can give to the subject, and I have therefore merely outlined the general principles by which you are to be guided in diagnosticating these cases.

TREATMENT.—The treatment of hysteria is to break up at once the harmful home surroundings, if such exist, and by means of gentle but firm compulsion to make the child understand that its symptoms are unreal. The various local symptoms connected with the digestion and general health of the child should be carefully treated, as the hysterical symptoms are often largely dependent on conditions of this nature.

This little girl (Case 347), ten years old, is about to leave the hospital, as she has recovered entirely from the disease for which she was brought here for treatment.

The history of the case is that her parents are living and well, and that there are a number of other healthy children in the family. This child had always been well until eighteen days before she entered the hospital. At that time she complained of headache, and on going to school returned feeling sick and apparently unable to speak. She is said to have been unconscious at times, to have had spasms, and to have been very restless at night. She evidently had had great lack of care in her home life, and had been given only poor food. She showed the evidence of this lack of care in the condition of her skin and her digestion on entering the hospital. A physical examination showed nothing abnormal in connection with the thorax and abdominal organs. The pupils were slightly dilated, but were equal and reacted to light. The knee-jerks were decreased. There was no ankle-clonus. She was apparently unable to walk, and she lay in bed taking no notice of anything, but winked her eyes if anything was thrust towards them. Her hearing did not seem to be especially impaired. She lay in bed in a very limp condition, with the legs drawn up in various positions. Her head kept rolling from side to side, and occasionally was retracted. When asleep her head was retracted so as to make nearly a right angle with the body. It was difficult to feed her, as she would not swallow. Her temperature was 37.2° C. (99° F.), her pulse 66, and her respirations 16. When being examined she cried out a great deal.

She was given plenty of good food, and in three or four days her condition was much improved. She took her food well, but was apparently unable to feed herself. A few days later she showed more intelligence, and on being taken up and dressed it was found that she could sit alone and could walk a little with support. On beginning to walk she threw her legs about wildly, but after being scolded she walked much better. At one time when she was sitting quietly in a chair the visiting physician came into the ward, and she imme-

allowed herself to slip from the chair and roll onto the floor, but evidently was care-
to hurt herself. She at this time cried out a great deal, but stopped when no notice
en of her. She was still unable to speak, and, although she could sit up in a chair,
itly noticed nothing.

neteen days after entering the hospital she appeared much brighter, and began to
ght notice of what was going on about her. When questioned, she moved her lips as
t to speak, but made no sound. She continued to improve slowly, and a few days
id "sister," understood what she was told to do, and attempted to do it. She also
three steps without being assisted. Some days later it was found that she would
almost any word that was said to her, but in a whisper. After this she improved
and began to articulate fairly well, but slowly and with an effort. She also spoke
rily two or three times. She could not walk without assistance, as she would put
too far forward. She had been very much constipated through the whole attack,
his time the constipation grew less. A definite training of the arms and legs was
gun by means of passive movements and massage. Under this treatment she has,
see, greatly improved, and to-day, the thirty-fifth day from the time when she
the hospital, has recovered completely. She speaks and walks, although she is still
awkward.

ave concluded that this is a case of hysteria, as she has shown no definite symptoms
other disease, and on account of the emotional character of her symptoms since she
n in the hospital.

YPNOTISM.—Hypnotism is an artificial mental condition which can
duced in children as well as in adults. It is supposed to be a tempo-
beyance of the powers of the higher cerebral centres. In the ordinary
he child is thrown into a condition in which the consciousness of his
al surroundings is lost. This condition in outward appearance closely
les sleep, but is produced artificially and can be artificially removed.
the sensation of pain can be temporarily abolished, at least to a con-
ble extent. For this reason it has been supposed that it might be
in the treatment of cases requiring minor surgical operations. It has
een advocated by some physicians as a form of treatment in various
es; but our experience at the Children's Hospital has proved it to be
ient.

ATALEPSY.—Catalepsy is only a symptom. It denotes a condition,
ntly of cerebral origin, in which, together with total or partial loss of
usness, the limbs assume a peculiar form of rigidity called *waxy*, and
for a considerable time in any position in which they may be placed.
urs at all ages, but is very rare in childhood. The youngest case that
w of is that of a little girl three years old, reported by A. Jacobi.

e prognosis and treatment are those of the primary disease. There is
cial treatment for a single attack.

MULATED DISEASES.—On the boundary-line between children
vidently are suffering from the need of judicious discipline and those
nay be said to have the definite disease hysteria, is a class of cases
ich *simulation* appears to play an etiological part. These children are
y in the later period of childhood, and seem to have such perverted
ons of their nervous centres as actually to represent pictures of diseases
are easily proved not to be present. Deafness, blindness, pains of all

varieties, palpitation, dyspnœa, vomiting, spasmodic attacks of various kinds, and many other symptoms arise, and, may persist for long periods.

The best treatment for these cases is at once to show the child that you know his symptoms are unreal and of no importance.

You will remember the boy (Case 348), ten years old, who was brought by his mother to my clinic a few months ago with a history of convulsions which had been going on for two years, once or twice in a month. He was well nourished and robust-looking. The information was elicited from his mother that he never hurt himself when he fell down in a convulsion, and that the attacks followed attempts to make him do something which he did not wish to do. You may remember that I then suggested in his hearing that he should be sent to prison, and that he immediately fell on the floor, had a violent convulsion, foamed at the mouth, and was apparently unconscious. I then picked him up and told him that if he ever behaved in that way again he would surely be shut up in prison away from his mother. He instantly recovered, and has since been a reasonable member of society. You must be prepared to meet with all these different phases of nervous manifestations in early life, and learn to recognize to which class of nervous diseases they belong.

INSOLATION.—Heat-insolation, or heat-stroke, is a condition apparently represented by a functional disturbance connected with the cerebral circulation and produced by heat. This affection in varying degrees is of somewhat frequent occurrence in children, and is supposed to be accompanied by a hyperæmia of greater or less intensity of the meningeal blood-vessels. It is met with most commonly in the middle period of childhood, because at that age the child is most likely to be exposed to the influences which produce it.

The clinical picture of this class of cases is, as a rule, quite characteristic. The child has perhaps been playing on a hot summer's day somewhat more vigorously than usual, possibly romping with an older child of more highly developed nervous resistance, getting intensely excited, and greatly overtaxing its muscular strength. It may be that it has been exposed to the direct rays of the mid-day sun; or it may have been playing in some covered but heated and stifling place. The nurse of the child, noticing the extremely flushed condition of its face and head and its excited, sparkling eyes, takes alarm and hurries it to its home. Intense headache soon comes on, and in a few hours delirium may supervene. The skin is hot, dry, and reddened; there may be vomiting in the beginning; the carotids and temporal arteries throb perceptibly. The heart's action is violent, and the temperature is raised to 38.9°–39.4°–40° C. (102°–103°–104° F.); the pulse is much accelerated, perhaps 140 to 150, and is full, but usually rhythmical. The conjunctivæ are congested and the pupils contracted. Photophobia to a greater or less degree is almost invariably present. Beyond this there may be no symptoms except a slight amount of muscular twitching, and in some cases a convulsion may occur if the temperature runs as high as 40°–40.6° C.

104°–105° F.). The temperature, however, in accordance with the rule in his disease as in others which occur in children, does not always produce he same or equally severe symptoms. Thus, a temperature of 39.5° C. 103° F.) may in one individual give rise to marked nervous symptoms, rhile in the next child that you are called upon to see of the same age and rith the same disease, a temperature of 40° or 40.6° C. (104° or 105° F.) nay produce no nervous symptoms whatever, beyond possibly a slight pathetic condition. Convulsions may occur as a very common form of ervous explosion where fever and disturbance of the cerebral circulation re present, but, as a rule, this symptom is absent.

PROGNOSIS.—Be careful as to the prognosis which you give in these ases. Although they often simulate closely a beginning meningitis, a disease n which the prognosis, as I have already told you, is unfavorable, yet they re very amenable to treatment, and should therefore be carefully differeniated from that disease. In very severe cases the children may, of course, lie of insolation, as do adults.

DIAGNOSIS.—The diagnosis from meningitis is based upon the history, he milder grade of the symptoms, except the headache, and finally, in loubtful cases, the quick recovery and speedy disappearance of the fever.

TREATMENT.—The treatment of heat-insolation should be prompt and rigorous. A stimulating enema of salt, one teaspoonful to a quart of vater, should first be given. The child should then be placed upon a bed rotected by a rubber sheet in a cool, darkened room; a warm mustard ack should be applied to the lower extremities, and the neck and chest gently sponged with water at 25° C. (77° F.) for fifteen minutes out of every our. Leiter's coil should be applied to the head with water at 5° C. (41° F.); bromide of potassium should be given, 0.3 gramme (5 grains) every hour or four doses; a little iced milk may be taken if the child cares for it, not nore than one or two ounces at a time; and complete rest and quiet for at east twenty-four hours are usually indicated. The child should be watched carefully for some days and not allowed to play actively enough to get heated. Great care should be taken for the rest of the summer to protect the child from the direct rays of the sun, as after one attack the cerebral circulation remains in a very sensitive condition for a considerable period.

I have found in my notes an account of two cases of this kind occurring in my practice, which I shall report to you.

A boy (Case 349), ten years old, healthy and well developed, was perfectly well on the morning of August 20. The weather was hot and sultry. He played unusually hard with some older boys for several hours in the sun. He then went into the house at 3 P.M. with his face and neck intensely reddened. The skin was hot and dry, the blood-vessels were throbbing, and there was severe frontal headache. His temperature was 40.5° C. (105° F.); his pulse was 160, full and bounding, and the respirations natural. He complained of photophobia. He was put to bed in a darkened room and the bowels were moved freely by an enema of soap and water. His head and neck were sponged with ice-water. At 4 P.M. he was slightly delirious, but could be aroused; his temperature at this time was 40° C. (104° F.). Bromide of potassium in doses of 0.3 gramme (5 grains) was given at 4 P.M. and at

5 P.M At 6 P.M. the headache was less, and he fell asleep. He awoke at 10 P.M., and the temperature was found to be 38.9° C. (102° F.). He then took some iced milk and a dose of bromide of potassium. Later in the night the temperature was found to have fallen to 37.7° C. (100° F.), and the pulse to be 100. On the following day the temperature was 36.7° C. (98° F.), and the pulse 90. He complained of slight headache, but there was no photophobia. He felt weak and drowsy, and was kept in a dark room all day, taking small doses of milk.

On the following day he felt well, had a good appetite, and was up and dressed.

On the next day after driving in an open carriage in the sun for an hour he had a headache of moderate severity, but no fever, and was free from headache and perfectly well on the following day.

During the next two or three years, although he did not have any recurrence of a severe character, from time to time during the hot weather he showed that his cerebral circulation was still in a sensitive condition, as slight exposure to the rays of the sun caused considerable headache.

The next instance of this kind was a boy (Case 350), five years and three months old. On August 24 the weather was hot and sultry. The boy was perfectly well during the day, and was not exposed to any especial excitement or exertion. He went to bed well. The night was hot, no air was stirring, and the room in which he slept was very hot, close, and oppressive.

The next morning he awoke at 5 A.M. with severe frontal headache; he was very drowsy, had no appetite, and his temperature was found to be 38.9° C. (102° F.). He was kept all day in bed in a cool room. At times he would cry out from the pain in his head. His mind was perfectly clear. A dose of bromide of potassium, 0.3 gramme (5 grains), was given at 6 P.M. His temperature at that time was found to be 39.5° C. (103° F.); the pulse was 140, full and bounding. In the night he became delirious and had to be closely watched, as he would jump out of bed and cry out with pain in his head. Cold compresses were applied to his head during the night, and on the following day the symptoms were much relieved. Half a Rochelle powder was given in the morning and repeated in two hours. This was followed by a free movement of the bowels. He felt dull and complained of headache, but at 6 P.M. the symptoms were much relieved, and his temperature was found to be 36.7° C. (98° F.) and his pulse to be 100. On the following day he was reported to have had a good night and to have awakened perfectly well.

For two or three years following the attack he was liable to have attacks of this kind, either from undue exposure to the sun or at night if the room in which he slept happened to be ill ventilated and hot.

CONCUSSION.—By concussion we mean clinically a group of symptoms following some physical shock, with its resulting traumatic irritation of the nervous centres. I have met with a number of instances of this nervous phenomenon.

One was the case of a boy (Case 351), four years old, who fell from a table to the floor. I saw him an hour later, and found that his skin was cool, and his pulse slow, 60, and that he was nauseated and had been vomiting. No evidence of traumatic injury or proof of an organic lesion could be found. After a few hours the symptoms gradually improved, and he was perfectly well on the following day.

These indefinite symptoms are usually ascribed to the brain as the seat of irritation.

The treatment of a case of this kind is simply by perfect rest and quiet in a darkened room, with hot applications to the feet and abdomen, and small and repeated doses of stimulants given by enemata until the stomach is able to retain them, the treatment being continued until the circulation is normal and the pulse strong.

The next instance of this kind was the case of a little girl (Case 352), sixteen months whom I saw in consultation with Dr. Townsend.

The child was perfectly well, and was not of an especially nervous temperament. She ld speak a number of words and could walk. While sitting in her baby-carriage one at the top of a hill, another child took hold of the carriage and pushed it with great dity down the hill. The carriage was tipped over, and the child was thrown out on the walk, apparently striking on her head. She was unconscious when she was picked up, no signs of injury beyond a slight bruise on the right side of the head could be detected. re was no vomiting. Her extremities were cold. Consciousness soon returned, and ing abnormal could be detected on a physical examination. The pulse was 120, and temperature 36.6° C. (98° F.). After the accident she had no appetite, and became very s and easily frightened. The bowels were moved regularly, a slight amount of blood, ever, appearing in the first discharge which occurred after the accident. It was difficult to make her go to sleep, and she would wake up screaming and at times not knowing one. For several days she could not make use of the words which she ordinarily did, did not recognize her father, but was afraid of him, while before the accident she yed playing with him. She seemed to have the same fear of a number of other people he house, but did not show any symptoms of fear when being examined by Dr. Townd or by me, although we were entire strangers to her. These symptoms continued for e weeks, gradually subsiding, and were not accompanied by any other abnormal conon, such as a rise of temperature. The child recovered entirely.

The treatment of the case was simply to keep her in a rather dark room separate from rybody but her mother.

I have here to-day a number of instances of other central and functional eases to show you.

As the causes of these nervous manifestations are manifold, and as know nothing about their pathology, I can best describe their symptoms d treatment by describing individual instances of the various cases of this nd which have come under my observation.

TEMPORARY AMNESIA.—Here are two cases which belong to a ass of nervous disturbance which is represented by temporary amnesia.

This child (Case 353), now ten years old, was playing when he was nine years old an asphalt tennis-court with some older boys. One of the boys threw him down on e hard court so that he struck the back of his head. He got up, but felt dizzy, so that did not attempt to play any more, but sat looking at his playmates and occasionally king foolish remarks. This finally attracted the notice of his companions, who took him me. He was put to bed, and seemed drowsy, but did not have any nausea or any other mptoms, except that he could not remember anything, even that he was present at the dding of his aunt on the previous day. He articulated plainly and spoke naturally. ter sleeping for about twelve hours he woke up with his memory perfectly restored, cept that he had a very dim remembrance of what had happened to him. Since the cident his mental condition has been normal, and as you see him to-day he is a bright, ll-developed boy.

The probable cause of his amnesia was a physical shock with resulting abeyance of nction in the nerve-centres connected with memory.

This next boy (Case 354), thirteen years old, is a case of the same kind. While running, about six months ago, he struck his head against a tree. I saw him three hours later. had walked home, but was a little nauseated, and was put to bed. I found that he had rtial loss of memory and was drowsy, but that he had no especial pain. He was pertly well on the following day, and is, as you see, an intelligent boy, without mental turbance of any kind.

TEMPORARY APHASIA.—An instance of suspension of the cerebral function nnected with the elaboration of words is illustrated by Demme's case (Case 355) of a child six years old, who, previously well and bright, suddenly lost the power of speech. This phenomenon occurred during an operation for talipes, which was being performed without an anæsthetic. After the operation the child was perfectly well, but was unable to elaborate words until the ninth day, when she began to use the one word "mamma" for everything that she wanted to say. She then gradually increased her vocabulary until the twenty-first day, when her aphasia disappeared entirely, and she developed mentally and physically in a normal manner.

ARRESTED PSYCHICAL DEVELOPMENT.—Arrested psychical development is a term used in speaking of an apparent lack of mental growth which is sometimes met with in infancy. So far as we know, it is a functional and not an organic condition of the brain. Infants with this affection develop both mentally and physically for a variable period, perhaps five or six months, and then continue to develop physically but cease to develop mentally. This condition lasts for a variable period of months, when they begin to develop mentally again, and, although for some time they are backward in comparison with other children of their age, they finally show no trace of an abnormal mental condition.

Arrested psychical development seems to be rather commonly associated with rhachitis, and may also occur in the course of severe illnesses, but nothing else is definitely known concerning it.

RETARDED SPEECH.—At varying periods during the latter part of the first year and the beginning of the second year infants begin to make their first attempts to speak. By the middle of the second year they are usually able to communicate their ideas by means of short, broken sentences. In the third year most children speak quite plainly, though they do not correctly use the prepositions and adverbs until some years later. When during the second year the power of speech does not develop with the usual rapidity, it is spoken of as retarded speech.

This lack of power to speak may be from a simple lack of development of certain portions of the brain, or from organic or functional cerebral disturbance. It may also arise from abnormal conditions outside of the brain. The cases which are caused by a lack of development may be of congenital origin, or may be due to an arrested cerebral development produced by a number of causes. These causes are usually connected with some serious interference with the cerebral growth, such as a severe illness. The organic aphasia is like that produced by some such organic lesion of the brain as exists in cases of cerebral paralysis. It may also be connected with the condition of idiocy. The functional aphasia I have already described. It may be produced by many causes, among others the infectious diseases. A child may for a time during a severe illness, and after convalescence has been established, apparently be unable to use the words that it was accustomed to before the illness. I have in a number of cases, however, noticed that the child speaks better than it did before the illness.

Retarded speech may also be caused by such physical defects as disease of the ear resulting in deafness, and from such a physical malformation of the mouth, palate, or vocal cords as to render articulation impossible.

In this connection stammering may be spoken of as a cause of retarded speech.

When a child of this kind is brought to you to decide why it is unable to speak, you should carefully investigate the previous history. In this way you can eliminate organic disease of the brain by means of the absence of the usual symptoms of such disease, especially hemiplegia, and by ascertaining that the child has not had any disease sufficiently severe in its character to interfere with the development of the centres of speech. After determining that the child is not an idiot, you should make a physical examination of the ear and mouth. If, on examining the ear, you find that the child is deaf, you will at once have a good reason for his not being able to speak. Even where young children have learned to speak fairly well, if they later become deaf from a disease like scarlet fever they are very apt to become mute also. Where such lesion of the ear has occurred before the child has learned to speak, he almost invariably is found to be a deaf-mute, although there may be no defects in articulation or in his mental condition. It is seldom that any defect in the mouth or throat is found which nterferes with articulation, except in cases where very extensive lesions are present, such as cleft palate, and sometimes enlarged tonsils combined with a high-arched palate and a large adenoid growth. The tongue-tie which he parents usually consider to be the cause of the retarded speech is eldom present. Where no symptom of organic, functional, or developmental cerebral disease exists, where there is no physical deformity, and where the child hears well and seems bright and well developed in other ways, you can, as a rule, assure the parents that the speech is merely retarded and will probably develop later.

HEADACHES.—When pain in the head occurs in early life it is to be regarded more seriously than when it occurs at a later period, as it is more apt to indicate some grave central lesion. The various forms of organic headache which arise in children can be spoken of best as symptomatic of the various diseases in which they occur.

There also appears to be a type of headache which occurs in the later years of childhood irrespective of any disease and often unaccompanied by nausea. These headaches, as a rule, are not of serious import, and are usually classed under the term functional. They occur irregularly, and may be in any part of the head. They are often so severe that the child has to lie down. The intervals between the attacks are variable, and the length of the attacks varies from two or three hours to a day. Of these functional headaches the most frequent form in children is that due to anæmia.

Although in many cases headaches are caused by an improper regulation of the diet, yet there is evidently some other cause which we do not recognize in their production, as with exactly the same diet for many months a child will show no symptoms whatever of headache. In like manner, although we know that headaches in children may depend upon constipation, yet this class of cases occurs whether constipation is present or not.

Migraine also may exist in children, and is characterized by severe pain in the head, sometimes unilateral, sometimes bilateral, accompanied by nausea, dizziness, and generally vomiting. The attacks occur at irregular intervals, and usually last the greater part of a day. They may be brought on by apparently slight causes, such as over-fatigue or very mild indiscretions of diet, in those predisposed to them. These headaches are markedly hereditary.

Although all these forms of headache are ordinarily very intractable to cure, especially where no bad hygienic surroundings exist which might account for them, and where the child does not lead a sedentary life, yet, as a rule, the attacks have a tendency to lessen and disappear as the child grows older.

The treatment of these headaches is usually unsatisfactory, as the attacks seem to arise from some functional disturbance which, irrespective of any cause that we can ascertain, resists the best known hygienic and medicinal treatment. Strict directions should be given as to exercise and food. A change of air and scene is often a valuable adjunct to the treatment. In many cases the administration of fluid extract of ergot, as recommended by Dr. Russell Sturgis, has proved to be of benefit. I know of no better treatment for cases of this kind during the presence of an attack than absolute quiet in a darkened room and the use of bromide of potassium or bromide of sodium in sufficient doses to produce sleep, or at least to lessen the acute pain.

This boy (Case 356) is thirteen years old. He has usually been strong and well, active in his habits, and bright in his studies. When he was nine years old he had a light attack of scarlet fever. Up to that time he is said to have been healthy and never to have had any nervous disturbance during the dental period. Just before his attack of scarlet fever he had a severe headache. At first these headaches occurred only twice a year, but now he is attacked by them four times a year. All the headaches have about the same characteristics as the one from which he is now suffering. The pain is usually most intense in the top of his head, and extends to the front and back. The attacks generally last a week. In the second attack which he had, the pain did not last so many days as in the first and those which have occurred later, but he was left in a rather weak condition afterwards, so that he could not walk. There was no paralysis of the legs.

Yesterday he went to school as well as usual, but soon began to feel pain in his head, and had to return to his home. The headache has continued, and to-day, as you see, he cannot sit up, but has to lie down. He has no nausea, his appetite is good, and there are no special digestive disturbances, but he has a slightly coated tongue and a slightly raised temperature. The bowels are regular, and he complains of nothing but such severe pain in his head that he has to lie perfectly still. His diet has always been simple, and there are apparently no direct causes, such as the use of tobacco, to account for the attack. He has never shown any delirium, has always been perfectly conscious during the attacks, does not complain of any photophobia, and merely wishes to be let alone.

On examining him to-day you see that his temperature is 36.6° C. (98° F.), and his pulse 84, a little irregular, but not intermittent. On physical examination nothing of an organic nature is detected in the thorax or abdomen. The heart's action is somewhat irregular, and there is a slight murmur with the first sound at the apex, and an accentuated pulmonary second sound. He has never had rhachitis. He is rather anæmic, but of course is looking unusually pale to-day, as he is in the midst of one of these attacks.

In this case there may be some slight organic disease of the mitral valve, but, as the ild is well and strong between the attacks of headache, the murmur may be of functional igin. In either case the headaches can scarcely be accounted for by the cardiac disturb- ice, and can well be spoken of under the term functional.

In treating this case small doses of tincture of digitalis are indicated, on the supposition at some disturbance in the circulatory organs exists, evidence of which is given by the rdiac murmur.

We should, in examining a case of this kind, before speaking of the tack as functional, eliminate other possible causes. One of the most mmon causes in children, but which does not exist in this case, is pain used by a strain of the eyes. In all cases of headache in children the use of which is not evident, a careful examination of the eyes should be ade, even though there be no symptoms which point to the eyes them- lves.

As an illustration of a class of headaches the cause of which was rmerly obscure, I show you this little girl.

She (Case 357) is twelve years old. She has suffered during the past two years with nost continuous headache, so that she has had to be taken away from school.

A careful physical examination of this child made by me two years ago failed to detect ything abnormal, except that she was suffering probably from the results of eye-strain. ith the exception of the headache she has been well and strong, has had a good color, od appetite and digestion, and has simply been incapacitated from studying and reading account of the pain in her head.

The child was examined by an oculist and was made to wear glasses. No benefit sulted, and until within a few weeks her parents supposed that she could not be cured.

Suspecting, however, that the eyes were really the source of the trouble, I referred the ild to another oculist, who has made a change in the glasses, and the headaches have sappeared.

VERTIGO.—Vertigo at times occurs in children. It is a term applied a condition in which the individual or the objects around him appear to rolling about. It is called subjective vertigo when the patient himself ems to be turning, and objective vertigo when it is the surrounding objects at appear to move.

Vertigo has a variety of causes. It may be due to organic cerebral seases, such as tumors of the brain, especially of the cerebellum, and to seases of the ear and of the eye. It may also be due to circulatory dis- rbances, as in cardiac disease, and to gastric vertigo, as from improper od, also from tobacco and tea.

This boy (Case 358), thirteen years old, was referred to me by Professor Blake with the story that he had had a purulent otitis several years since, but that this had healed three ars ago, leaving a condition of adhesions and cicatrices with considerable impairment of aring, but with no trouble of the labyrinth nor any symptoms pointing thereto.

The child was always strong and well until he was seven years old, when he had the rulent otitis which Professsor Blake treated. Three years ago he began to have attacks dizziness accompanied by seeing white spots. He at times had nausea, but no feeling of inning round or falling. He has since had this feeling continuously, and lately it has ther increased in severity. He has had no other abnormal symptoms, except that he feels mewhat weak. He sleeps well, his appetite is fair, and his bowels are regular. He has good hygienic surroundings, does not smoke, and has never lived in a malarial district. He is a close student, and is not fond of active sports. He has never had any headache.

There is a strong probability that the vertigo in this case is caused by his drinking too much tea and by his sedentary life. I shall therefore simply have him stop drinking tea, and have told him to ride on horseback every day.

(Subsequent history.) Within a few weeks after the active exercise had been begun and the tea had been omitted from his diet, the boy ceased to have attacks of vertigo.

SENSITIVE SPINE.—Among the nervous symptoms of central origin is what is called sensitive spine, a case of which I have here to show you.

This boy (Case 359), thirteen years old, previous to one year ago was perfectly well, but since that time has complained of headache at times when studying, has lost his appetite, and has become constipated. He began to complain of his back at the same time that the other symptoms developed. The other symptoms have not been especially pronounced, but the pain in his back has grown progressively worse, and there is sensitiveness on pressure over the spine.

After I had first seen him and prescribed exercise in the open air and omission of school and of study, he improved for a time, and all the other symptoms disappeared, with the exception of the sensitiveness of the spine. Although at times this sensitiveness disappeared entirely, yet it has lately returned, and has been just as painful as in the beginning.

I therefore referred him yesterday to Dr. Lovett, who reports that there are no indications for mechanical treatment, that the spine is normal in every respect, and that Pott's disease can be positively eliminated. The tenderness of which he complains is one which we are accustomed to see in neurasthenic women. The probability is that, owing to his poor physical condition and his slight muscular development, his spine has had to depend on the ligaments to maintain it erect, that the sensitive condition and the pain are due to the strain which is brought to bear on them, and that this will disappear as his condition improves. We can therefore provisionally make the diagnosis of sensitive spine from debility, and I shall have him treated by massage, gymnastic exercises, and electricity. Some of these cases prove very intractable to treatment.

(Subsequent history.) Within a month after this treatment was carried out the boy recovered entirely.

TETANY.—Tetany is a term which is used to represent tonic intermittent muscular spasms without loss of consciousness. The condition is simply a symptom of nervous irritation, probably of central origin and not produced by organic lesions. This symptom is very apt to occur in cases of rhachitis, but it may occur in otherwise healthy children when they have various disturbances of the gastro-enteric tract. It is also met with in the course of many of the acute diseases, such as pneumonia.

The spasm usually affects the extremities and not the face, and is accompanied apparently by a certain amount of pain. The legs and arms are flexed and rigid, the hands and fingers tightly flexed, the thumbs usually beneath the fingers across the palms of the hands. In like manner the feet may assume various positions of flexion, such as that of talipes equinus or that of equino-varus. The length of time which the tetany lasts is very varied; it may be a few minutes or it may be hours or days.

The symptom in itself is not a serious one, the danger existing in t special disease which causes the tetany.

Tetany is to be distinguished from tetanus by the spasm of the masset occurring early in tetanus, and by its being absent or occurring late the child is attacked by tetany.

ebro-spinal meningitis is also to be differentiated from tetany by the ned temperature, the severity of the general symptoms, and the con- s and opisthotonos, which I have already described as characteristic disease, while these symptoms are not pronounced in connection with

treatment is to be directed to the special cause of the disease in the tetany occurs. Warm baths are indicated for the relief of the and bromide of potassium is the most efficacious drug in cases of nd.

VOR NOCTURNUS (Central).—The night-terrors of children cur from a variety of causes, and should not be considered as one but as a symptom of a number of diseases. Any nervous disturb- hether central or peripheral, may produce so profound an impression sensitive cortical cells of the brain that the child's sleep may be ed by a cortical irritation.

special form of pavor nocturnus which may be considered central has occurred in (Case 360), six years of age, who has been brought for advice to the clinic this . He has always been a delicate, thin, pale child, not caring much for open-air but inclined to remain in the house and to be read to or to have exciting stories told His appetite is poor. He is mentally bright and precocious. Otherwise he appears ll, and shows no signs of any organic disease. Last evening he was allowed to sit r later than usual, and a number of terrifying stories were told to him. He went to usual, but in about an hour waked up screaming. He was found sitting up in bed terrified. His eyes were staring at some invisible object, evidently a picture in his d not a reality; he was pointing at this imaginary source of his terror, and kept that it was a black dog. It was impossible to pacify him for about ten minutes, lid not recognize his mother during the attack. He then became more quiet; the k passed from his eyes; he recognized his mother, and soon lay down and went o sleep. The cause of this attack, which is typical of the central form of pavor us, was evidently undue excitement of the cells of the cortex in a bright, nervous fore going to sleep. The treatment of a case of this kind is to accustom the child to ercise in the open air, to prevent his reading anything but the most ordinary and ooks, and to have no stories whatever related to him.

LECTURE XXXVII.

IV.—FUNCTIONAL NERVOUS DISEASES.—(Concluded.)

(2) REFLEX.

Pavor Nocturnus (Peripheral).—Dental Reflex.—Reflex Nystagmus.—Reflex of the Ear.—Reflex of the Larynx.—Paroxysmal Gasping.—Reflex of the Lung.—Reflex Cough.—Reflex of the Heart.—Reflex of the Stomach.—Reflex of the Bladder.—Reflex of the Vagina.—Reflex of the Rectum.

PAVOR NOCTURNUS (Peripheral).—At the last lecture, gentlemen, I showed you a case of pavor nocturnus in which the symptoms were evidently of central origin. To-day I have to show you a little girl three years old who also has attacks of pavor nocturnus.

The child (Case 361), as you see, is robust-looking. She is said to be always well and strong; to have a good appetite; not to be nervous or excitable; to be fond of playing out of doors, and not to care to have stories told to her. Her mother also states that she is constipated, and that she has a tendency to overload her stomach. She has had disturbance of sleep for some time, and last night she had an unusually severe attack of pavor nocturnus. She had eaten a very heavy supper, and on going to bed she immediately fell asleep, but soon began to be restless, to throw herself about, to groan, and to grind her teeth. A little later she woke up screaming, and apparently had a certain amount of dyspnœa. She did not recognize her mother, but sat up in bed looking very much frightened and clutching at her throat. Her mother made her drink some warm water, which produced copious vomiting. She then became rational again, recognized her mother, and soon lay down and went to sleep. She has no recollection of these attacks on the following day.

This is evidently a case in which the irritation is of the terminal filaments of the pneumogastric nerve in the stomach, causing reflex symptoms of the nervous centres to such an extent that the child is terrified and feels as though she would stifle.

It is a case, therefore, of peripheral pavor nocturnus, and should be treated by moderating the diet and allowing the child to have only a light and digestible supper. You see that the two classes of cases are distinct and that their treatment is entirely different. You will also often meet with a mixture of both of these forms in which it is not possible to make a clear distinction between them.

I have collected a number of cases to show you which represent some other illustrative types of reflex nervous symptoms.

DENTAL REFLEXES.—The twitching which occurs in children at the time when a tooth is the apparent cause of a certain amount of discomfort and fever should be referred to here as a significant illustration of nervous phenomena from reflex causes. The cases are numerous, but scarcely of sufficient importance to report. In certain instances, however, convulsions of a reflex nature occur at this time and cease when the tooth has assumed its place above the margin of the gum. I have also met with some interesting cases of local œdema arising during the period of dental irritation.

One of these cases was a male infant (Case 362), fifteen months old, who some months eviously, while cutting one of the second molars, had an attack of œdema of the hands, iich was not accompanied by irritation or any other symptom, and which passed off after ew hours.

This same boy when the canine teeth were about to come through the gums was again acked by œdema of the face. This local œdema, as in the previous instance, disappeared ickly.

At times I have seen a local œdema attacking one eyelid, so that the eye uld not be closed.

Although we cannot say that the irritation from the teeth is necessarily e cause of these conditions, yet they so often arise during the dental riod, and not during other periods of childhood, or before the fourth or th month, that we can at least say that in individuals of an excessively rvous temperament the irritation which evidently arises in certain cases hen the teeth are developing may be sufficient to cause a nervous explosion, hich in this sense may be spoken of as of dental origin.

These are only a few instances of the reflex disturbances which occur ring the period of dentition, and I shall speak of the subject as a whole der the heading of difficult dentition (page 794).

NYSTAGMUS.—By nystagmus is meant a peculiar rhythmical oscil- tion of the eyeballs, usually from side to side.

It may be produced by many causes. It is sometimes dependent on ganic disease of the brain, and sometimes it arises from local diseases of e eye. In certain cases it is reflex from various peripheral stimuli.

Nystagmus of reflex origin is not a very uncommon symptom in young ildren. I have notes of two cases, brothers, who during the dental period owed this oscillation of the eyeballs with no other symptoms. Complete covery resulted when dentition was concluded.

This child (Case 363), three years old, has, as you see, nystagmus. She is rhachitic, d did not walk until three months ago. She shows no signs of organic disease, and will erefore probably recover from the nystagmus when the rhachitis is cured.

REFLEX OF THE EAR.—The reflex connection between the roots the teeth and the membrana tympani by means of the otic ganglion pro- ces the well-known reflex earache which occurs during the dental period. his phenomenon I shall speak of more fully later (page 795).

REFLEX OF THE LARYNX.—In certain cases reflex symptoms cur in the larynx. This condition is usually noted during infancy rather an in childhood. The affection has been called *laryngospasmus*, and, though it is more usual for it to occur in rhachitic children than in others, is not necessarily confined to rhachitis. It is not in my experience a very mmon disease, but when met with it is very characteristic.

The infant is suddenly attacked by a spasmodic contraction of the rynx. This condition may be precipitated by various causes, such as ight and excitement. I have also seen it produced by various peripheral ritations, such as those of the nose. At times the attack is so severe that the infant becomes unconscious and cyanotic. The attack lasts for only a few minutes, and on recovery the infant seems as well as ever. There does not seem to be an inflammatory condition connected with this disease, and apparently it is purely of a reflex nature. In some cases a crowing laryngeal sound will frequently precede and often succeed the more severe stage of the attack.

The prognosis in cases of laryngospasmus is, as a rule, favorable, although very weak infants may die in an attack.

As the infants are usually delicate and of a highly nervous organization, the treatment should be directed to improving their general health and to protecting them from nervous excitement until they have attained an age when their nervous system is less irritable and is in more stable equilibrium. During an attack the treatment is to endeavor to produce relaxation of the spasm by peripheral irritation elsewhere. This is usually done by showering the child on the chest and face with cold water and lightly slapping the back.

Among a number of cases of this kind which have come under my notice was this one:

A boy (Case 364), one year old, had always shown a nervous temperament and had had a number of convulsions when he was cutting his first teeth. With the exception of a light attack of epidemic influenza, he had been well and strong. Following the attack of epidemic influenza, in which the nasal symptoms were prominent, he was left with a very irritable naso-pharynx. He then began to have attacks characterized as follows:

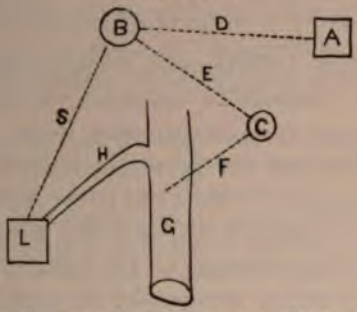
Whenever the nurse, while giving him a bath, attempted to cleanse his nose, no matter how gently, he would immediately gasp, have a catching sound in his breathing, become rigid, draw himself back sometimes almost to the position of opisthotonos, become unconscious and cyanotic, and then after a few seconds the spasm would pass away and he would seem perfectly well again. These attacks continued for some months without apparently harming him, and they then grew less frequent and passed away entirely.

Two other cases, in which the attacks were similar to the one which I have just described, were of infants in their second year. In these cases the attacks were usually brought on by excitement and when the children were thwarted in anything which they wished to do. In addition to the symptoms which I have just described, there was in the younger infant a crowing laryngeal sound just as the attack was taking place and for a few inspirations after it had ceased.

As additional examples of reflex phenomena of the larynx having their origin in the ear may be mentioned the hoarseness which sometimes accompanies the impaction of cerumen in the ears, and which disappears almost immediately after the removal of the mass. Professor Blake reports a case where a persistent laryngeal cough of several months' duration was immediately relieved by the removal of a bead from the external auditory canal. These cases, as well as one of reflex cough (Case 368) which I shall presently report to you, can be explained by means of this diagram (Diagram 9, page 749), which shows the reflex connection between the ear and the larynx.

The irritation of the sensitive fibres of the auriculo-pneumogastricus distributed in the meatus and to the membrana tympani is reflected along the motor fibres of the superior laryngeal nerve, exciting in the larynx the ct of coughing by causing contraction of the crico-thyroid muscle. Where he original irritant, either in the meatus or in the membrana tympani, by ts continued presence involves the vaso-motor fibres associated with the uricular nerve, they conduct their impression to the ganglion of the pneu-

DIAGRAM 9.



Reflex connection between the ear and the larynx. A, auditory canal, membrana tympani, and iddle ear; B, second ganglion of vagus; C, first cervical ganglion of sympathetic; D, auriculo-pneumo-astric nerve; E, sympathetic fasciculus connecting B and C; F, nervi molles, vaso-motor connection ith external carotid; G, external carotid; H, laryngeal artery; S, superior laryngeal nerve; L, larynx.

nogastric, and thence it is deflected through a sympathetic fasciculus pro-eeding from it to the first cervical ganglion. This again through the nervi nolles carries the impression to the external carotid artery, and by its ranches to the mucous membrane of the larynx, and as a result of reflected aso-dilator impressions we may have congestion of the vessels supplying he mucous membrane of the larynx, and perhaps effusion from these essels.

The detailed description and an illustrative diagram (Diagram 10) of he anatomical conditions underlying these reflex phenomena will be found n a later lecture (page 795).

PAROXYSMAL GASPING.—This boy (Case 365), eight years old, has always een delicate, and has evidently been ill fed and ill cared for. He is pale and thin, is of a ervous temperament, and has been overworked at school. Physical examination shows othing abnormal in either his thorax or his abdomen.

The attacks from which he now suffers have occurred for the past month, and are rowing shorter in their intervals and more severe in their character. The child, from being n a state of perfect quiet, suddenly becomes cyanotic, rolls his eyes up, stops breathing, asps, and looks as though he were about to die. The attack lasts about half a minute, nd he is then apparently as well as ever. Since he was brought to the clinic the intervals ave been about fifteen minutes. The seizure is apparently a reflex irritation of the iaphragm, and could be classed under hysteria. These cases respond quite readily to good are, well-regulated food, and relief from the duties of school.

A boy (Case 366), twelve years old, with the following history was brought to me for dvice:

He was of a nervous temperament. He was studious, and did not care to play with ther boys, preferring to remain in the house and read. His appetite was pretty good; e was pale and rather apathetic. An examination of the thorax and abdomen revealed othing abnormal.

Three weeks previous to my seeing him he began while sitting quietly in school to have aroxysmal attacks of gasping which he could not control. These attacks were of a much milder grade than in the case which I have just shown you, but were quite as frequent. They did not occur when he was playing out of doors or exercising, and he had never suffered from palpitation or dyspnœa.

Treatment with various drugs, such as arsenic, nux vomica, and iron, given at different times and separately, had no effect upon the paroxysms. Taking the boy out of school, preventing him from studying or reading much, and making him go to the gymnasium and ride a bicycle, lessened the attacks in a few months, and he recovered entirely in about eight months.

REFLEX OF THE LUNG.—In young infants pulmonary attacks closely simulating the symptoms of asthma occur from gastric irritation of the terminal filaments of the pneumogastric nerve. They are evidently reflex in their character, and are promptly relieved by treatment of the stomach. They are spoken of under the term asthma dyspepticum. In cases of this kind it is usually found that the peripheral irritation either arises from the too high percentages of the solid constituents of the milk which is given to the infant, or is caused by the total amount of milk given being too great for the infant's gastric capacity.

The first symptoms noticed in these cases are the pallor of the infant's face, and a slight cyanosis around the mouth. The respirations then become quickened, and the infant is evidently in great distress. It becomes cyanotic, breathes very rapidly, and often looks as though it were about to die. On examining the chest the lung is found to be resonant, and there is nothing abnormal on auscultation except roughened respiration and a few sonorous râles.

An emetic will quickly relieve this condition, which disappears as soon as the stomach is emptied. The attacks are sudden and often recur. After the attack has passed off, the abnormal sounds heard in the lung are found to have disappeared completely, and the infant seems perfectly well again.

Another class of reflex pulmonary symptoms which has at times come under my notice consists of cases in which from some peripheral irritation elsewhere marked pulmonary symptoms simulating pneumonia arise.

This little girl (Case 367), six years old, is an interesting case of this kind. The first attack occurred at a time when she was having an exacerbation of an attack of subacute purulent otitis media. This happened one year ago, and she is brought to the clinic to-day apparently suffering from an attack of the same kind, so that I shall probably be able to illustrate its reflex nature.

As in the first attack, she has a heightened temperature, 40° C. (104° F.), and rapid respirations (60 in a minute). The alæ nasi are moving slightly, and she shows a certain amount of orthopnœa. Her face, as you see, is flushed, and she has a short, dry cough. She has a discharge of pus from both ears. On looking at this child you would naturally say that she had pneumonia. Evidence of this is given by the temperature, the respirations, the alæ nasi, the cough, and the orthopnœa. On examining the lungs, heart, and throat, they are found to be normal. The pulmonary symptoms are evidently reflex in their nature, as it is believed that in these cases the reflex symptoms are usually produced by the occlusion of the Eustachian tubes.

Now watch the effect of inflating the Eustachian tubes with the air-douche. You see that within a few minutes after I have inflated the Eustachian tubes her breathing has become normal in rate, 24 to 28 in a minute, the alæ nasi have ceased to move, the cough has disappeared, and the child can now lie down with comfort. The temperature will soon in to fall, and in place of the picture of a pneumonia which you saw a few minutes ago see she is assuming the natural condition of a child with slight fever and a heightened perature caused by inflammation of the middle ear.

REFLEX COUGH.—The nervous connection between the ear and the ynx gives rise at times when there is disease of the former, such as an tis media, to a persistent cough which is evidently reflex, and which is ieved only by treatment of the ear. A very interesting case of this kind s for some time under my care in conjunction with Professor C. J. Blake.

A little girl (Case 368), four years old, had an attack of measles which was complicated an otitis media. She recovered entirely from the measles, and seemed perfectly well, ept that the perforation of the membrana tympani had not entirely healed. Somewhat r the cough began. Nothing was found to account for this symptom in the throat, lung, arynx, except a slightly reddened appearance of the latter from coughing. The cough intractable to all local treatment until the ear, which had been in the process of heal- again showed signs of increased inflammation. Whenever the ear was discharging, cough ceased entirely. When Dr. Blake treated the ear and the discharge grew less, cough began again, and there was an evident reflex connection between the larynx and ear.

These reflex phenomena continued for some months, the child always coughing when ear got better and ceasing to cough when the ear got worse. Finally, on the child's g taken to Switzerland and having an entire change of air, its general health was much roved and the reflex cough passed off. There has been no recurrence of this condition he following ten years.

Where there is an irritation in the naso-pharynx a reflex cough often urs, and is best treated by local applications to the pharynx and naso- arynx. It is important for the physician to recognize this class of ghs, as he might otherwise be very unsuccessful in treating these cases. ny children are treated with drugs for a cough which is usually ascribed bronchitis, where no physical signs of irritation can be found in the g, larynx, or throat, and where the irritation is in the nose or the naso- arynx. In place of the many drugs usually given in these cases, a simple ay in the nose is indicated.

REFLEX OF THE HEART.—I occasionally meet with cases of ex- me palpitation in children where nothing organic can be detected, and ere no cause, such as tea-drinking, is discoverable. The children are of ighly neurotic temperament, and are usually much influenced by exciting roundings in their homes.

As an illustration of this class of cases I will show you this boy (Case 369), ten years , who is subject to fits of great excitement brought on by the most trivial causes, such as paring to go to school or to take a journey. For some hours before the proper time for ting comes he is apt to grow more and more agitated, thinking that it must be time to t. He will then often be seized with violent palpitation lasting for several hours and ing him to lie perfectly still on his back. At these times his skin will be cool and pale, his pulse weak and irregular. Nothing abnormal has ever been detected on an exam- tion of the heart or any other organ.

(Subsequent history.) The attacks in this case lasted until he was twelve years old, and e never occurred since.

REFLEX OF THE STOMACH.—There are a number of reflex conditions connected with the stomach arising from different causes but represented by the same symptom, vomiting. Instances of this condition are those cases of vomiting which arise from irritation of the larynx and pharynx and which are cured by local applications made to these parts. There is another reflex gastric condition in which the vomiting is apparently caused by shock, probably affecting the abdominal sympathetic ganglia.

These cases can best be spoken of in detail when I describe the various affections of the stomach. I shall therefore merely refer to them here as instances of reflex functional disturbance.

REFLEX OF THE BLADDER.—Reflex spasm of the bladder occurs very frequently in young children. I shall consider it under the head of incontinence of urine when speaking of diseases of the bladder.

REFLEX OF THE VAGINA.—There is almost always a direct cause to be found for the reflex nervous symptoms which arise from vaginal irritation. One of the most common causes is the ascaris vermicularis, which at times gives rise to extreme and severe symptoms when it has migrated from the rectum. In addition to the local irritation, which causes the child great uneasiness, so that it cannot sit still and is continually moving its legs about, its temperament may be much affected. A child with this trouble is apt to be very fretful, to have violent outbursts of temper, to lose its appetite, and to grow thin. A case of this kind has lately come under my notice.

A little girl (Case 370), five years old, had the most extreme vaginal irritation. Whe I saw her she had been affected for several months and was in a very weak condition. times the irritation seemed to be more than she could bear, so that she would lose all con of herself, would throw herself on the floor, and would have violent spasmodic contracti of the legs. Her sleep was much interfered with, and her whole appearance was that child suffering from some serious disease. An examination showed that the ascaris micularis was the cause of the vaginal irritation, and after a few days' treatment directed expelling this parasite the child ceased to have any irritation and subsequently recove entirely.

REFLEX OF THE RECTUM.—In certain cases reflex symptoms a most exaggerated type are localized in the rectum. An instance of t phenomenon is this little girl, who, you will remember, was brought to t clinic several weeks ago.

She (Case 371) is four years old. She has always been remarkably strong and robu and has never had any especial local trouble with the bladder or the rectum. She is, h ever, of an excessively nervous temperament, and is surrounded by exciting influences her home.

A few months ago she began to have spasmodic contractions of the sphincter When she attempted to have a movement of the bowels it frightened her, and she wo clutch at any piece of furniture which happened to be near her and try not to have movement. She would scream and cry out as if she were in much pain.

An examination under ether showed nothing abnormal in the rectum or sphincter, as from pressure or from lesions, and the condition was apparently that of spasm simply

For the last two weeks she has been treated by the daily dilatation of the sphincter with bougies, the size gradually being increased. This has been followed by marked improvement, and her mother reports to-day that the trouble has passed away.

(Subsequent history.) The rectal spasm did not return in this case, but the child began to have incontinence of urine, from which she is still suffering two years later.

Another case which came under my care and which was a form of reflex connected with the anus was a little girl (Case 372), eight years old.

This child for a whole year was affected by intense irritation in the region of the anus, which prevented her from sitting down for any length of time and kept her in a continual state of irritability. Nothing could be detected during this period which caused these symptoms. No trace of intestinal parasites could be found, and nothing abnormal, either at the anal orifice or in connection with the bowels, was seen, the skin around the anus being in a perfectly normal condition.

The child was treated with bromide of potassium for several weeks, and recovered entirely.

What I have said concerning the reflex phenomena of infancy and early childhood must not be supposed to be a complete enumeration of these affections. Thus, various involuntary movements of the head in infants sometimes occur, such as *spasmus nutans* (antero-posterior movements) and *gyrospasm* (rotary movements).

LECTURE XXXVIII.

CONVULSIONS.—TREMOR.

CONVULSIONS.—Attacks of motor disturbance represented by continuous rigidity or contractions of one or more groups of muscles, lasting for a variable time and accompanied usually by unconsciousness, are designated convulsions. The term convulsion is applied to a symptom, and not to a disease.

Convulsions may be divided, as to their type, into (1) *clonic* and (2) *tonic;* as to their form, into (1) *general* and (2) *partial;* and as to the seat of irritation which causes them, into (1) *central* and (2) *peripheral.*

The *clonic* convulsion is an active spasmodic contraction of the muscles followed by immediate relaxation. The convulsions of epilepsy are illustrative of this type.

The *tonic* convulsion is a more or less continued spasmodic rigidity of the muscles. This type is seen in tetanus neonatorum.

The convulsive movements may affect the entire body and limbs, including the face, or they may affect only certain groups of muscles. Thus, they may be localized, as of one limb. They may be unilateral or bilateral.

The seat of irritation which produces the convulsion is very varied. Thus, it may be a lesion of the central nervous system or of the peripheral nerves; in the former case the convulsions are spoken of as *central,* in the latter they are termed *reflex.* Convulsions are much more apt to occur in infancy than in later childhood and in adult life, probably because the power of inhibition is not developed in the former. Not only, therefore, do we meet with convulsive attacks more frequently the younger the individual, but, as a rule, we are led to look upon these convulsive attacks as of much less import than in the older subject. The reason for this is that the causes of reflex convulsions in infancy are innumerable, and, as a rule, they do not result seriously, while in older children and in adults convulsions are almost always representative of some serious central lesion. Convulsions are in fact so common in infancy that they have been compared to the chill which occurs in the initial stage of many diseases arising in adults. It is a fact that the various acute diseases accompanied by high temperature, such as pneumonia and the exanthemata, are very commonly preceded by a convulsion, and that a chill is rare under these conditions in infancy. We must, however, not be misled by the frequency and comparatively benign character of convulsions in infancy and by the rule that they are not fatal. The convulsions of infancy may represent just as serious conditions as they do in later life, and we must always look upon them as a grave symptom until we can be sure, by eliminating serious organic lesions as a cause, that we are dealing

h one of the common and mild forms of this phenomenon. We must
nember that the convulsion does not in itself show us whether it is the
ılt of serious or of benign disease. The convulsions which occur from
ne organic lesion, such as cerebro-spinal meningitis, may differ in no way
m those which arise from some simple cause, such as indigestible food.
is therefore well to speak of convulsions apart from the diseases in which
y occur, and which I have already described.

We are frequently called to see an infant in convulsions where the con-
lsion is the first and only manifestation of the disease which is presented
us. The infant after a few signs of uneasiness suddenly becomes rigid
a second or two, makes a sound as though choking, the eyes turn
ward and become fixed, there may be strabismus, the skin becomes
mewhat cyanotic, and then the convulsive movements begin. The eye-
ls open and shut; the face and usually the head are drawn to one side;
e fingers are clinched; the arms move up and down, as do also the legs.
he back may at times be somewhat arched and the head somewhat re-
acted. The infant is apt to foam at the mouth to a greater or less extent;
is perfectly unconscious, and the breathing soon becomes stertorous.
hese symptoms after lasting for two or three minutes are followed by
mplete relaxation, an apparent state of coma, and sleep. The child on
aking may be bright and well, or the convulsion may recur and continue
r a much longer time, as in one of my cases, where an infant had fifty-
vo convulsions in twenty-four hours. There may be involuntary discharges
f urine and of fæces.

I have had an infant brought to show you to-day who illustrates very
early the fact that numerous convulsions do not necessarily lead to a fatal
sue.

This infant (Case 373), six months old, is well developed, healthy, mentally bright,
d has not had any convulsions since it was a month old. During the first two weeks of
life it had convulsions almost continuously.

This table (Table 105) gives the hours and intervals of the convulsions from 9 A.M. one
y till 9 A.M. the next day. The attacks, as a rule, lasted only a few minutes.

TABLE 105.

(Forty-one convulsions in twenty-four hours.)

Time of Convulsions.

9 A.M.	12 Midnight.	2.42 A.M.	6.22 A.M.
11.35 "	12.35 A.M.	3.18 "	6.32 "
3.10 P.M.	12.40 "	3.25 "	7.10 "
3.50 "	12.50 "	3.42 "	7.40 "
4 "	1 Noon.	4.40 "	7.52 "
4.07 "	1.25 P.M.	5.08 "	8.07 "
4.28 "	2 "	5.20 "	8.17 "
4.40 "	2.12 "	5.30 "	8.25 "
5.55 "	2.25 "	5.45 "	8.35 "
6.13 "	2.38 "	5.55 "	9.02 "
11.50 "			

sease which most commonly gives rise to convulsions of the reflex
rhachitis. Rhachitic children seem to be predisposed to spasmodic
of all kinds, and a general clonic convulsion in children with rha-
orresponds to the spasmodic contractions in the larynx which occur in
is, and which I have already spoken of as laryngospasmus.

is probable that there is no especial lesion in connection with the
is which necessarily gives rise to convulsions, but that all the tissues
disease are especially sensitive to causes which may produce reflex
ons. The most common cause of reflex convulsions in infants is
er food. Convulsions from this cause arise not only where mani-
ndigestible articles are given to young children, but even in infants
e being fed from the breast. In the early days and weeks of life,
the breast has acquired its normal functions connected with elabo-
a milk in which the solids are in proper proportion to each other and
water, it is not uncommon for the infant to have convulsions produced
listurbance of the mammary function. In cases of this kind it is
found that the percentage of the proteids is high, and that the con-
is will continue until this high percentage has been lessened, if the
is allowed to continue to nurse. Whether the teeth of themselves
their development are a source of sufficient irritation to produce
sions has been questioned by many observers. It is, however, evident
ring the different periods of dentition reflex convulsions are more apt
r than when a tooth is not disturbing the infant's nervous system.
ition to the convulsions arising from improper food in the stomach
the dental period, foreign bodies in the intestine, whether in the
f food or in that of intestinal parasites, may cause reflex convulsions.
n bodies in the nose and in the ear have been known to produce
sions, as also has an inflamed tonsil in the initial stage of a follicular
tis. Hot baths are so often given to infants when they are in con-
s that they should be spoken of as a source of convulsions, for
ave been known to produce this result when care has not been taken
the temperature of the bath before the infant is put into it.

OGNOSIS.—The prognosis of infantile convulsions must, as you will
understand, vary much in connection with the especial cause. On
ing from the attack the infant may show signs of some serious
lesion, such as paralysis, or may be left apparently perfectly well.
le convulsion followed by perfect recovery is of slight consequence,
ere the convulsive attacks recur frequently and last longer than in
acks which I have just mentioned, the prognosis becomes graver.
though no central lesion be present, continued convulsions may
shock to the infant's vitality finally cause death from exhaustion,
th may occur from spasm of the glottis. We must, therefore, no
what the cause or what the apparent result of a convulsion may
ays look upon it as a grave symptom and endeavor to prevent its
ence.

TREATMENT.—When you are summoned to treat an infant who is in convulsions, you should first see that the bath, in which you usually find that it has been immersed, is not too hot, and should order the infant to be taken out of the bath before it becomes conscious, or it may be so frightened as to excite again the reflex spasm. You should quickly examine the thorax for pulmonary and cardiac lesions, and should make inquiries as to the history of the case, especially as to the infant's diet. The temperature should be taken, and you should notice whether the fontanelle is bulging or depressed.

Having obtained this information, you can eliminate quite a number of causes for the attack, such as the onset of one of the exanthemata if the temperature is normal, and reflex convulsions from food or foreign bodies in the nose or in the ear. You can soon determine whether the convulsions arise from exhaustion, so that you can proceed at once to order stimulants, if necessary, and, if the convulsions continue, to make use of the general treatment which is indicated for all forms of convulsions.

You should be prepared to act promptly, and for this reason you should acquire the knowledge which will enable you readily to classify the attack under its proper head and thus treat it intelligently. The parents are so terrified when a convulsion attacks an infant that it is necessary for the physician to be able to inform them as soon as possible whether or not the convulsion is injurious. In order to aid you in differentiating the various causes of convulsions from each other I have prepared this table (Table 106).

TABLE 106.

Infantile Convulsions.

Central.	Peripheral (Reflex).
I. Diseases with high temperature. (Insolation, meningitis, the exanthemata, pneumonia, and others.)	Rhachitis.
II. Diseases accompanied by vascular stasis. (Pertussis, cardiac disease, tumors, hydrocephalus.)	Food.
III. Diseases characterized by anæmia and exhaustion. (Loss of blood, diarrhœa.)	Intestinal parasites.
IV. Various toxic causes, such as drugs, or uræmia. (Belladonna, nephritis.)	Dental irritation.
V. Organic central lesions. (Cerebral paralysis, or any other lesion of the brain.)	Foreign bodies in the ear and nose.
VI. Presumably organic disturbance of the brain. (Epilepsy.)	Hot baths.
	Mental disturbance, such as fright, and numerous other causes.

The treatment of infantile convulsions should be directed to the especial cause of the convulsion. In general, however, as often when the convulsion is present it is impossible to determine whether it is of central or of periphral origin, it becomes necessary to endeavor to control the attack at once. or this purpose in all forms of convulsions the inhalation of ether in small mounts, and the emptying of the bowels by means of enemata, are indi- ated. Where the convulsions are of a very severe type, continuing with erhaps intermissions of only a few minutes, and the infant's life is evidently n danger from the continuous shocks which are taking place in its nervous ystem, a rectal injection of 0.6 gramme (10 grains) of bromide of potassium nd 0.3 gramme (5 grains) of hydrate of chloral in 30 c.c. (1 ounce) of warm ater, repeated if necessary every hour for three or four doses, is indicated. f the convulsions still continue and a fatal issue is anticipated, a subcu- aneous injection of sulphate of morphine, beginning with 0.001 ($\frac{1}{60}$ grain), hould be tried.

In most cases of infantile convulsions, of whatever form, the warm bath t a temperature of not over 37.7° C. (100° F.) can be used, for, although it not in any sense curative, it tends to quiet the nervous excitability and o lessen the muscular strain produced by the continuous spasmodic muscular ontractions. The class of cases in which this is contra-indicated are those hich are caused by a loss of blood, an anæmic condition, diarrhœa, and ardiac disease, and those in which venous stasis exists with a lowered temperature. In these cases stimulants are indicated.

In those cases which are symptomatic of the diseases which I have already spoken of as accompanied by high temperature, the application of cold to the head and the administration of the bromides are indicated.

The treatment of convulsions caused by the other diseases which I have numerated is simply symptomatic while the convulsions continue, and later he proper care of these diseases. All the reflex convulsions from various auses are treated in like manner symptomatically and by the removal of e especial cause.

I have already shown you an infant (Case 342) in clonic convulsions, d described to you the characteristics of the attack, while speaking of ilepsy (page 727).

I have here a few cases which may be of interest in this connection for u to see.

This little girl (Case 374) is six and one-half years old. She was healthy at birth, and s never had any disease. For the last three years she has from time to time had a con- lsion, clonic in type. When in the convulsions she does not bite her tongue. The first nvulsion occurred when she was three years old; the next when she was four years old; e next when she was four and one-half years old; and the last one when she was five ars old.

As all these convulsions have apparently been produced by the same cause, it will nly be necessary to describe them in a general way. They have been characterized by metimes continuing much longer than is usual in infantile convulsions, one of them aving lasted for one hour and a half, during which time the hands were clinched, the eyes were rolled up, and the entire body and limbs were convulsed. Previous to each attack the child for a number of days has had indefinite symptoms which she could not describe accurately, connected with the abdomen and accompanied by a feeling of weakness and slight muscular twitching.

At the time of the earlier attacks her mother found that these symptoms could be dissipated and apparently a convulsion prevented from occurring by giving her a dose of castor oil about once a week. After the third convulsion she passed a lumbricoid worm. From that time whenever she showed the premonitory symptoms of a convulsion she was treated with santonin followed by a cathartic, a lumbricoid worm was each time passed, and the symptoms disappeared. When she was five years old she was thoroughly treated for these lumbricoid worms with santonin, and after a large ascaris had been passed the nervous symptoms ceased entirely. The child has now not had a convulsion for over a year. There has been no reappearance of the worms.

This child represents the class of cases which I have described when speaking of reflex convulsions, the cause of the peripheral irritation evidently being an intestinal parasite.

I have here an infant (Case 375), thirteen months old, whose nervous system has always been in so irritable a condition that the slightest cause was sufficient to produce a convulsion.

When he was eight months old he had an attack of pertussis, and during the course of the disease he had a number of convulsions. At one time when the pertussis was at its height he had from fifteen to sixteen convulsions within thirty -six hours, each of them lasting from five to ten minutes.

When the first teeth began to press upon the gums he occasionally had a convulsion. In addition to the general muscular spasms he had nystagmus of the right eye. For the last two or three months he has had no convulsions, and the nystagmus is much less noticeable.

This boy (Case 376), four years old, has from time to time had convulsions, which, so far as I can ascertain, are simply reflex, and are not connected with epilepsy or with any organic disease.

When he was six months old he had a number of convulsions while cutting his incisors. When he was two years old he had an attack of epidemic influenza, which was ushered in by a convulsion; and the same phenomenon occurred when he had an attack of catarrhal laryngitis some months later.

This little girl (Case 377), four years old, is, as you see, a bright child, and is in fairly good health. She is apparently recovering from convulsive attacks which occurred with great frequency in her second and third years, and which were apparently produced by epilepsy. At one time she had fifty-four convulsions in forty-eight hours.

She has been treated simply by carefully regulating her diet and with bromide of potassium.

The prognosis in this case is not very favorable, as she is probably an epileptic, and the convulsions are liable to return at any time, especially as puberty is approached.

This next child (Case 378), a girl, four years old, is a case of considerable interest, as presenting an instance of some slight organic lesion occurring when she was two and one-half years old, accompanied by a convulsion. The convulsion was of the general clonic type, lasted for a few minutes, and was accompanied by a temperature of 39.7° C. (103.5° F.) and a pulse of 140. On recovering from the convulsion she was found to have a slight hemiplegia of the left side, which lasted for only a few hours. She then recovered entirely, and has since had no convulsions, but she has never developed either mentally or physically in accordance with what would be expected in a child of her age, so that she has to be watched over by her nurse as carefully as though she were three years old, as she is liable to fall and does not go up-stairs easily.

This child (Case 379), three and a half years old, has always been well and strong. She was suddenly attacked about a month ago by a chill, and was found to have a high temperature and a quick pulse. A few hours later, the temperature having risen to 40° C. (106° F.) and the pulse to 170, she suddenly had a general clonic convulsion. After the convulsion had ceased she remained unconscious, and some hours later had another convulsion. She was placed in a warm bath, and after the temperature had been reduced two or three degrees the convulsive movements ceased. A little later a general papular efflorescence of measles appeared on her face and neck, afterwards spreading to the body and limbs. She e perfectly conscious, and did not have any other severe symptoms during her attack asles, nor any return of the convulsions.

er case is an instance of convulsions produced by a high temperature in the initial ›f one of the exanthemata.

'he next three infants whom I have had brought here to show you iteresting examples of the necessity of regulating the solid constituents e milk which is given to young infants.

he first case (Case 380) is that of a little girl, four months old. Her mother, who was and well and apparently had plenty of good breast-milk, nursed her at birth. When is three months old she began to have convulsions, which occurred almost every hour. :ting that the proportion of solids in the breast-milk was too high for the infant to them, and that they were producing a peripheral irritation which was the cause of flex convulsions, I had an analysis of the milk made, and found that the proteids d a percentage of from 4 to 5. The infant was then fed with a carefully modified n which the percentage of the proteids was made 1. Within a few hours the conns ceased, and they have never returned. As you see, the infant is perfectly well and ng to-day.

have in instances of this kind so regulated the percentage of proteids e mother's milk by the means which I have described to you in a pre- lecture (Lecture VII., page 188) that an infant who before this fication of the mother's milk had been made was having continued ulsions ceased entirely to have them, and was nursed successfully for months.

'his next infant (Case 381), a little girl, six weeks old, began to have convulsions when as four weeks old. The convulsions occurred every twenty minutes for twenty-four , and sometimes as often as every fifteen minutes. They lasted for only a few seconds. nfant was being fed on the milk of a Jersey cow. She was then fed on a carefully led milk with a moderate percentage of fat and proteids, and the convulsions did not .

'he third case (Case 382) is a boy, six weeks old. He was healthy and strong at birth, 'as nursed by his mother for three weeks. During this time he gained in weight and ed the milk perfectly. The mother, however, was unable to continue nursing him the third week, and it was decided to feed the infant on modified milk. The prescrip- or this modified milk sent to the laboratory by a physician was as follows:

PRESCRIPTION 72.

Fat	5.50
Sugar	7.00
Proteids	3.50

oon after this milk was given to the infant it began to have convulsions, which ued for twenty-four hours, at intervals of two or three hours, until the milk was d. Another modification of the milk was then substituted for the first, and the infant to have convulsions and has since digested the milk perfectly. The percentages in st prescription were as follows:

PRESCRIPTION 73.

Fat	3.50
Sugar	6.50
Proteids	1.50

LECTURE XXXIX.

THE MYOPATHIES.

Progressive Muscular Atrophy.—Pseudo-Hypertrophic Muscular Paralysis.—Myotonia Congenita (Thomsen's Disease).

PROGRESSIVE MUSCULAR ATROPHY.—Progressive muscular atrophy is a name used to denote certain conditions which were originally supposed to be due to a disease of the spinal cord. Later, however, it was found that two forms of lesions produce this atrophic condition of the muscles. One of these, the neuropathic form, is an affection of the spinal cord, and is designated the Aran-Duchenne or thenar type. The other form is found to be a primary disease of the muscles, and is classed as one of the myopathies.

Neuropathic Progressive Muscular Atrophy.—The neuropathic atrophies are so rare in infancy and early childhood that little need be said concerning them. The neuropathic progressive muscular atrophy is caused by a chronic degeneration of the ganglion-cells of the anterior cornua, and this is the form which I have just alluded to as the Aran-Duchenne type. In this form the atrophy almost always begins in certain muscles of the hand, especially those of the ball of the thumb, and it is for this reason called thenar. It is rarely seen before the twentieth year, and is not hereditary. Hypertrophy of the muscles does not occur.

Myopathic Progressive Muscular Atrophy.—The myopathic atrophies show a marked hereditary tendency. They have been diyided by various authors according to the different portions of the body in which they begin. The disease in each case is essentially the same, and this division seems to be unnecessary and misleading, because, although the affection may begin in any part of the body or extremities, yet, as a rule, it may be said that the primary myopathies begin in the muscles of the shoulder, face, or back. In all these cases the atrophy usually begins before the twentieth year.

Where the muscles of the face and scapulo-humeral groups are involved early, it is called the *facio-scapulo-humeral type* of Landouzy and Déjerine.

Where the atrophy begins in the gluteal muscles and those of the thigh, arm, and shoulder, it is called the *juvenile type* of Erb.

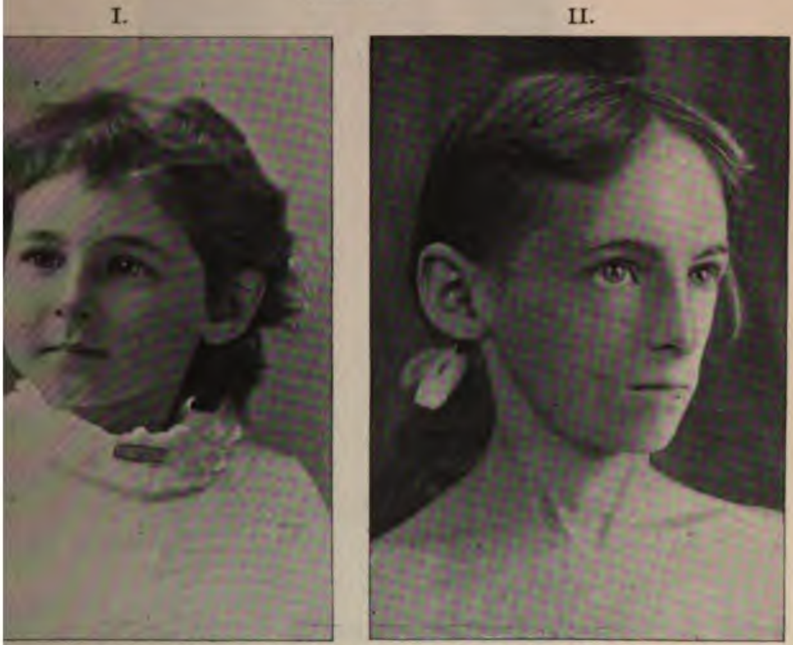
Where the atrophy first affects the muscles of the legs, it is called the *peroneal type*, and the affection shows itself in the peripheral muscles of the lower extremities, such as the extensors of the great toe, and afterwards in the common extensor of the toe and in the peroneal group. There is, however, regarding this latter type a doubt as to whether it is a primary myopathy.

ıll these forms the muscles react to both the faradic and the galvanic , and there is no reaction of degeneration.

ɪɢɴᴏsɪs.—The diagnosis of myopathic progressive muscular atrophy first be made from the neuropathic form. The former is hereditary; er is not. In the former the atrophy usually begins in the muscles houlder, face, and back, while in the latter it is exceedingly rare for gin elsewhere than in the muscles of the hand. Hypertrophy of muscles and the beginning of the atrophy early in life, usually he tenth year, are characteristic of the myopathic variety, in contraion to the late development and the absence of hypertrophy in the ıthic form.

muscular atrophy which accompanies certain cases of chronic multiritis may be mistaken for a myopathic affection, and must therefore differentiated. At times the resemblance of the two diseases is quite , but it does not last for a sufficiently long time to leave the diagnosis ı doubt. You must remember that chronic multiple neuritis is never ıry, that the paralysis which accompanies it is out of proportion to

Cᴀsᴇ 383.



facio-scapulo-humeral type of primary myopathic atrophy. I. Before the disease began. II. After the disease was well advanced.

phy, and that there may also be distinct symptoms of ataxia, all of ymptoms are unusual in the primary myopathic atrophy.

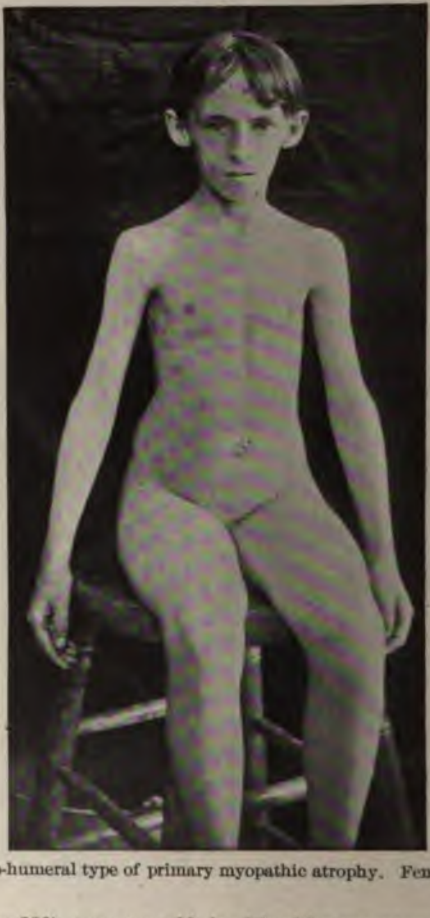
ɢɴᴏsɪs ᴀɴᴅ Tʀᴇᴀᴛᴍᴇɴᴛ.—The prognosis is very unfavorable, and no known treatment which benefits the disease. The patient should ed under the most favorable surroundings for his general health.

Precautions should always be taken to prevent the contractures which nec sarily occur in the later stages of the disease from producing awkwa positions of the body and limbs.

The *facio-scapulo-humeral type* of primary myopathic atrophy is exceedingly rare that I am fortunate in having a case here to-day to sho you. It has been carefully attended in my wards by my colleague D Bullard.

CASE 383.

III.



The facio-scapulo-humeral type of primary myopathic atrophy. Female, 10 years old.

This little girl (Case 383), ten years old, is of healthy parentage. There are four oth children in the family, who show no signs of disease. This child, although she has ha various diseases, such as varicella, measles, and pertussis, has on the whole been well a strong, and until three years ago was unusually well developed. Here is a picture (I., pa 765) of her taken at that time, just before she was attacked with the disease from which s is now suffering.

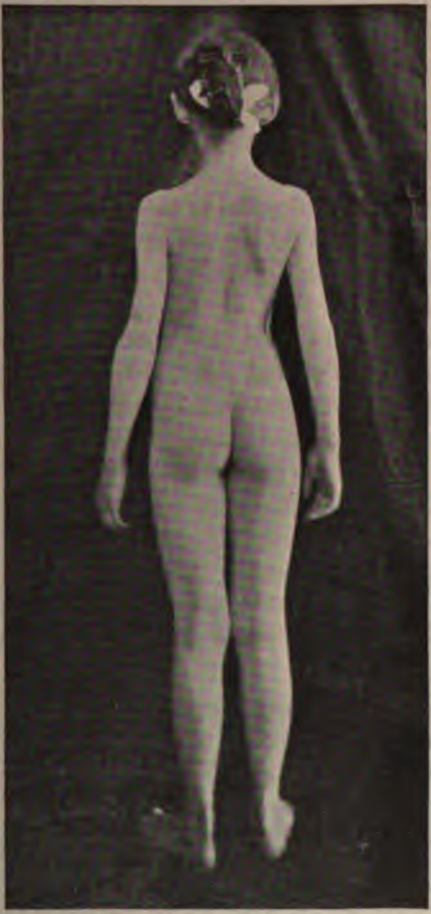
You see that the face is unusually full and plump, and at that time there was eviden no sign of muscular disturbance.

If you will now look at the child's face as she stands before you (II., page 755) a compare its emaciated old look with the young, well-nourished look shown in the pict (I.), you will at once understand that she is affected by a disease of serious import.

e years ago she had an attack of epidemic influenza. Since then she has been weight and strength. She has complained of pain in the abdomen, not localized, continuous for a few hours, and then remitting for an hour or so. This disturb- last for two or three days, and during this time she does not care to do anything, own, usually on her back. She occasionally vomits; there is nothing character- it the vomiting, but it relieves the pain of the acute attacks. She may have at ght nausea; she seldom has headache; the bowels move regularly; she has no it a slight nasal catarrh is usually present, as she catches cold very easily. She nuresis, and during the acute attacks of pain she is apt to have attacks of pavor s. She is very nervous, and cries easily.

CASE 383.

IV.



The facio-scapulo-humeral type of primary myopathic atrophy.

xamining the child in front as she sits on a stool (III., page 766) you see that the nusually well developed, in marked contrast to the atrophy of the face, body, and er respiration is free and equal on both sides. The face and neck are extremely the muscles are atrophied. The muscles of the upper extremities and chest are small, but firm and of fair strength, while those of the abdomen and legs are well d and firm. The skin is dry, the eyes are bright, and the reaction of the pupils is On physical examination nothing else abnormal is found, with the exception that the action of the heart is rather rapid. There is slight ankle-clonus, and the patellar reflexes are slightly increased. The glands of the neck, axillæ, and groins are very slightly enlarged. The tongue is normal and can be protruded steadily. The examination of the urine shows it to be normal, with the exception of a slight trace of albumin.

The only other case of this disease which has to my knowledge been reported in this country is one by Osler.

On examining the child's muscles more closely you will see (III.) that the most marked atrophy is situated above the diaphragm, while the abdomen and legs are remarkably well developed.

The muscles affected are those which I have already enumerated in the general description of the disease.

On turning the child so as to look at her back (IV., page 767) and making her stand up, you will notice the striking difference between the arms and upper part of the body and the legs and lower part.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.—The form of primary muscular atrophy which I shall next speak of is what is called pseudo-hypertrophic muscular paralysis. This disease is characterized by a diminution of power in certain muscles, accompanied by an abnormal increase in their size and a diminution in the size of other groups of muscles. Although an apparent increase in the size of the muscles takes place, yet the enlargement is produced by an hypertrophy of the connective tissue and an unusual deposit of fat.

The disease affects males more frequently than females. It usually occurs between the ages of two and eight, although in exceptional instances its appearance is delayed until about the twentieth year.

PATHOLOGY.—According to Gowers, the pathology is represented by a primary interstitial change in the muscles, showing a growth of fibrous tissue or of fat-cells which produces an increase in the size of the muscles. The muscular fibres are secondarily affected by this interstitial change, and are apparently narrowed by pressure. Atrophy, which is an especial feature of the affection, exists in the later stages of the disease in the muscles of the legs, and is frequently the primary pathological change in the muscles of the trunk and upper extremities. The development of fat-tissue between the atrophied fibres may prevent any diminution in the apparent size of the muscles. This often, in the muscles of the calves, may cause them to be much increased in size. In the upper extremities the deltoid and triceps are most commonly involved, in the lower the gastrocnemii. The infra-spinatus, the latissimus dorsi, and the pectoralis major muscles are also commonly affected. The lesion is usually symmetrical, affecting similar muscles on both sides of the body, but it may be unilateral. The muscles may be affected partially or completely.

SYMPTOMS.—The disease develops slowly, and the symptoms are those which would naturally be expected from the muscular lesions, and are quite characteristic.

The first symptoms usually noticed are a weakness of the muscles, and a shuffling, unsteady, swaying gait, with the feet apart and a tendency to stumble and to fall. The children get tired very easily, and are noticed ave difficulty in walking up-stairs. These general symptoms usually ede any noticeable enlargement of the muscles. The position on standing pt to be that of lordosis, and on sitting down this curvature disappears. The next symptom which is noticed is an enlargement of the calf-cles, which are usually hard and firm. In addition to this there is an phic condition of the muscles of the shoulders and back. The muscles t to those of the calves which are most likely to be affected are the nsors of the leg, the glutei, the lumbar muscles, the deltoid, triceps, infra-spinatus. The muscles of the neck, face, and upper arm are lly not affected, but in rare cases these muscles, as well as those of tongue, have been involved. Exceptionally an hypertrophy begins in upper extremities, and in these cases the deltoid muscle is usually first ted. At times only part of the muscle is involved. These children n to walk late, and assist themselves by leaning on the furniture or other cts in their path. Sometimes when they are kneeling on the hands and s there is noticed a very characteristic saddle-shaped depression of the k, which is due to the weakness of the erector spinæ muscles. This, ever, is a symptom of a late stage of the disease. When the child is ed on the floor on its back it has difficulty in getting up. It has to turn on its face first, and then to aid the weakened muscles of the legs and k by means of the hands and arms, climbing up, as it were, upon itself lacing the hands upon the knees and then farther and farther up the hs. Fibrillary contractions do not occur. The knee-jerks in some cases ppear as the disease advances. Sensation, as a rule, is normal. There ldom any disturbance of the bladder or rectum.

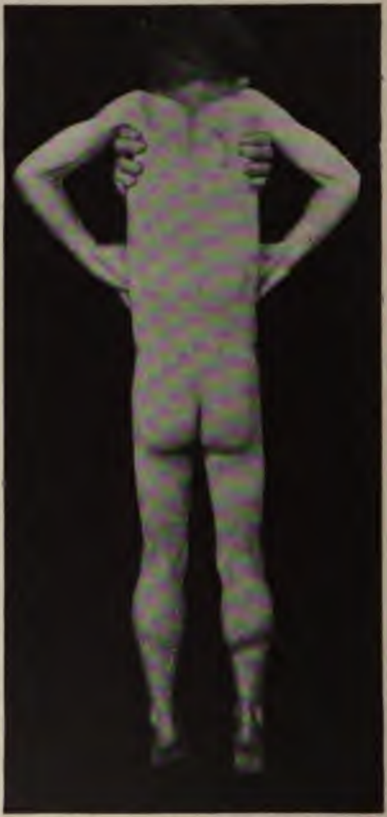
As the disease advances, the pseudo-hypertrophic condition disappears is succeeded by atrophy. In some cases the atrophy occurs without the eding hypertrophy. In the later stages of the disease contractions of muscles occur, and in this way permanent distortions of the joints may lt. The most common deformities are talipes equinus and flexion of knees and hips. There may be such a contraction of the biceps as to ent full extension of the arm, and in some cases the contraction of the cles of the calves is so great as to prevent the child from placing the s upon the ground.

According to Bradford and Lovett, lateral curvature of the spine may r, and at other times a permanent flexion of the spine from weakness he erector spinæ muscles, so that the child sits bowed forward. The trical reactions are not altered to any degree until the muscles have hed a marked stage of atrophy, then they are diminished in proportion he muscular wasting, and finally they are lost. The reaction of degen-ion is never present.

DIAGNOSIS.—The diagnosis of pseudo-hypertrophic muscular paralysis n the disease is well established is not difficult. The peculiar gait, the of the calf-muscles, entirely out of proportion to their strength, and the racteristic manner in which the child rises from the floor, at once suggest this affection. Gowers also attaches diagnostic importance to the coexistence of enlargement of the infra-spinatus and wasting of the latissimus dorsi and the lower part of the pectoralis major muscles. We should remember that in pseudo-hypertrophic muscular paralysis, in contradistinction to progressive muscular atrophy, the small muscles of the hand and of the face are, as a rule, not affected, that pain is usually not present, and that changes in the nutrition of the skin and nails do not occur.

CASE 384.

I.



Pseudo-hypertrophic muscular paralysis, showing enlarged calves.

We differentiate true muscular hypertrophy from pseudo-hypertrophic paralysis by the strength which accompanies the former, and by the weakness which occurs in the latter.

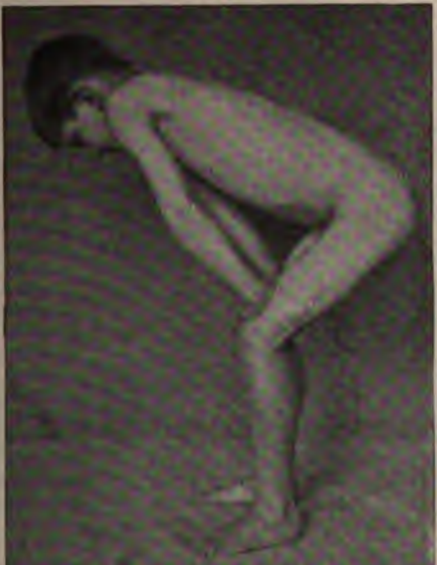
In the early stages of the disease it is at times difficult to distinguish simple backwardness in walking from early pseudo-hypertrophy, but the characteristic symptoms of pseudo-hypertrophy, which develop very soon, do not leave the diagnosis long in doubt. The same thing may be said of differentiating pseudo-hypertrophy from the muscular disturbances occurring in idiocy, spastic paralysis, rhachitis, and Pott's disease.

GNOSIS.—Recovery in this disease is unknown, and the children live to middle life. Death usually occurs from some intercurrent The course of the disease is chronic. The muscular weakness, the and the peculiar gait last for several months or a year. The hyper-f the muscles then begins, and continues progressively for one or s, when it reaches its maximum and becomes stationary, remaining veral years or even longer. A stage of increasing feebleness and n of the paralysis then begins, the muscles become more wasted, and er of motion is lost in the legs and arms. Sometimes the disease aining stationary rapidly advances to a fatal issue.

ATMENT.—At present we know of no way of curing the disease. has proved to be more beneficial than the use of electricity in these Systematic muscular exercise, for the purpose of preserving the of the unaffected muscular fibres and to ward off the permanent ures, is indicated. Where the muscles are drawn up, tenotomy is much use, and division of the tendo Achillis on both sides may ng time restore the power of walking. Bradford and Lovett enotomy of the hamstring tendons also, in severe cases. Strict to the health and hygiene of the patients, combined with muscular and tenotomy, will often improve the general condition for a con- period of time.

CASE 384.

II.



do-hypertrophic muscular paralysis. Showing position assumed in rising from the floor.

ve here a case which represents certain points which I have just of in describing pseudo-hypertrophic muscular paralysis, and which abled to show you through the kindness of Dr. Rupert Norton.

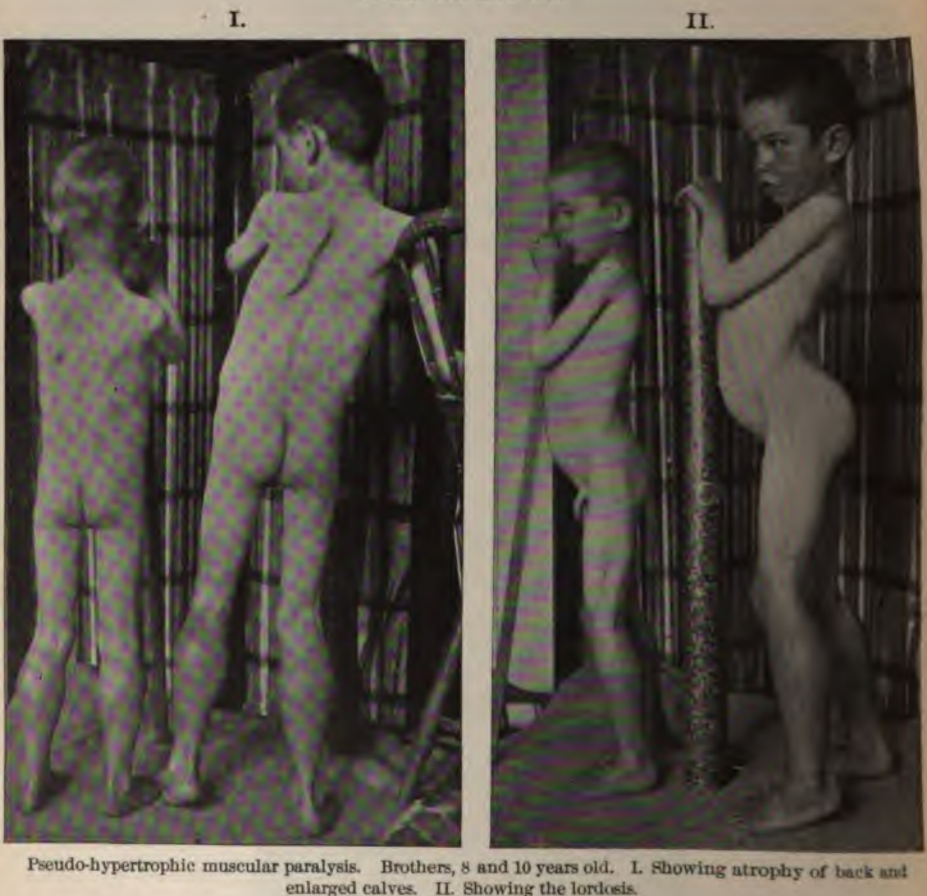
Looking at this boy from behind (Case 384, I., page 770), you will notice at once that the calves of the legs are greatly enlarged.

On making the boy lie down on the floor and then telling him to get up (II., page 771), you will notice that he assists himself by putting his hands on his knees and gradually higher and higher on the thighs until he assumes the erect position.

I shall now ask you to look at these illustrations of two brothers which I am enabled to show you through the kindness of Dr. H. N. Thomas, of Baltimore.

The history of these cases (Cases 385 and 386) is as follows. The smaller boy is eight years old, the larger ten years old. They have always lived in the country, and no mention of any especial disease has been obtained, but the history of both cases is unsatisfactory.

Cases 385 and 386.



Pseudo-hypertrophic muscular paralysis. Brothers, 8 and 10 years old. I. Showing atrophy of back and enlarged calves. II. Showing the lordosis.

The older boy began to walk when he was nineteen months old, but was clumsy and never walked well. When he was seven years old he began to have difficulty in going upstairs, and it was noticed that the calves of his legs were growing larger, while his arms were becoming smaller. The curve in his back was first noticed when he was eight years old. When nine years old he lost the power of walking, and is said to have grown rather stupid.

The younger boy was always delicate, but never had any disease, and began to walk when he was fifteen months old. He learned to walk pretty well, but when he was four years old he began to show weakness in the legs and ankles, and this weakness increased steadily. When he was six years old his arms began to get smaller and his legs to increase in size.

You see that both boys show marked lordosis.

MYOTONIA CONGENITA (Thomsen's Disease).—A third form of primary myopathy, which is usually termed Thomsen's disease, from the name of the physician who first thoroughly described it, is characterized by an inhibition of the voluntary movements. This disturbance of movement is due to a stiffness and tension of the muscles occurring at the commencement of motion. The most important etiological factor in the disease is that it is hereditary. In almost every case it begins in early childhood.

The pathology of the disease has not been accurately established by means of autopsies, but an examination of sections of muscle taken from these cases has shown, according to Erb and Jacoby, that the morbid changes are the result of an enormous hypertrophy of all the muscular fibres, great proliferation of the nuclei, and a slight increase of the perimysium. The disease appears to be a congenital affection of the muscular fibres.

The symptoms of this disease are noticed only during voluntary movements, the contraction of the muscles responding very slowly to the will, and persisting for a little time after the individual has willed the muscular movement to cease. The muscles of the arms and legs are those usually implicated. The sensation and reflexes are normal. The muscles are apparently enlarged, giving at times the appearance of hypertrophy, but the strength of the muscle is not proportionate to its size. Erb has described a characteristic electrical reaction, called the myotonic reaction, in which the contractions caused by either current attain their maximum slowly and relax slowly, and wave-like contractions pass from the cathode to the anode. One of the peculiarities of the disease is that when exertion is made, such as attempting to go up-stairs, the muscles which previously were quiescent become very stiff and will scarcely respond to the will. Another peculiarity is that long-continued rest makes the disorder worse. It is also exaggerated by heat, cold, and excitement.

Since the discovery of the myotonic reaction the diagnosis of the disease is not difficult.

Although at times it may recover temporarily, the disease is incurable, and there is no known treatment which is of benefit to it.

DIVISION XII.

DISEASES OF THE MOUTH, NOSE, NASO-PHARYNX, AND PHARYNX. DIPHTHERIA.

LECTURE XL.

DISEASES OF THE MOUTH.

STOMATITIS CATARRHALIS.—STOMATITIS HERPETICA.—STOMATITIS ULCEROSA.—STOMATITIS MYCETOGENETICA.—GLOSSITIS.—MICROGLOSSIA.— MACROGLOSSIA.—DIFFICULT DENTITION.

IN speaking of diseases of the mouth you must understand that much confusion exists as to the nomenclature of this class of affections. Thus, a great variety of names has been used by different authors to describe the same disease, so that at times it is quite difficult for one investigator to compare his work with that of another. Such terms as "canker" and "aphthæ" are so commonly used for almost any morbid condition of the mucous membrane of the mouth that they have ceased to convey any definite idea.

It has therefore been found necessary, in order that any advance should be made in this most difficult department of medicine, to adopt some more exact nomenclature, so that physicians in different parts of the world should in every case use the same name for the same disease. In furtherance of this object the American Pediatric Society has adopted a provisional nomenclature of the diseases of the mouth which was prepared by Dr. Forchheimer, of Cincinnati, and myself. To Dr. Forchheimer's extended investigations on this subject I am much indebted.

Diseases of the mouth occur more frequently and in much greater variety in infancy and in early childhood than at any later period of life. This depends partly on the anatomical conditions at different periods of development and partly on the external influences which are brought to bear upon the buccal mucous membrane. During the first three or four months of life the function of the salivary glands is not developed, and the flow of saliva is insignificant. This lack of saliva allows the mucous

brane of the mouth to be dry in comparison with that of the older
ect. Even after the saliva is secreted the infant is more apt under
in conditions to let it flow from the mouth than to swallow it, so
the mucous membrane of the lips and mouth may present a different
arance in young infants, when they are attacked by various morbid
esses, from that seen at a later period of development. We must also
ember that the salivary glands in addition to their especial function are
etory organs, and that substances which are absorbed by the stomach
be eliminated by the mouth and in this way become sources of irri-
on and disease in the latter. The mucous membrane of the mouth
ing almost the whole period of infancy is subject to external sources of
ation to which older children, as a rule, are not liable. Thus, during
first year the mucous membrane is subjected to more or less mechanical
ation through the mechanism of sucking. At this period, also, it is
common for foreign organisms to be introduced into the mouth by
ns of the fingers either of the infant itself or of its attendant. It is
surprising, therefore, that we should meet with a great variety of patho-
cal conditions in the mouth in infancy.

The organisms which occur in the mouth are so numerous that very
of them have as yet been differentiated in such a way that they can be
wn as the cause of the specific disease in which they are often found.
cannot, therefore, at the present time describe the various diseases
he mouth under their proper etiological headings, and we are forced
dopt provisionally the name of the pathological lesion which exists in
n.

In almost every disease of the mouth which occurs in infants and in
ng children you will find a coexisting inflammation of the mucous
brane of the mouth. This inflammation may at times be very mild
often difficult to detect as such, but it still presents a recognizable
ological condition. This inflammatory condition, though not neces-
ly preceding the various diseases, yet in a large number of cases either
ts as a basis on which the disease develops, or so closely accompanies it
the general name *stomatitis* (inflammation of the mucous membrane of
mouth) seems to be a proper term to use in connection with all these
ases.

Under the general heading stomatitis we can speak of most of the
ortant diseases which affect the mucous membrane of the mouth in
ncy and early childhood. These diseases may be divided into four
ral headings, according to the character of the lesions which occur in
n. In order that you may readily understand the classification of each
ase when I speak of it, I shall first show you a table (Table 107) of the
isional nomenclature which has been adopted by the American Pediatric
ety.

TABLE 107.

Provisional Nomenclature of Diseases of the Mouth.

STOMATITIS	Catarrhalis	Simplex.	
		Exanthematica	Secondary to the Exanthemata.
		Traumatica	Mechanical. Thermal. Chemical.
	Herpetica	Aphthosa.	
	Ulcerosa	Scorbutus.	
		Mineral Poisons	Arsenic. Lead. Mercury.
		And other diseases.	
	Mycetogenetica	Hyphomycetica	Thrush.
		Pseudo-Membranosa	Diphtheria. Tuberculosis. Syphilis, and like diseases.
		Gangrænosa	Noma.

Following this table, you will see that the four general names which cover all these diseases are *stomatitis catarrhalis*, *stomatitis herpetica*, *stomatitis ulcerosa*, and *stomatitis mycetogenetica*.

STOMATITIS CATARRHALIS.—The form of stomatitis which is called the *simple* or *erythematous* form (stomatitis simplex) is commonly seen in young infants as a hyperæmic condition of the blood-vessels, causing diffuse redness of the whole buccal mucous membrane. This erythematous form is so common and so entirely without clinical significance that it may be considered as physiological and need only be referred to.

The second form, which is called *exanthematica*, is the condition of the mucous membrane which occurs secondarily to the exanthemata. This condition of the mucous membrane has already been described in connection with these diseases, and therefore need not be spoken of again.

The third form, which is called *traumatica*, is the one which represents the characteristic *stomatitis catarrhalis*. The causes of the traumatic form of stomatitis catarrhalis are very numerous. They may be *mechanical*, *thermal*, or *chemical*. Among the most common mechanical causes may be cited the irritation produced by rubber nipples, too vigorous cleansing of the mouth, injudicious rubbing of the gums during dentition, and local irritation from a tooth. The thermal form of traumatism may result from the administration of food which is too hot. The chemical irritation may arise in various ways, as from lack of cleanliness in the mouth, with its resulting fermentation, and from the elimination of irritating products from the glands apparently connected in some way with disturbance in the gastro-enteric tract. It is probable also that various forms of bacteria or their products may cause both mechanical and chemical irritation of the buccal mucous membrane. Our knowledge of the bacteriology of the mouth is as yet, however, so limited that we can scarcely undertake to describe the relation between special forms of bacteria and special lesions of the mucous membrane.

Pathology and Symptomatology.—As the lesions which are seen in the mouth of an infant with stomatitis catarrhalis during life almost entirely disappear at death, and as very few post-mortem examinations have been made of these lesions, we can speak of the pathology and symptoms of this disease together.

The lesion is essentially an inflammatory one, and occurs in different grades. On examining the mucous membrane in these cases it is seen that the entire lining of the mouth is intensely reddened, that the temperature of the mouth is increased, that there is usually a certain amount of swelling, and that, although the mucous membrane may be under certain circumstances, especially at first, dry, yet, as a rule, later there is a hypersecretion of mucus and saliva. The blood-vessels are so distended and their walls are apparently so weak that the slightest traumatism may cause their rupture, and the saliva is frequently mixed with a little blood. In older children the mucous membrane may be considerably swollen, especially behind the incisor teeth. In addition to this general condition of the mucous membrane of the mouth, at times the lips are found to be swollen and much reddened. The surface of the mucous membrane shows a number of small round prominences, which are the muciparous follicles. If complete occlusion of the ducts of these follicles occurs, great dilatation of the gland will take place, and a cyst may be formed. This, however, is a comparatively rare complication. In connection with the disturbance of the glands in the mouth the lymphatic glands are usually involved secondarily.

When the catarrhal condition is at its height the mucous membrane is so vulnerable that even slight traumatisms may cause abrasions. The most marked symptom of stomatitis is pain. The infant is restless, usually has a heightened temperature, and refuses to take its nourishment. The saliva is acid in its reaction, and when secreted in large quantities flows out of the mouth upon the chin and neck, sometimes causing considerable irritation. The tongue is dry and white at first, then becomes of a grayish color, and as the secretion of saliva increases the coating of the tongue is washed off and its surface becomes red.

Prognosis.—The prognosis of stomatitis catarrhalis is, as a rule, good. Although the disease does not run a definite course, yet in most cases after a few days the pathological condition improves and the symptoms grow less severe. The course of the disease is, however, often lengthened by the secondary conditions which arise from the gastric disturbances, which may be caused by swallowing the irritating secretions of the mouth. In weak, poorly nourished infants who refuse to nurse or to take the food which is given them, serious results may arise from a lack of sufficient nourishment, so that in these cases the prognosis is always grave. In older children the disease may be considered to be of a benign nature.

Treatment.—Although stomatitis catarrhalis may run a favorable course without any treatment whatever, yet there are so many causes which may prolong its course or give rise to secondary affections that it is exceedingly important to treat the disease at once. The indications for treatment are to relieve the pain and to allay the irritation of the mucous membrane so that a sufficient amount of nourishment may be taken by the infant to prevent it from being harmed by a lack of nourishment or by a secondary disturbance of the gastro-enteric tract. If the cause can be ascertained, it should be removed at once. The local application of a one to two per cent. cold solution of bicarbonate or borate of sodium in distilled water is indicated. This solution should be used very gently every half-hour when the infant is awake, by means of a dropper, and occasionally on a clean swab of absorbent cotton. The infant should be systematically fed at regular intervals, whether it resists or not; and if it is not being nursed or will not suck from the nipple, a carefully modified milk at a temperature of about 32.2° C. (90° F.) should be administered with a spoon or dropper. There is no necessity for giving any drug internally in this disease.

Where the stomatitis proves to be intractable and lasts for more than three or four days, the mouth can be gently touched with a cotton swab wet with a one per cent. solution of nitrate of silver. This should be done once a day, and the mouth washed carefully with cold sterilized water after the application.

Where there are any abrasions which show a tendency to extend or to form an ulcer, they should be touched with a little nitrate of silver melted on the end of a silver probe. These abrasions are often so painful that in themselves they may prevent the child from taking its food, and after they have been treated with the nitrate of silver the child will often again take its nourishment readily.

I have here an infant (Case 387), six months old, who is a marked case of stomatitis catarrhalis. This infant is reported to have always been healthy, and is being nursed by its mother. It cut its first tooth, a middle lower incisor, when it was five months old. Nothing abnormal was noticed about the infant until two weeks ago, when it became fretful, restless, had a heightened temperature of about 38.8° C. (102° F.), and vomited occasionally. Although it did not cry a great deal, it frequently whimpered, as though in pain, and kept putting its fingers to its mouth. A few days later it refused to nurse. When it was put to the breast it appeared to be hungry and would take hold of the nipple vigorously, but immediately afterwards would draw its head away, as though sucking the nipple caused pain.

A physical examination shows nothing abnormal about the infant except in its mouth. The mucous membrane of the mouth, tongue, and gums is reddened, and small raised spots are seen corresponding to the positions of the muciparous glands. The mucous membrane of the tongue and lips is somewhat swollen and hot, and evidently sensitive to the touch. Where the tooth touches the tongue the inflammatory condition is especially marked, and it is possible that the sharp edge of the tooth was the original starting-point of the general inflammation which is now present.

In this case the indications are for active treatment, as the infant is losing in weight lack of sufficient nourishment, and if this continues the prognosis will soon become e. When the mouth is in this condition there is also a great liability to other diseases g implanted upon it, as the mucous membrane is very vulnerable when a pronounced atitis catarrhalis is present. The saliva is flowing from the mouth in such quantities is so irritating that an eczematous condition has been produced by it on the chin. child is rather apathetic and does not like to be disturbed. The treatment which I order in this case is that the mouth be carefully washed with the following solution scription 74):

PRESCRIPTION 74.

Metric.	Gramma.			*Apothecary.*	
odii boratis	1	8	℞	Sodii boratis	gr. xxx;
lycerini	7	5		Glycerini	ʒii;
q. destil.	ad 120	00		Aq. destil.	ad ℥iv.
M.				M.	

This should be applied every hour while the child is awake. The chin should be uently dried gently and a little vaseline applied to the eczematous surface. Until the d is willing to nurse again, the milk should be given by means of a dropper regularly y two hours. Under this treatment I shall expect rapid improvement within four or days.

STOMATITIS HERPETICA.—The name herpetica has been adopted the next form of stomatitis, because it seems to represent most nearly lesion which is seen on the mucous membrane, although it is not detively settled that it is a true herpes.

The disease consists of a catarrhal stomatitis in the course of which tain lesions resembling subepithelial vesicles surrounded by areolæ occur gularly and in different parts of the entire buccal cavity. This form of matitis has usually been known as stomatitis aphthosa (ἄφθα, an eruption ulceration.) This name was given to it by Bohn as distinctive from the er forms of stomatitis, but it does not represent the affection especially ll.

ETIOLOGY.—As a rule, when the mucous membrane of the infant's uth is in a normal condition it is not readily affected by the various irrits which produce its special diseases. When a catarrhal condition is sent the mucous membrane becomes more vulnerable and the various eases have an opportunity to develop. This apparently is illustrated in case of stomatitis herpetica, in conjunction with which affection a arrhal stomatitis is always found. No cause, either local or general, has yet been determined for this disease. Various micro-organisms have n observed in the mouth when it is affected by stomatitis herpetica, but causal connection has been discovered between them and the disease. is affection may be found associated with a number of other diseases, but ally occurs alone. It does not seem to be contagious, nor to be especially nected with diseases of the gastro-enteric tract or with dentition, although very commonly occurs during the dental period. It appears to be the ult of certain deleterious influences which act upon the nerve-centres and duce an herpetic efflorescence on the mucous membrane which corresponds ely to that which is seen in herpes on the skin.

PATHOLOGY AND SYMPTOMATOLOGY.—In addition to the usual lesions of a stomatitis catarrhalis, spots, not necessarily symmetrical or unilateral, of different sizes and of different shades of white or grayish-white, appear in various parts of the mouth, especially on the inner surface of the lip, on the side and under surface of the tongue, and on the gums. These lesions do not affect the follicles of the mouth, and the efflorescence cannot be called follicular, as it is closely connected with the muciparous glands. The lesions make their appearance with great rapidity, and develop very quickly from a macule into what is supposed to be a vesicle. The action of the secretions of the mouth upon these lesions necessarily prevents them from having the same definite appearance that they would present on the skin. The course of the disease so strongly simulates that of herpes that at present it would seem wise to consider the efflorescence herpetic.

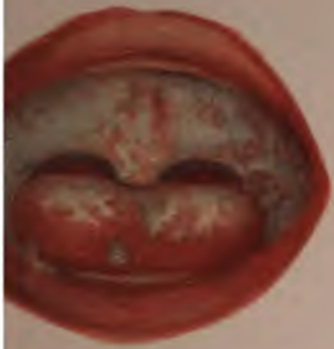
The general appearance of the efflorescence when at its height is that of a subepithelial vesicle, somewhat glistening, of a whitish-gray color, and surrounded by a red areola. The lesions may be only a few in number, scattered irregularly over the parts of the mucous membrane which I have already described. At times, however, the efflorescence is very diffuse, sometimes appearing as minute grayish points, which may become much larger and cover the mucous membrane so thickly as almost to simulate a false membrane. In a still later stage of the disease these lesions may break down and form small superficial ulcers.

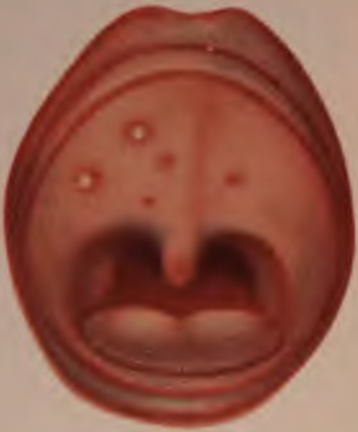
An infant or young child affected by stomatitis herpetica presents a very characteristic appearance. It looks dull and apathetic, and wishes to lie quietly in bed. It usually has a heightened temperature, and evidently suffers from pain and heat in its mouth. The saliva flows from the mouth in large quantities, and often irritates the chin and neck to such an extent that an eczematous condition results. The child refuses to take its nourishment, and is very fretful and restless. These symptoms continue for four or five days or a week, and sometimes extend over a period of two weeks, the disease then disappearing of itself: in fact, it appears to be self-limited. Unless the lesions of stomatitis herpetica are complicated by those of stomatitis ulcerosa, the saliva is never fetid.

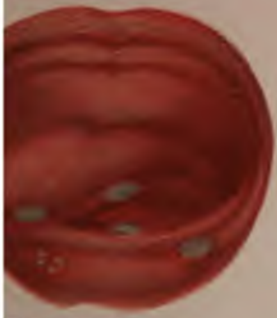
PROGNOSIS.—The prognosis of stomatitis herpetica is very favorable, although infection from other diseases may take place. This latter occurrence is, however, exceedingly rare. Relapses are very uncommon in this form of stomatitis, and the lesions usually heal readily.

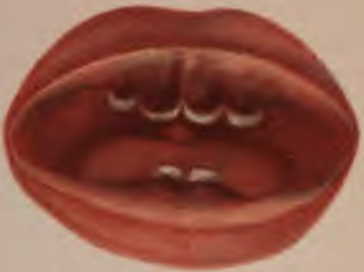
TREATMENT.—There is no internal treatment which is of benefit in this disease. The indications for treatment are to allay the irritation of the mucous membrane and to prevent its infection by some other poison. The mouth in general should be treated as I have just recommended in the case of stomatitis catarrhalis. As a rule, very little treatment is necessary beyond occasionally cleansing the mouth with the solution (Prescription 74, page 779) already mentioned. The ulcers which do not heal readily can be touched with nitrate of silver. After the first few days, and earlier if

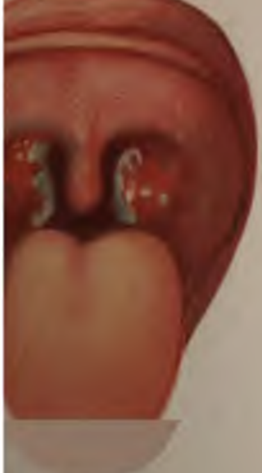
PLATE VIII

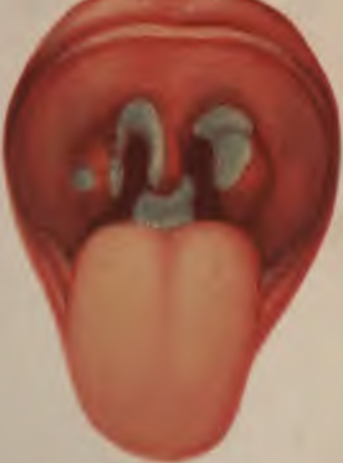

Thrush


Varicella


Stomatitis Herpetica.
(Aphthosa.)


Stomatitis Ulcerosa
(Scorbutus.)


cular Tonsillitis


Diphtheria

the disease has attacked a puny, ill-nourished infant, great care and perseverance should be exercised to feed at regular intervals.

This boy (Case 388, Plate VIII., Stomatitis Herpetica), four years old, whom you see here in a darkened corner of the ward, is a pronounced case of stomatitis herpetica. He was perfectly well until two days ago, when he began to be feverish, was restless at night, refused to take his food, and seemed quite sick. On the following day the entire mucous membrane of the mouth was found to be affected with stomatitis catarrhalis, and somewhat later the herpetic form of stomatitis, which you now see in different parts of the mouth, appeared.

On drawing down the lower lip you see on the right side a number of small grayish-white spots surrounded by a somewhat deeper, reddened mucous membrane. At a little distance from them, on the left side of the lip, close to the gum, is apparently a subepithelial vesicle. On the inner side of the lower gum one of these vesicles has broken down, and a small superficial ulcer covered with a grayish-white exudation is seen. There are also lesions of the same vesicular character along the left edge of the tongue. The entire mucous membrane of the mouth is intensely reddened, and the case illustrates stomatitis catarrhalis as well as stomatitis herpetica.

The child absolutely refuses to take food, and, as he is robust, I have not advised that a great deal should be forced upon him. In a few days the more severe stage of the disease will have passed away and he will take his food. In the mean time the inflamed mucous membrane can be bathed with cold sterilized water, and small quantities of an alkaline modified milk can be given to him. As you look at this child lying with his eyes half closed, with flushed cheeks, in an apathetic condition, occasionally whimpering as if in pain, and with the saliva flowing continuously from his mouth on the pillow, you can readily diagnosticate the disease stomatitis. When in addition you see these characteristic lesions of the mucous membrane irregularly distributed throughout the buccal cavity, and do not find any evidence of a membranous exudation, there need be no doubt of the diagnosis. Internal remedies are not needed in a case of this kind. Chlorate of potassium, which is so commonly used in all diseases of the mouth, is not indicated in the forms of stomatitis of which I have just spoken.

In connection with this form of stomatitis may be mentioned certain esions occurring in the mouths of new-born infants which have been called *Bednar's aphthæ.* These lesions consist of small superficial ulcers usually aving a grayish coating, and appearing on the posterior part of the hard alate and on the soft palate. They are now supposed not to represent a pecific disease, but to be the result of traumatism, such as may arise from a badly-shaped rubber nipple or from undue violence in washing the mouth.

They are to be treated as any local irritations of the mouth should be, —namely, by removing the cause, applying a solution of bicarbonate of sodium, and, if necessary, touching them with nitrate of silver.

STOMATITIS ULCEROSA.—By stomatitis ulcerosa we mean a peculiar pathological process of the mucous membrane of the mouth occurring only where there are teeth and affecting the gums around the teeth.

ETIOLOGY.—This affection of the mouth may occur in the course of a number of diseases, notably in scorbutus. It may also be produced by the internal administration of such mineral poisons as arsenic, lead, or mercury. Occasionally it may occur as a local affection without known cause, but it is probably produced by the irritation of some form of micro-organism not yet determined, although the pyogenic bacteria are very commonly present.

The most common form of stomatitis ulcerosa produced by the mineral poisons is that which is seen in connection with mercurial salivation.

As in the other forms of stomatitis, it is probable that the mucous membrane is first affected by a catarrhal process which renders it vulnerable to the special irritation which produces stomatitis ulcerosa. This preceding stomatitis catarrhalis may be produced directly by local irritation in the mouth itself, or may be the result of some disturbance of the general system. For this reason stomatitis ulcerosa, as a rule, does not affect primarily a healthy individual. Thus, a poorly nourished child, and one whose mouth is not properly cared for, will be more apt to have this disease develop than one who is correctly fed and whose mouth is clean.

PATHOLOGY.—The pathological condition is one of necrobiosis; that is, there is softening as well as death of the tissues. The disease, although starting in the mucous membrane, may extend to the periosteum, and even produce necrosis of the bone. It begins at the free border of the gums, and can extend in all directions, but it never passes beyond the mucous membrane of the mouth. The softening of the tissue not only changes its consistency but also renders it more movable, and in this way the gums at times become so swollen and loosened that they may entirely cover the teeth.

SYMPTOMS.—Stomatitis ulcerosa is usually preceded by moderate constitutional symptoms, such as fever, loss of appetite, and fretfulness. The mucous membrane of the gums at the free margin of the teeth becomes reddened and soon begins to swell. The normal curve of the gum becomes almost a straight line and covers the lower part of the teeth. The gums in the spaces between the teeth remain unaltered at first. The mucous membrane then begins to change in color and becomes purplish. Extreme congestion and softening of the tissues allow hemorrhage to take place from the slightest pressure. Although the anterior surface of the gums is most commonly affected, yet in severe cases the posterior surface is also involved. As the process develops further the gum becomes more and more loosened as it extends over the teeth. A muco-purulent secretion collects between the gum and the teeth and causes a fetid odor. According to Forchheimer, a yellowish seam then appears at the top of the swollen outline of the gum. This is due to the molecular destruction which has already begun. This seam is at first very narrow, but later it may become broader and involve almost the whole of the gum. In connection with this characteristic appearance of the gums there is a great hypersecretion of saliva. At the height of the disease the child evidently suffers from pain in the mouth, cries a great deal, and rapidly emaciates. The lymphatic glands are usually swollen, and remain so until the disease has ended. When the yellowish material which constitutes the seam already referred to is removed, an ulcerated surface will be found beneath. Although stomatitis ulcerosa may begin about any of the teeth, its most common starting-point is around the lower incisors. As the disease improves, the gums gradually become less swollen gested, returning to their normal relation to the roots of the teeth, salivation disappears.

GNOSIS.—The differential diagnosis of stomatitis ulcerosa when the of the disease are marked presents no difficulty. Although an efflorescence may occur coincidently with the ulcerative form, yet ures of the two diseases are so different that you will at once know are dealing with two affections rather than with one. There is no isease of the mouth in which the gums assume the purplish hue swollen, soft, and loosened condition which are characteristic of is ulcerosa.

GNOSIS.—The prognosis of stomatitis ulcerosa depends upon its nd whether it is treated or not. The tendency is, however, after a period of discomfort to the child, for the disease to disappear. he affection is the result of one of the constitutional diseases, such as or scorbutus, it disappears if the treatment of the specific disease is l, otherwise it continues, and may finally lead to death by exhaustion.

ATMENT.—The local form of the disease is best treated by the administration of chlorate of potassium or by this drug in solution a wash for the mouth. Chlorate of potassium must, however, be ith great precaution to infants and children, as in certain cases it a poison, some infants being affected by even minute doses. The ns which show that chlorate of potassium is producing deleterious infants who are most likely to be affected by the drug are drowsi- d suppression of urine, with weakness of the heart and sometimes . When these symptoms follow the administration of the drug it be omitted at once and a simple wash of borate of sodium used. of potassium when given internally has been found to be secreted aliva within five or ten minutes, and thus has an opportunity of ng a direct effect upon the lesions of the gums. The doses of chlo- potassium which it has been found can be safely administered to and children should be remembered when prescribing the drug. I licated in this table (Table 108) the minimum doses which can safely in the twenty-four hours at different ages, and which are sufficient uce the specific effect of the drug in treating cases of stomatitis .

TABLE 108.

t of Chlorate of Potassium which can be safely given in Twenty-Four Hours at Different Ages.

Age.	Gramme.	
Under 1 year	1	0
1 to 2 years	1	5
2 to 6 years	2	0
6 to 8 years	2	5
8 to 14 years	3	0

rder that the chlorate of potassium shall produce the best effects it e given frequently. The total amount for twenty-four hours which is to be given at any special age is to be placed in a tumbler and dissolved in as many tablespoonfuls of sterilized water as there are doses to be given within the twenty-four hours. I usually tell the nurse to calculate about how many hours the child will sleep out of the twenty-four. Supposing the number of hours is ten: I then tell her to prepare fourteen tablespoonfuls of the solution and to give the child one tablespoonful every hour that it is awake. The administration of chlorate of potassium at first usually produces considerable smarting and pain in the mouth as it passes over the inflamed surface of the mucous membrane. These symptoms, however, last for only a short time, usually disappearing entirely after from thirty-six to forty-eight hours.

Under this treatment the disease is ordinarily cured in a week or ten days. The treatment should, however, be continued for a number of days after the mouth is apparently entirely well.

Where deeper ulceration has taken place, its disappearance may sometimes be expedited by the application of nitrate of silver. Where a sequestrum has formed, it must be removed. Frequent washing of the mouth with sterilized water administered by means of a dropper is also very important, especially after the taking of food.

I have here an infant (Case 389, Plate VIII., facing page 781, Stomatitis Ulcerosa in Scorbutus), ten months old, in whose mouth you will see the characteristic lesions of stomatitis ulcerosa. In this case the disease happens to be secondary to scorbutus, the affection for which the infant is being treated.

You will notice that the infant has six teeth, and that the mucous membrane is affected only at the junction of the gums with the free surface of the teeth. The other parts of the mucous membrane of the mouth are reddened, but not markedly so. The portions of the gums affected are swollen, purplish, loosened, and almost cover the teeth. There is a considerable flow of saliva, with a fetid odor from the mouth. An appearance of this kind is diagnostic of stomatitis ulcerosa.

I also have here a case of stomatitis ulcerosa which apparently is of local origin. This little girl (Case 390) is three and a half years old. She has always been healthy, and has had no diseases of any kind. She was perfectly well until five days ago, when she began to have loss of appetite, a temperature varying from 38.3° to 39.4° C. (101° to 103° F.), and to be very fretful. Three days later the gums were noticed to be swollen and to be of a dark red color, and her breath had a fetid odor. During the past two days 1.5 grammes (25 grains) of chlorate of potassium have been given to her in divided doses in the twenty-four hours, and, although she has been rather apathetic and has wished to remain in bed, her mouth to-day is in a much healthier condition, and she is brighter and has a little return of appetite.

In two or three days more the disease will probably have run its course and entire recovery will have taken place. The salivation, which was very marked in the early days of the disease, is now quite moderate.

During the first three days her restlessness was so excessive at night that 0.3 gramme (5 grains) of bromide of potassium had to be given to her to produce sleep.

STOMATITIS MYCETOGENETICA.—There are three forms of vegetable parasites which occur in or upon the human body: (1) bacteria, or fission-fungi (schizomycetes); (2) yeasts, or yeast-fungi (saccharomycetes); (3) moulds, or mould-fungi (hyphomycetes). The changes in the tissues which are due to fungi are termed mycetogenetic metamorphosis, and thus he pathological conditions in the mouth which are produced by any of hese forms of fungi may be designated by the general term mycetogenetica. Under this general heading of mycetogenetica we can include the various forms of stomatitis which are caused by fungi.

Stomatitis Hyphomycetica (Thrush).—The disease which is commonly called thrush is produced by a fungus which finds its nidus upon the urface of the mucous membrane of the mouth, usually in young infants. This fungus was formerly supposed to be the *oïdium albicans*, but it is now known not to be this organism, and the precise form of mould which it represents has not yet been determined. We merely know that this growth of thrush is one of the mould-fungi, and we can therefore at present only classify it as *stomatitis hyphomycetica.*

The moulds are complex in their structure, and as commonly described consist of a series of delicate jointed threads (mycelium) in which spores are developed. Hyphomycetic growth is characterized by having the spores naked on conspicuous threads. The fungus of thrush may be found on any of the mucous membranes of the body. It has also been found in various organs, as in the brain and the lungs, and from the surface of ulcers it has on rare occasions penetrated the blood-vessels and given rise to visceral metastasis. The usual place for it to appear, however, is the mucous membrane of the mouth. It is a local disease, and may occur in the mouths of healthy children as well as in those who are diseased. It is more likely, however, to be ingrafted upon a diseased than upon a healthy mucous membrane, in accordance with the rule which I have already stated. A catarrhal condition of the mucous membrane, by displacing the epithelial cells and thus interfering with their protection of the mucous membrane, affords the readiest means for the development of the fungus of thrush. It is therefore more likely to be found in the mouths of children who are suffering from various diseases or who are ill cared for. It may be carried to the mouth in various ways, either on dirty nipples or by the finger.

Pathology.—The growth may take place on both squamous and cylindrical epithelium. According to Forchheimer, the first lodgement of the fungus comes between the epithelial cells of the mouth, and from this the growth works its way under the free surface of the mucous membrane. When directly on the free surface the growth is not so luxuriant and is principally in the mycelium form. In the case of a mucous membrane lined by flat or squamous epithelium, the growth is facilitated by the relation of the cells to one another. In a membrane lined by cylindrical epithelium the growth takes place, but not so readily, because there is but one layer of cells. After the first development the growth goes on very rapidly, and after it has found a nidus the cells are pushed aside and are surrounded by mycelium, the whole presenting the characteristic appearance of thrush. The growth begins in small spots, sometimes one, sometimes more, and at times the entire surface of the mucous membrane is covered with it. The fungus develops within the epithelium, and it requires considerable rubbing to remove the growth from the mucous membrane.

SYMPTOMS.—An attack of thrush usually begins with local symptoms of catarrhal stomatitis. At times, however, no symptoms are present, the fungus being the first abnormal condition which is noticed. The appearance of the fungus resembles closely that of curdled milk, though it is often of a rather grayish color. It does not look like a membranous exudation, but is raised in small patches above the level of the mucous membrane. The fungus usually develops on the inner borders of the lips, on the gums, on the tongue, and on the hard and the soft palate. It may extend to the tonsils and pharynx, and even into the œsophagus. In the latter locality at times it has been found to grow so thickly that the lumen is almost entirely occluded. The local symptoms are commonly those of a mild catarrhal stomatitis. The general symptoms depend upon the extent of the local disease from which the infant is suffering. Infants affected with this disease soon become atrophic, from a lack of proper nourishment, as they are often unwilling to take their food or cannot swallow it without difficulty.

DIAGNOSIS.—The differential diagnosis is seldom difficult to make. Curdled masses of milk on the inner surfaces of the lips and on the gums may resemble closely the fungus of thrush, but the former is easily wiped away, while the latter is difficult to dislodge. The disease is definitively determined by placing some of the growth under the microscope, where it presents characteristic appearances which I shall presently show you.

PROGNOSIS.—The prognosis of thrush varies according to the general condition, the vitality, and the age of the subject on whom it is engrafted. The disease may last indefinitely if the mouth is not carefully treated, and its prolongation may render the prognosis more grave. Where the growth is very extensive, as in the cases where it has invaded the œsophagus, the prognosis is very unfavorable. In these cases disturbances of the gastro-enteric tract are apt to arise and to increase the likelihood of a fatal issue. As a rule, however, if the infant's health can be maintained, and if the local treatment is carried out thoroughly, the prognosis is favorable.

TREATMENT.—The treatment should be directed to the local care of the mouth and to supporting the strength by proper nourishment and stimulants until the fungus has been eradicated. Care should be taken that everything connected with the infant, especially the nipples and bottles from which it is to be fed, should be aseptic, so that it shall not be continually reinfected or infect other children. The mouth after each feeding, and also between the feedings, should be thoroughly and somewhat vigorously rubbed with the solution (Prescription 74, page 779) which I have already recommended in the treatment of stomatitis catarrhalis.

Where the disease is in the œsophagus it is best treated by the introduction of a soft rubber tube, in order that the growth may thus be mechanically separated from the mucous membrane.

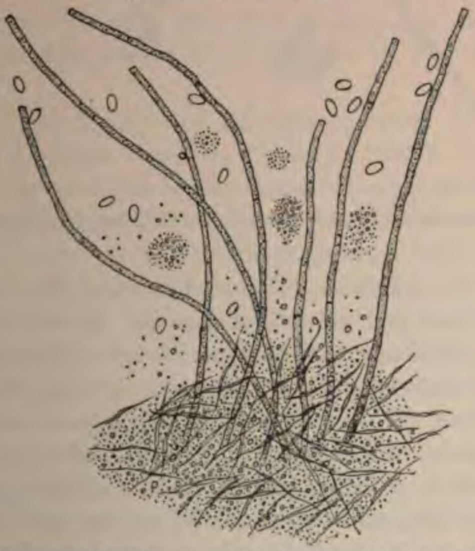
In many cases the disease is very intractable. No special drug appears o be of use in these cases, and they can be cured only by the unremitting nd patient removal of the growth as I have just described.

I have here an infant (Case 391, Plate VIII., facing page 781, Thrush), three months ld, who has refused to take the bottle for the past month, is emaciated and fretful, and at imes vomits.

A careful physical examination fails to detect anything abnormal except in the infant's nouth. On gently depressing the tongue and lower jaw, it is seen that the soft and the hard alate, the tongue, the gums, and the inner surface of the lips are covered almost entirely rith white and grayish-white masses, in texture somewhat resembling curdled milk, and ising above the level of the epithelium. Between these patches the mucous membrane is eddened. There is a moderate flow of saliva. This morbid growth apparently does not xtend into the pharynx. On endeavoring to remove one of these patches you see that cannot be done readily, as would be the case if it were curdled milk, but that it has vidently passed between the epithelial cells down to the underlying mucous membrane, here it is held so closely that it requires considerable rubbing to separate it. In this case he growth is so extensive that it simulates a membrane in some places, but its generally oughened surface, its elevation above the level of the mucous membrane, and the charac-eristic appearances in other parts of the mouth render its recognition quite easy.

On placing some particles of this growth in glycerin under the microscope (**Fig. 98**), ou see a tangled mass of fine, almost translucent, membered threads.

CASE 391. FIG. 98.



Mycelium of thrush interspersed with spores and fatty degenerated cells. (Low power Zeiss Oc. 3, Objective DD, glycerin.)

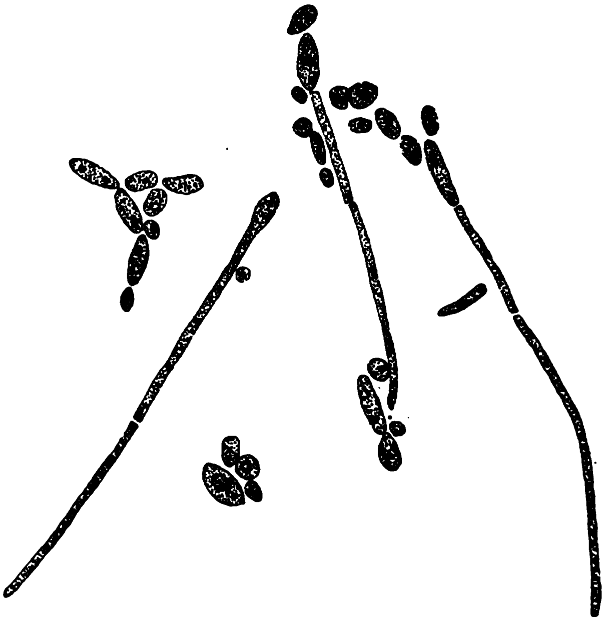
Interspersed among these are bright, glistening, oval bodies, which are the formed pores, and also fatty degenerated cells and fine detritus. This combination of appearances epresents the pathological processes which we find in thrush.

Under this second microscope (Fig. 99, page 788) you will see some shreds from the ame specimen, but much more highly magnified.

In this specimen you can see the formation of the spores in the mycelium.

Under this same heading of stomatitis mycetogenetica I shall merely efer to those pseudo-membranous conditions which occur in diphtheria, tuberculosis, syphilis, and diseases of a like class. The former two are so rarely seen in the mucous membrane of the mouth that it is not necessary to describe them. The lesions which occur in the mouth in syphilis I have already described when speaking of that disease (page 494).

CASE 891. FIG. 99.



Thrush showing the formation of spores in the mycelium. (Zeiss Oc. 3, homogen. immer. 2.0 mm.)

STOMATITIS GANGRÆNOSA (Noma, Cancrum Oris).—Stomatitis gangrænosa is the rarest and most fatal form of stomatitis which occurs in children. It is usually met with between the ages of three and seven years. It is a disease characterized by a gangrenous process which begins on the gums or on the inner surface of the cheek and spreads with great rapidity to the adjoining tissues, all of which can be involved and quickly destroyed.

ETIOLOGY.—It is probable that there is a specific germ which causes this disease. This organism has, however, not yet been determined. It is supposed that it does not attack a healthy mucous membrane, and that one of the other forms of stomatitis, especially stomatitis catarrhalis, and in some cases stomatitis ulcerosa, precedes it. Furthermore, stomatitis gangrænosa seldom attacks healthy children, but usually affects those who have other diseases and are greatly debilitated. It occurs most commonly secondarily to the acute exanthemata, especially measles. The disease is also said to result from the administration of mercury in too large doses.

It begins as a reddened, hard spot in the mucous membrane, usually of the cheek. This soon becomes gangrenous and extends rapidly through the entire thickness of the cheek, producing perforation. It may also

end laterally in all directions, attacking the bone as well as the other
ues.

SYMPTOMS.—The first symptom which is apt to be noticed is the gan-
nous odor which comes from the mouth. On examination an ulcer will
found which tends to spread rapidly. The cheek becomes much swollen,
ard and œdematous, the œdema especially affecting the tissues under the
. The gangrenous process extends very rapidly, at times destroying large
tions of the face, and also involving the bones, which become denuded.
e teeth become loose and fall out. The odor from the gangrenous tissue
excessive. The flow of saliva is very much increased. The degree of
fering which the children undergo varies very much: sometimes it seems
if they suffered no pain whatever. The temperature varies, at times
ng raised and again being subnormal. The pulse is weak and rapid.
e appetite is diminished, and the children are likely to have diarrhœa,
bably due to the infectious nature of the products of the mouth which
swallowed. Hemorrhages are rather rare, according to Forchheimer, as
blood-vessels are usually filled with thrombi. Secondary affections,
h as catarrhal pneumonia from the inhalation of septic material, are
uncommon. The child may die from one of these secondary affec-
ns, or it may become more and more weakened by the local condition,
l unless the morbid process is arrested it will die eventually from ex-
stion.

DIAGNOSIS.—The diagnosis of this disease, except in its earlier stages,
ot difficult. At times, however, a local ulcerative process produced by
ecayed tooth may simulate closely stomatitis gangrænosa. In these cases
diagnosis is made more difficult by the fact that the tissues of the cheek
y become hard and look as though perforation might take place. Co-
idently with this condition the ulceration of the gum and often of the
cous membrane of the cheek, with the foul odor which emanates from
makes the similarity of the two diseases very striking. In simple ulcer-
on from a tooth, however, active local treatment with solutions of myrrh
of soda combined with frequent washing of the mouth with sterilized
ter is soon followed by marked improvement, while where stomatitis
grænosa is present the morbid process continues to extend.

PROGNOSIS.—The prognosis in cases of stomatitis gangrænosa where
y are untreated is almost universally fatal. Cases have been known,
vever, where a line of demarcation has formed around the gangrenous
t, granulations have arisen, and cicatrization has followed, leaving exten-
scars. If the disease is treated by extirpation of the diseased structure
the very beginning, the prognosis becomes more favorable. Where the
ease has perforated the cheek and the gangrenous process has become
ensive, the child is seldom relieved even by surgical treatment.

TREATMENT.—Care should be taken when a child is affected with a
ease of an exhausting nature that its mouth is kept thoroughly cleansed,
we can never tell when or in what individual the mucous membrane

may become vulnerable to the organism which produces stomatitis gangrænosa. In stomatitis gangrænosa it is very important for the success of the treatment that it should be begun very early in the disease. Where the diagnosis has been definitively made, it is wiser not to temporize with applications of nitrate of silver and other drugs, but at once to place the case in the hands of a surgeon and have the entire area of the invaded tissues excised. It is also well after the gangrenous process has been removed by the knife to destroy an area of healthy tissue by means of the Paquelin thermo-cautery or by the galvano-cautery. There should be no delay in operating upon these cases, as great destruction of the tissues may take place in even a few hours.

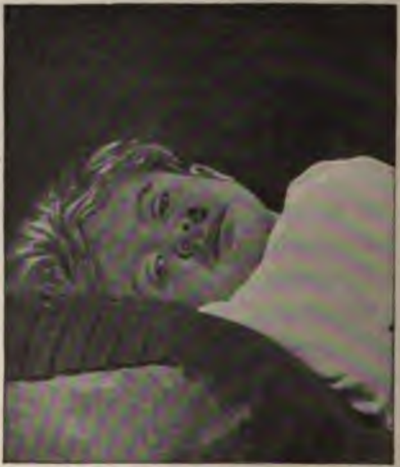
After the operation the tissues should be inspected frequently, to see whether there is any return of the gangrenous spots, and, if found, these spots should be removed immediately. As the disease is very apt to return, plastic operations to obviate deformity should not be undertaken very early after the operation.

In treating these cases surgically it must be remembered that the child is in a very debilitated condition, and that if it is suffering from any especial disease treatment directed to that disease is indicated, also that stimulants are required to prevent the already weakened child from dying of exhaustion following the operation.

Here is a little girl (Case 392), four years old, who has been brought to the hospital to be operated on for stomatitis gangrænosa.

CASE 392.

I.

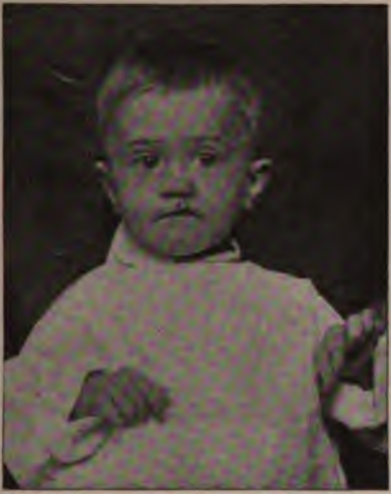


Stomatitis gangrænosa, left cheek (before operation). Female, 4 years old.

In this case the disease was apparently primary, and began on the the left side of the mucous membrane of the mouth. It spread rapidly, and, although treated by local applications to the mouth with various solutions, has now, as you see, broken through the left cheek close to the ala nasi. The teeth are loose in the middle of the upper jaw, and there amount of alveolar necrosis. There is a strong gangrenous odor from the mouth sues of the cheek, and a considerable flow of saliva. The child's general condi-, but she is becoming more debilitated, has lost her appetite, and has a slightly perature. The operation should be performed immediately.

CASE 392.

II.

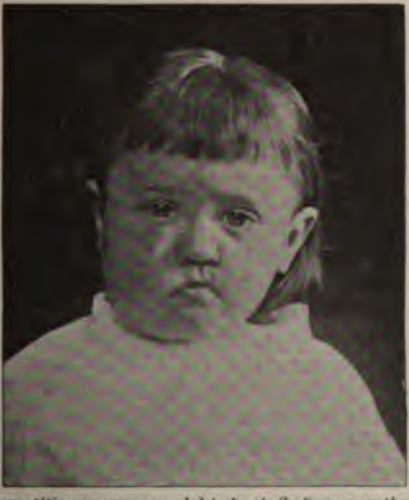


Stomatitis gangrænosa, left cheek (after operation).

equent history.) The cheek was operated on the day after the child entered the y Dr. H. W. Cushing. The wound healed readily, and this picture (II.), taken ths afterwards, shows the scar on the cheek close to the ala nasi and also on the

CASE 392.

III.



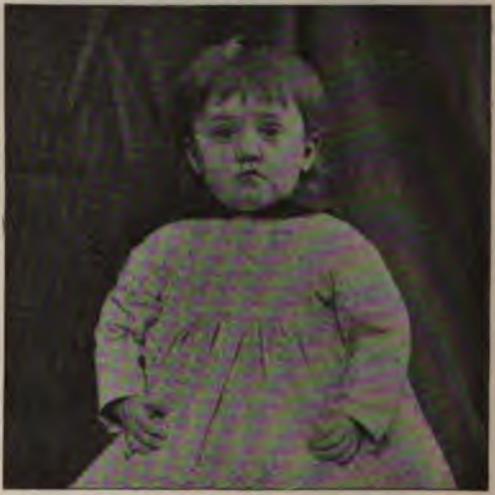
Stomatitis gangrænosa, right cheek (before operation).

ear later the child again returned to the hospital, and on examination was found the appearances which are seen in this picture (III.), taken at that time.

The right cheek was much swollen and indurated, especially under the right eye. The periosteum of the lower jaw on the right side was found to be affected, and the necrotic process had undermined the whole cheek as far as the orbit. The child was operated on by Dr. Bradford without any external opening of the cheek. The wound healed, and the child was discharged from the hospital, but returned some months later with a spontaneous opening on the right cheek. This was again apparently cured by operation. Two months later the child was found to have in the lower jaw a process similar to that which had occurred in the upper jaw. Her health was poor, she was pale and weak and had loss of appetite. She was operated upon again, and a sequestrum was removed from the lower jaw. She then improved, and this picture (IV.) was taken some months later, when she was apparently in fair health.

Case 392.

IV.



Stomatitis gangrænosa, right cheek (after operation).

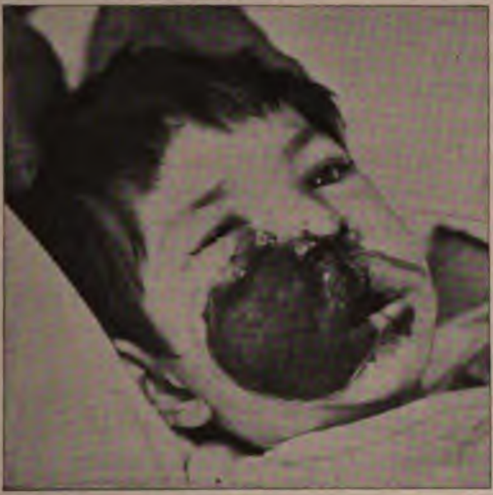
The microscopic examination of the gangrenous tissues removed at the operation presented nothing significant of any especial disease, and a culture made by Dr. Stone showed only a few streptococci.

You will remember the case of measles (Case 257, page 587) complicated by stomatitis gangrænosa which I showed you at a previous lecture, and the result of which I now report to you.

As I told you at that time, the disease was preceded by pertussis, measles, and a broncho-pneumonia. After she had the pneumonia for seventeen days her right cheek began to swell and a bad odor to come from her mouth, but nothing especial could be found in the mucous membrane of the buccal cavity. Four days later the swelling of the cheek had much increased, and there was œdema of the lips and eyelid so that the right eye was partly closed. The swelling was semi-fluctuating. The temperature varied from 38.3° to 39.4° C. (101° to 103° F.), and the cough had much lessened. On the following day a bluish-black spot about 1.5 cm. ($\frac{5}{8}$ inch) in circumference appeared at the right corner of the mouth, and this rapidly increased during the day. Two days later the dark-colored area had increased considerably in size and presented a circular outline with a clearly marked line of demarcation.

The child also had a profuse greenish diarrhœa. On the following day the dark area rapidly extended, and soon involved the whole of the right cheek, the right side of the mouth, and the right nostril. There was no external loss of tissue. The child was extremely emaciated, and from the beginning of the attack was in a hopeless condition, so dical treatment of the disease was deemed inadvisable. It died suddenly on the ng day.

CASE 257.



Stomatitis gangrænosa secondary to measles and pneumonia. Female, 5 years old.

OSSITIS.—Glossitis is so rare a disease in children that the pos- of its occurrence only need be mentioned. In this affection there is ute inflammation of the tissues of the tongue, accompanied by fever, ement of the organ, and considerable pain. There is usually a hyper- on of saliva, and at times the obstruction of respiration from the ion of the throat by the greatly enlarged tongue produces somewhat ing symptoms, though, as a rule, not serious ones.

is disease may be caused by direct injury to the tongue from corro- bstances, by heat, or by the stings of animals, and sometimes probably sis. It runs a variable course; it is not especially serious, and tends over after a few days. The treatment is purely symptomatic. The nt local application of ice and of ice-cold alkaline solutions to the and mouth is indicated.

condition of the dorsum of the tongue is sometimes met with which nt of a better name is called *lingua geographica*, "mappy tongue," or lering rash." One or more small patches appear on the dorsum or the tongue, which in a few days may spread and coalesce, covering a large portion of the surface. They diminish in size or fade with rapidity, to recur at variable periods. The patches are red and h, and the filiform papillæ are absent. The rest of the tongue appears l, except that the papillæ on the borders of the denuded portions are and prominent. The etiology of the disease is unknown. It occurs exclusively in children or in young adults who have been subject to n childhood. It is very benign, and gives no discomfort to the child. incipal importance lies in the fact that it is sometimes mistaken for a om of some more serious disease. No form of treatment has been found useful. It recurs periodically for months or years, but does not tend to increase in severity nor to lead to other diseases.

MICROGLOSSIA.—In some individuals an arrest of development of the tongue produces the condition called microglossia, in which the tongue is to a varying degree smaller than normal.

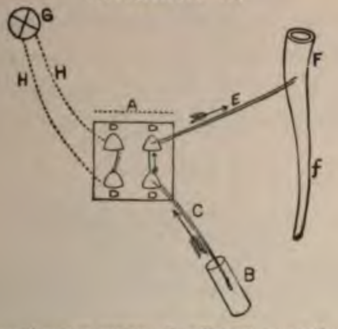
MACROGLOSSIA.—The opposite condition, macroglossia, in which the tongue is enlarged, is more common than microglossia. It is usually a congenital lesion, and is especially marked in cretins. The prominent feature of the affection is a prolapse of the tongue, which is often enormously enlarged in every direction, is usually of a deep violet color, and is covered with a thick, whitish coat. The protruded tongue is indented and even ulcerated by the teeth, which are often pushed forward and become carious. The saliva flows continuously from the mouth, the lower lip becomes thick and ulcerated, and the forcing forward of the lip, larynx, and velum palati by the weight of the tongue renders suction, mastication, and deglutition difficult. The nutrition of the child is thus much interfered with, and this interference is one of the most serious results of the disease. This condition is not a glossitis, but a deformity which seems to be associated with certain other malformations of the body. In these individuals the hands and feet are apt to be large, thick, and purplish.

Macroglossia appears in two forms. One is the fibrinous, in which the connective tissue is pathologically increased between the muscular fibres. The other is a cavernous cystoid degeneration of the interstitial connective tissue, by which the resulting spaces come into connection with the lymph-vessels, constituting a condition closely resembling cavernous angioma, from which it receives its name of lymphangioma cavernosum.

The disease seldom tends to recover, and the treatment is to give as much relief as possible to the great discomfort which arises from it, by cleansing the mouth frequently with alkaline solutions. Especial care should be directed to the nourishment of the child. In extreme cases surgical interference is indicated where the child's respiration and general nutrition are affected, and in some cases great improvement is accomplished by the removal of part of the tongue.

DIFFICULT DENTITION.—I have already described to you the process of the normal development of the teeth in infancy and childhood, and have impressed upon you that this process is a physiological one. The teeth are developed at birth to a certain degree, and merely increase in size during infancy until they pierce the gums and assume their places in the mouth. In many cases the process of dentition gives rise to no morbid conditions whatever. The idea that dentition occasions the various diseases with which it was formerly supposed to be associated is an erroneous one. From the fourth or fifth month, however, until the completion of dentition in the latter part of infancy, various nervous disturbances are so closely associated with irritation in the mouth that in this sense dentition may be considered responsible for many of the slight ailh arise at this period of life. The mouth at this time frequently ot, and sometimes dry, although there may be a hypersecretion There is evidently much discomfort in the region of the gums, nt is continually rubbing them with its fingers and seems to get biting on hard substances. Such infants may become much and may lose their appetite, and thus their nutrition may be with, without any discoverable cause for these abnormal condind the general nervous irritation which arises from the feeling ort in the mouth and head. In the more extreme cases the be so restless at night that it scarcely lies still for half an hour and may spend night after night crying out occasionally as pain, and knocking its head against the sides of its crib, so that in the crib will have to be padded. These infants also have to be metimes from knocking their heads against the floor or wall, as to become almost frantic from the continued irritation from which uffering. These symptoms occur with such regularity at a time th is in its final stage of development, and cease so uniformly ooth has attained its growth, that the causal relation between the these nervous symptoms seems more than probable. This rather clinical association of dentition and nervous symptoms is, howlly explained by the analogous symptoms arising from the anaationship which exists between the roots of the teeth and the ear. been noticed that in certain individuals during the completion

DIAGRAM 10.



ganglion ; B, sensori-motor nerve ; C, afferent sympathetic fibres from sheath of B ; D, ls ; E, efferent sympathetic fibres proceeding to artery *f* ; F, artery dilated ; *f*, normal size eyond the sympathetic influence ; G, general vaso-motor centre ; H, H, the dotted lines inle course of the fibres forming the roots of the ganglion in the spinal cord to the general centre G. (Woakes.)

elopment of a tooth symptoms connected with the ear will manielves. These symptoms are usually produced by congestion of vessels of the ear, which is accompanied by pain, and sometimes nflammation. They are evidently of reflex origin. If you will diagram (Diagram 10) you will understand the influences which n of some distant part of the economy may exert on the bloodthe ear.

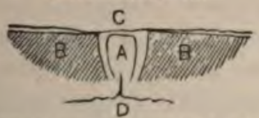
onclusion that it is never necessary to lance the gums. In cases of difficult dentition, however, as I have just explained, irritation arises very commonly in the later stages of the development of a tooth, and the question, herefore, remains whether this irritation in various parts of the economy, notably in the ear, can be relieved by lancing the gum. With regard to the question of gum-lancing, it may be said that it should be resorted to only under very exceptional circumstances.

During the dental period two classes of irritation are met with in connection with the teeth: (1) irritation of the dental nerves, with symptoms of reflex aural disturbance; and (2) irritation of the gum over the crown of the tooth from pressure, with symptoms of local irritation. We here have two entirely different conditions. If, where pain or symptoms in some other part of the economy seem to arise from dental irritation, we find that the gum which covers the crown of the still undeveloped tooth is soft and flat as in other parts of the mouth where a tooth is not about to come through, lancing the gums is manifestly absurd, as there is evidently no reason for making a wound in the mouth.

The second class of cases, however, though exceedingly rare, must still be recognized as distinct in themselves and requiring especial treatment. In this class it is very evident that the gum for some reason does not give way to the growth of the tooth. Where the gum covers the crown of the tooth the tissues are swollen, tense, almost cartilaginous in their feeling, and hot. As in like conditions, either in the mouth or elsewhere, when this combination of abnormal conditions is found over the crown of the tooth, it can be relieved at once by the lancet.

I have here two diagrams which represent the condition of the gums in relation to the teeth in the two classes of cases which I have just mentioned. In this first diagram (Diagram 12) you see that the mucous membrane over the crown of the tooth is flat and on a level with the rest of the gum.

DIAGRAM 12.



A, tooth in bone socket; B, jaw; C, gum, soft, not inflamed or swollen; D, dental nerve.

This is the condition of the gum in the majority of cases of difficult dentition, yet very severe symptoms of disturbance of the ear and cerebral circulation may apparently arise in these cases. The symptoms, of course, are very varied, the most definite ones being connected with the ear. In this class of cases the gum should never be lanced, even for the purpose of bleeding, as the mouth is not a fit place for such a procedure. The treatment of these cases should be directed to the especial part of the economy from which the symptoms arise. For instance, if the ear is affected, the indication is to relieve the reflex congestion. This can be done by the instillation into the ear of a few drops of an atropine solution (Prescription 75).

Although much has been said about the danger of hemorrhage in these cases, and of infection of the wound by pathogenic organisms, yet instances where such results have occurred are so exceedingly rare that they should not deter us from treating the case properly as we would treat an abscess in the mouth, tonsil, or pharynx. It has also been said that a cicatrix may form on the gum over the crown of the tooth as a result of lancing. This is an exceedingly rare occurrence, and need scarcely be taken into account. The probability is, where such an instance has occurred, that the case was not one in which the gum should have been lanced, and the fear of such a result as this should certainly not weigh in the balance against the possible exhaustion and acute pain which may continue for days unless relief is given by cutting.

I have a number of cases to show you which will serve to illustrate what I have endeavored to impress upon you in speaking of *difficult dentition*,—namely, that the indications for lancing the gums very seldom arise.

This infant (Case 393), ten months old, has been brought to the clinic with the following history:

It has one lower incisor. At the time when this tooth was about to appear above the margin of the gum the infant was very restless, and had considerable fever, and pain in its ear. Somewhat later a muco-purulent discharge came from the ear, but the general symptoms of restlessness, pain at times, and the local symptoms of heat and irritation in the mouth continued until just before the tooth had pierced the gum. After that time, which was three weeks ago, the discharge from the ear ceased, and the infant became perfectly well, the local irritation also having disappeared.

During the last three or four days, however, the same symptoms have returned. The infant is evidently suffering from irritation in its mouth. Sometimes the gums are hot and dry, and again there is a hypersecretion of saliva. It continually puts its finger to the gum of the lower jaw, sometimes almost locating it near the place where the first tooth has been cut. The ear has begun to discharge again, and the infant shows signs of general discomfort by rubbing its nose and head continuously and at times crying out as though in pain.

On examining the gum you see that it is not swollen, and that there is no especially tender point. On examining the ears an old perforation of the membrana tympani is found in the right ear, which is discharging, while in the left ear there is a simple congestion.

Such cases as this are often treated by lancing the gum, yet this procedure is not of the slightest use,—is, in fact, contra-indicated, as it will only increase the already existing irritation of the mouth. The treatment is the internal administration of bromide of potassium and appropriate local treatment for the ear.

The other cases are so similar and are so commonly met with that I need not dwell upon them, but shall report one of the rare cases in which lancing of the gum is indicated.

An infant (Case 394), eight months old, and in good health, cut its first tooth when it was seven months old. At this time there were no nervous disturbances, the tooth coming through the gum without any reflex or local symptoms whatever.

When the second tooth was pressing on the gum I was called to relieve the following symptoms. The infant, who had been perfectly well, and who on examination showed no disease of any organ, was reported to have been feverish, restless, and crying out with pain for the previous twenty-four hours. It had refused to nurse, had not slept for thirty-six hours, had vomited a number of times, and was found to have a temperature of 40° C.

(104° F.). It twitched from time to time, and apparently was in danger of having general convulsions. On examining the mouth I found that one of the lower middle incisors was entirely through the gum. The gum next to this incisor was greatly swollen, tense, cartilaginous in feeling, hot, and tender, so that whenever it was touched the infant screamed with pain. I then lanced the gum. The expression of pain, which had been most marked on the infant's face, disappeared immediately, and was replaced by an expression of perfect tranquillity, and it was evident that the severe pain had been relieved instantaneously. The infant went to sleep at once, and slept two hours. When it awoke its temperature was normal, it took the breast with great eagerness, and from that time it had no more trouble in its mouth. All the rest of its teeth were cut without any abnormal symptoms.

I have also to report to you another instance which illustrates to a still greater extent the necessity of lancing the gums in certain cases.

An infant (Case 895) began to have irritation from its teeth when it was five months old. At this time it woke up in the night screaming, and continued to scream with pain for several hours, during which time its parents had to walk continually up and down the room with it. Various remedies were administered, but without the slightest relief, and finally, after two days of suffering, in which it refused to take its nourishment, it lost in weight, and seemed very ill. An incision was made over the hot and swollen gum, with immediate relief.

The same symptoms occurred when the next tooth appeared beneath the surface of the gum, but were relieved, after waiting for a few hours, by lancing. Of the remaining eighteen teeth, six or eight gave rise to similar symptoms, but in every instance immediate relief was afforded by the lancing of the gum.

LECTURE XLI.

DISEASES OF THE NOSE, NASO-PHARYNX, AND PHARYNX.

NOSE.—The nose is the normal passage for the entrance of air to the lungs, and it is principally here that the air is modified before entering them. In normal respiration the mucous membrane of the nasal cavities, on account of the peculiar shape of the turbinated bones, presents a large surface to the inspired air, and is therefore admirably adapted to filter it of particles of dust and micro-organisms. The air is also warmed and changed so that before it reaches the larynx it is saturated with moisture and heated to a temperature of 35° C. (95° F.). This modification of the air is especially important in the new-born, since the lung has so lately been brought into use and is in such a comparatively undeveloped condition that it cannot withstand unchanged air, to which it adapts itself better later in life. I have already described to you (page 33) the extremely narrow passage through which the air passes in going to and through the naso-pharynx in young infants, and how easily this passage can become occluded. There are not many diseases which occur in the nose in infants and young children, and those which we find are serious chiefly by being the cause of occlusion. In case of mouth-breathing due to nasal occlusion in an infant, the air which has not been modified by passing through the nose and naso-pharynx may have a detrimental influence on the lung and general circulation, thus striking a serious blow at the infant's vitality. In later childhood, although the occlusion which arises in the nares may not be so serious as regards the life of the patient, yet you will see the results of such a condition represented by retarded development of the child and interference with the function of hearing, with its resulting mental dulness.

The most common pathological condition which occurs in the nose in infancy and childhood is some form of *rhinitis*. This may be acute or chronic, catarrhal or purulent, hypertrophic or atrophic. New growths are rare. Of these the more common is myxoma or simple mucous polypus. Bleeding from the nose, called epistaxis, may arise from an ordinary non-inflammatory condition, and is generally due to the breaking of a superficial vessel on the septum.

ACUTE RHINITIS (Acute Coryza).—Acute rhinitis is an inflammation of the mucous membrane of the nasal cavities. The cause of the disease in most cases is apparently undue exposure to cold, though it may be proved eventually that this exposure merely prepares the way for the attack of some micro-organism. This condition may in almost all cases be considered as part of a disease which affects the mucous membrane of the naso-pharynx and pharynx as well as the nares.

The symptoms are a sense of fulness, burning, and dryness in the nostrils, succeeded in a few hours by a serous discharge, which later becomes muco-purulent. There is usually a slight rise of temperature, and, although the general symptoms are often slight, there is commonly a very evident sense of discomfort, along with loss of appetite and general malaise. In some cases, by direct extension of the inflammation through the Eustachian tubes, an otitis media may be caused. Especially in young infants, the entrance of air into the naso-pharynx is blocked by the swelling of the erectile tissues covering the turbinates, and almost complete occlusion takes place. The patient is then forced to breathe with the mouth open, and a resulting condition of dryness of the mucous membrane of the mouth and throat and a choking sensation arising from it follow. The natural tendency of an infant or young child is to keep the mouth shut, so that often when the nose is occluded it breathes with great difficulty when asleep, and its face becomes congested and even cyanotic. On forcing the mouth open the symptoms of congestion and cyanosis disappear, and the child begins to snore, and breathes with comparative comfort so long as its mouth remains open, until the dryness of the throat wakes it up.

The prognosis in these cases of acute rhinitis is usually good. The disease runs its course in a variable period of from three days to a week, and, unless the child is subjected to fresh exposure, it recovers entirely. The prognosis, however, as I have already stated, varies in accordance with the age of the individual attacked. The danger that a young debilitated infant may die from exhaustion where the nares are occluded is considerable. You will remember the case which I described to you in a former lecture (page 34), where a puny, ill-cared-for infant died of a simple acute rhinitis. Instances of this kind should warn us that active treatment is indicated.

The treatment should be directed primarily to relieving the nasal occlusion. This is best accomplished by atomizing the nose. In most cases the oil atomizer containing oleum petrolatum album is sufficient to afford relief. In addition to the local treatment, the administration of stimulants where there is exhaustion is indicated. You should also be sure that the infant is taking a sufficient amount of nourishment. This is especially difficult to determine if it is nursing, as under these circumstances it will often hold the nipple in its mouth and apparently suck, while its breathing is so much disturbed by the nasal obstruction that it does not draw much milk from the breast. The various drugs which have been recommended for acute rhinitis have not in my hands proved to be of much use. I have occasionally found that a few drops of the tincture of euphrasia repeated three or four times, at intervals of an hour will seemingly lessen the nasal secretion.

As an instance of this class of cases I shall report to you the case of an infant (Case 396) who had an attack of acute rhinitis when she was four months old. Although she was well nourished and fairly strong, yet the occlusion of the nares, which took place apidly, produced serious symptoms. She was somewhat cyanotic, refused to take her ood, which had to be forced down her throat, and was sleepless, while her strength failed apidly. She was cared for by a trained nurse night and day, the oil spray was used at requent intervals, and stimulants were given, with the inhalation of oxygen once every hree or four hours. Under this treatment she improved slowly and recovered entirely.

In older children the serious symptoms which I have described do not ccur, as a rule, and the disease is not much more significant than the coryza f the adult.

PURULENT RHINITIS.—A rather rare form of rhinitis is at times met vith in which there have been a number of acute attacks and the process as become somewhat chronic. In these cases the discharge is essentially urulent, and the name purulent rhinitis has therefore been adopted.

This form of rhinitis is not accompanied by any especial enlargement of he turbinated bodies, and narrowing of the nasal passages is not a promi-ent symptom. The symptoms are chiefly a purulent discharge from the ostrils, and redness and excoriation produced by the acrid character of the lischarge.

The prognosis of purulent rhinitis is good, except in extremely debili-ated children.

The treatment is the same as in the catarrhal form, especial attention eing paid to cleansing the nose with alkaline solutions and thus alleviating he irritation produced by the discharge.

I have a case here in the ward which illustrates the purulent form of hinitis.

This boy (Case 397) is two and one-half years old. So far as we can ascertain, there has een no especial disease in his parents which would be significant in connection with the resent condition of his nose. He is said to have been sick for four weeks. The attack egan with fever and general discomfort in connection with the nose. Somewhat later a ischarge began to come from the anterior nares and also from the right ear. Up to the ime of this attack he had always been healthy and well developed, and is said to have een bright and to have talked as well as is usual for children of this age. During the last wo weeks he has grown worse. There has been an increased discharge from the nares. Ie has become rather dull and apathetic, has lost his appetite, and has stopped speaking. he child lies in bed, or at times gets restless and sits up; his face has a dull expression; e will not speak, and he shows considerable hebetude. There is very little discharge om the ear, but a profuse purulent discharge from both nares. The discharge is evi-ently irritating, as the upper lip has become excoriated and swollen. He has now had e disease for five weeks. I have detected nothing abnormal in any of the organs except e nose. An examination made yesterday by Dr. Coolidge, one week after the child ntered the hospital, showed that there were no adenoid growths or foreign bodies in the ose or naso-pharynx. The pharynx was somewhat congested, but showed no especial athological condition, and the tonsils were not enlarged. The temperature has varied om 36.6° to 38° C. (98° to 100.5° F.). No cause has been discovered for the attack.

(Subsequent history.) The child, under simple treatment directed to washing out the ose with warm alkaline solutions, and with especial attention to a nourishing diet, nproved gradually, and three months from the beginning of the attack was discharged om the hospital cured. The hebetude passed away; he talked as well as ever; he had a ood appetite, and a normal temperature; the bowels were regular, and the ears, nose, naso-harynx, and pharynx were in a normal condition.

epithelial lining of the muciparous and follicular glands. The glandular function is thus impaired, and the muco-purulent discharge becomes thick and firmly adherent in the form of crusts to the sinuosities of the nose. This film of desiccated muco-pus in drying contracts the underlying turbinated tissues in such a way as to interfere with the circulation of the blood, a condition which limits glandular action still more and conduces to general atrophy.

The symptoms of atrophic rhinitis are the formation of crusts and the presence of fetor.

Although the tissues which have actually been destroyed by the atrophic process cannot be restored by treatment, the patient can be entirely relieved of the crust formation and fetor by persistent and patient local washing and applications. The details of treatment differ according to the extent and character of the disease. Crusts may be removed by spraying or douching, great care being taken to prevent the washing fluid from entering the Eustachian tubes. If this is not sufficient to remove the crusts, the nasal cavities must be illuminated with a head-mirror, and the crusts carefully brushed off with a cotton-stick. The formation of dry, hard crusts is often prevented by frequent spraying with an oil. Local applications of different substances are of use in many cases, but these should, as a rule, be carried out under the direction of a specialist in the treatment of diseases of the nose.

MUCOUS POLYPUS.—This is a pedunculated connective-tissue growth originating from the mucous membrane of the middle turbinate bone. It is rare in children. It does not grow on a healthy mucous membrane, and is always preceded by some morbid condition of the nose. It is often multiple.

The symptoms begin with a nasal discharge followed by nasal occlusion. The diagnosis is easily made by a mirror and a probe. The treatment is the removal of the growth.

EPISTAXIS (Hemorrhage from the Nose).—During the period of early childhood hemorrhage from the nose is not uncommon. I have occasionally met with epistaxis in young infants, but in my experience it is rare in the early months of life. In older children recurrent epistaxis, especially if unilateral, points to the presence of an erosion or a varicose condition of the veins in the cartilaginous septum near the external opening of the nose.

Unless the individual happens to be affected by hæmophilia, epistaxis is not especially dangerous, and usually its occurrence ceases as the child grows older.

The application of pressure on the side of the base of the nose and the use of ice are usually sufficient to stop the hemorrhage. If the epistaxis is due to the varicose condition just spoken of, it can be readily controlled temporarily by a plug of cotton pressed upon the bleeding part. For a permanent cure, cauterizing the bleeding part may be necessary.

NASO-PHARYNX.—I have described in a previous lecture (page 33) the anatomy of the naso-pharynx. Although this cavity is small and simply by the appearance of the child, as there is no other disease which especially simulates this condition. A definite diagnosis, however, can be made only after the hypertrophied tonsil has actually been seen or felt.

I would impress upon you the great importance of learning to detect by means of the finger the presence of an enlarged pharyngeal tonsil. This acquirement is necessary, not only for the purpose of diagnosticating the presence of this disease, but also in order to determine correctly the cause of many other abnormal conditions. The examination with the mirror in the throat is usually so difficult in young children that the direct detection by means of the finger is often the most applicable means to employ in these cases. The child should have a blanket pinned around it tightly, so as to keep it from moving its arms. It should be held firmly sitting in the nurse's lap. You can then hold the child's head with one arm, pressing the cheek between the back teeth with the forefinger, then pass the forefinger of the other hand gently, firmly, and quickly over the base of the tongue and behind the soft palate until it reaches the posterior wall of the pharynx. Then, quickly turning the finger upward, you can easily feel whether the cavity of the naso-pharynx is clear or whether it is more or less filled by a soft, spongy mass, the hypertrophied pharyngeal tonsil. There is usually a little blood on the finger when it is withdrawn, as the growth is friable and bleeds easily. This examination is not, as a rule, very painful to the child, but produces a certain amount of discomfort from a choking sensation. When the finger is once in the mouth, it is not wise to take it out again until you have completed your examination, as the child can rarely be induced to allow you to make a second examination. In passing the finger over the base of the tongue you must be careful to get the finger behind the soft palate, and not to push it upward and backward, for in this case the soft tissues of the palate may feel like an adenoid growth. The child can be prevented from biting the finger by simply keeping the cheek pressed between the teeth as I have just described.

Lack of development of the chest with flattening of the front of the thorax may be caused, not, as was formerly supposed, by the enlargement of the faucial tonsils, but by the occlusion caused by the hypertrophy of the pharyngeal tonsil. This hypertrophy with its resulting nasal occlusion may also be the cause of pharyngitis, laryngitis, and perhaps of bronchial catarrh or asthma, which can be cured only by the removal of the primary cause, the pharyngeal tonsil.

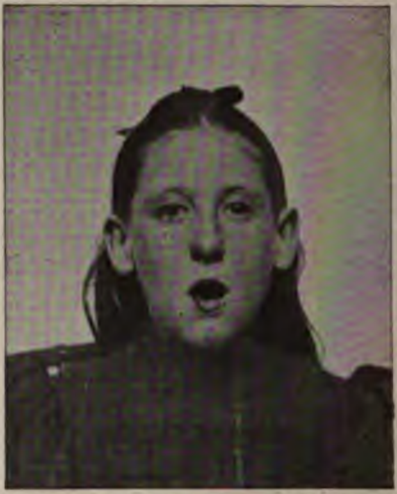
In order to impress upon you the chief points in the diagnosis of these cases I show you this little girl.

She (Case 398) is ten years old, and presents a typical picture of this disease.

You see that her mouth is held open and that she evidently has complete occlusion of the posterior nares, the anterior nares on examination being found entirely free. You will notice the pinched look of the face on either side of the nose and the prominence of the bridge of the nose. The child is dull, the dulness having increased as the other symptoms of the adenoid growth have developed, and her face now has a stupid expression. When you have once seen and studied a case of this kind, you will have no difficulty in making a diagnosis by simple inspection. On examining the child's mouth you see that the palate is very much arched, that the tonsils are enlarged, that the soft palate is slightly pushed forward, and that the pharynx is narrower than normal.

(Subsequent history.) After removal of the adenoid growths and faucial tonsils by Dr. Coolidge she found no difficulty in keeping the mouth closed, not only while awake, but also at night, and slept much more quietly than before. Her general health improved, and the development of her face during the remainder of its growth will undoubtedly be normal.

CASE 398.



Hypertrophy of pharyngeal tonsil (adenoid growths). Female, 10 years old.

PROGNOSIS.—The prognosis of cases of hypertrophy of the pharyngeal tonsil varies greatly, for there are all forms and degrees of the affection. In some cases the swelling of the lymph-tissues occurs only at intervals when the child has been subjected to exposure in inclement weather; it will then show itself simply by occlusion of the nares, with the resulting discomfort, lasting for some weeks, but disappearing eventually as the weather becomes milder or if the child is taken to a different climate. In most cases, however, where the affection is at all pronounced it becomes chronic, and the symptoms usually increase in severity up to about the time of puberty. You must remember that the naso-pharynx has an important function besides being a passage-way for the air. It lubricates the pharynx, and by the action of its muscles opens the Eustachian tubes during the acts of swallowing and yawning, thus ventilating the ear. You will see, therefore, that the prognosis must vary according to the degree in which any of these functions are interfered with. Where the children become deaf they may gradually lose the power of speech. Again, from being deaf they may fall into a condition of hebetude which sometimes closely simulates idiocy, though it is not true idiocy, for the mental condition quickly changes when the cause of the disturbance has been removed. Where the disease is diagnosticated in its early stages, or later, unless irreparable injury has been lone to the ear or the general development, the prognosis is very favorable, provided the proper treatment is carried out.

TREATMENT.—The best treatment of these cases is to remove the adenoid growth at once. The operation in the hands of a skilful surgeon s not dangerous, and should be unhesitatingly advised. There are a number of methods which have been employed in operating on these cases. The child should be thoroughly etherized. Some operators prefer to have the child held sitting in the lap of an attendant, others to have it lying down with its head bent backward. The soft palate is drawn forward by means of a palate-hook held in the left hand. A pair of post-nasal forceps held n the right hand is introduced, closed, into the naso-pharyngeal cavity. The blades are then opened, and pieces of the mass are grasped one after the other and pulled off gently: under no circumstances is force to be exerted. With proper care and assistance there is no danger to the child, and often in ten or twenty minutes a morbid condition which has existed for years may be practically cured. There are, of course, many details in this operation which must be thoroughly understood in order that it should be successful. These details, however, need scarcely be mentioned here, as the operation should be performed only by one whose work has especially adapted him for it.

These growths when not extensive are sometimes removed even without ether with the curette or the finger-nail.

I have here a little boy who illustrates the benefit of operative treatment for the removal of the pharyngeal tonsil when hypertrophied.

He (Case 399) is four years old. You see that he has a very bright expression, and he peaks well; he shows nothing abnormal in connection with the shape of his nose or face. He hears well, he sleeps with his mouth shut, and has a free passage of air through a perfectly normal nose and naso-pharynx. When he was three and a half years old it was noticed hat he snored at night, breathed with his mouth open, and was subject to continual attacks f rhinitis and naso-pharyngeal occlusion. Following these attacks his hearing became ffected, and, while in his second year he had been bright and always ready to play with his arents, he became dull, and did not care to play with others, but would sit for hours playng by himself with his toys.

A digital examination showed a mass of considerable size blocking the posterior nares. On the removal of this mass, which proved to be an hypertrophied pharyngeal tonsil, rapid mprovement took place in his general condition, the dulness and hebetude disappeared, nd within the last month he has returned to the normal condition which he represented in is second year.

There are other growths which occur in the naso-pharynx, such as arcomata. They are, however, too rare to need especial description.

PHARYNX.—Diseases of the pharynx in children are especially those ffecting the tonsils, the uvula, the soft palate, and the posterior wall of the pharynx.

TONSILLITIS.—By tonsillitis is meant an inflammation of the tissues of he tonsil. This inflammation may be acute or chronic.

Acute Tonsillitis.—Acute tonsillitis may be simply an inflammatory condition represented by enlargement and reddening, *simple tonsillitis*, or the inflammation may be especially located in the crypts of the tonsil, in which case it is commonly designated *follicular tonsillitis.*

The other affections of the tonsils, such as occur in the course of the exanthemata and in diphtheria, constituting the pseudo-membranous form of the disease, can best be described in connection with the especial diseases in which they arise, and I shall therefore speak only of the two forms to which I have just alluded.

Acute Simple Tonsillitis.—It is probable that the cause of the acute simple form of tonsillitis is a microbe. The child is usually attacked suddenly, with a heightened temperature, 38.8° to 39.4° C. (102° to 103° F.), fever, restlessness, and sometimes vomiting and loss of appetite. Young children do not complain of the throat so much where the tonsils are affected as do older children and adults. In fact, in many cases, unless the throat is actually inspected, it would seem as though it were not a local affection of the throat, but some general disease affecting other parts of the system. You should be especially on your guard, therefore, not to have your attention diverted from the throat, but under all circumstances where these symptoms arise in young children, even though they apparently swallow without discomfort, to examine the throat before deciding whether or not some other disease is developing.

On inspection of the throat the tonsils are seen to be enlarged in different degrees and to be of a uniform bright red color.

The mucous membrane of the pharynx is, as a rule, much reddened; the soft palate may also be reddened, but not necessarily. The symptoms continue for a day or two and then diminish, and the child usually recovers in about a week.

The local application of a cleansing spray, and the administration of ice if desired to relieve the discomfort, constitute all that is necessary for the treatment of these cases. It is best not to disturb the mucous membrane with applications on swabs or brushes.

Acute Follicular Tonsillitis.—In the acute inflammation of the tonsil which is usually called follicular tonsillitis, in addition to the general inflammation of the tonsils the crypts are especially affected. The cause of this form of tonsillitis is undoubtedly infection by some pathogenic germ. It is probable that more than one form of germ is capable of causing it. Many of the pathogenic germs which infest the mouth or the throat may be found in the crypts in this disease, but the especial germ by which we can characterize the disease has not yet been determined.

SYMPTOMS.—As a rule, the disease is characterized by an acute onset, with a heightened temperature, 39.4° to 40° C. (103° to 104° F.), loss of appetite, and general malaise. I have often noticed, however, that the symptoms of a marked follicular tonsillitis are not so acute and do not so definitely point to the throat in young children as they commonly do in older children and in adults. On examining the throat the tonsils are seen to be ged, reddened, and in the early hours of the disease to show a little ling of the orifices of the crypts, as though a secretion within them about to burst the overlying mucous membrane and appear on the free ice. Later this actually occurs, and the tonsils are seen to be studded white or grayish-white spots. These do not appear on the soft palate vula, though they may appear on the base of the tongue and the poste- pharyngeal walls. The mucous membrane of the pillars of the palate, ie uvula, and of the soft palate are usually reddened, and there is very o be decided reddening and even swelling of the mucous membrane and les of the pharynx. As the disease progresses these spots may coalesce adhering to the surface of the tonsil, form a pseudo-membrane which is impossible to distinguish from diphtheria without a bacteriological iination. As there is a direct connection between the tonsils and the ical glands, the latter are liable to be involved, though any great swell- of the cervical glands in connection with acute tonsillitis is uncommon.

The disease is self-limited, and runs its course in two or three days or a , at the end of which time the general symptoms subside, the appetite rns, the temperature becomes normal, and the child, although it is left what weakened by the disease, seems as well as ever. The tonsils selves, however, do not for some time regain their original size, and xudation often remains in the crypts and may cause a chronic irritation a tendency to recurrence.

DIAGNOSIS.—The differential diagnosis of follicular tonsillitis is to be e from the various forms of stomatitis, which I have already sufficiently ribed, and from diphtheria, of which I shall presently speak. It is now generally known that it is impossible absolutely to exclude diphtheria he morbid appearances seen on the tonsils. In the great majority of nces, however, where the attack is acute, where the cervical glands are especially involved, where the white spots on the tonsils are clearly ed in the orifices of the crypts, and where there is no appearance of a brane on the uvula or the soft palate, we can make the clinical diagnosis llicular tonsillitis with considerable certainty, but never surely without cteriological examination.

PROGNOSIS.—The prognosis of follicular tonsillitis is in almost every favorable, and is rendered unfavorable only by the complication of illar abscess. But you must remember that in an inflamed tonsil patho- c organisms, such as those of diphtheria, are more apt to develop.

TREATMENT.—The treatment of acute follicular tonsillitis, according to experience, should be entirely symptomatic. It is a self-limited disease, in a vast majority of cases is not benefited by the administration of any internally or by local applications. In order to avoid the invasion of various pathogenic germs during the progress of the tonsillitis I am in habit of having the throat kept thoroughly clean with mild solutions of rate of potassium or borate of sodium. Holding pieces of cracked ice ie mouth often affords considerable relief. In young children, as a rule, part, to true hypertrophy, generally accompanied by more or less inflammatory deposit. If the parenchymatous or glandular tissues are especially affected we find a soft, more or less red and vascular tonsil, with large crypts, often containing much secretion. In the interstitial form the tonsil is hard and tough, the crypts less prominent or even very small, and the vascularity much diminished. These types are the two extremes; in most cases the enlargement is essentially one of hypertrophy. The tonsils may be only moderately enlarged, or their size may be so increased that they meet, touching each other in the median line. The growth is usually towards the median line. In examining a child for enlargement of the tonsils care must be taken that the pharynx is not contracted by gagging at the time the examination is made. The act of gagging, which is easily brought about in children by a careless use of the tongue-depressor, brings the tonsils towards the median line, thus giving tonsils of normal size the appearance of being large and obstructive.

SYMPTOMS.—The symptoms of hypertrophy of the faucial tonsils vary according to the degree of enlargement. Normally the tonsils can scarcely be seen on inspection of the throat. When only moderately enlarged they may produce no symptoms whatever beyond a feeling at times of slight irritation in the throat. When in this condition, however, they are more apt to be irritated by various external influences and to be the source of recurrent acute affections of the throat. When considerably enlarged they may still not produce any marked symptoms, provided that the passage of air through the naso-pharynx is unobstructed. They may, however, even when the pharyngeal tonsil is not enlarged, cause obstruction in the naso-pharynx by pressure as they enlarge upward and backward. When this happens, the same interference with the breathing and development of the child takes place as when the obstruction is primarily in the naso-pharynx. These symptoms are the same as I have already described in speaking of hypertrophy of the pharyngeal tonsil, and therefore need not be detailed here.

Occasionally difficulty in swallowing and thickness of speech may arise where only the faucial tonsils are enlarged.

PROGNOSIS.—The prognosis in cases of hypertrophy of the faucial tonsils depends upon these varied anatomical conditions which I have just explained to you. So long as the tonsils do not encroach on the naso-pharynx the prognosis, so far as injury to the child is concerned, is good. You must always remember, however, that the enlargement of the tonsils is a fertile source of irritation which may prepare the way for serious disease produced by the various micro-organisms. The prognosis as to their disappearing is not especially good, as they seldom recover their normal size without active treatment when once hypertrophied, though they generally diminish slowly in size after puberty.

TREATMENT.—Local applications for the reduction of hypertrophied tonsils are useless. Some success has been obtained by Gampert by what is called discission of the tonsils. Leland has strongly advocated this treatment, which consists in making slits in the tonsil with a knife especially devised by him.

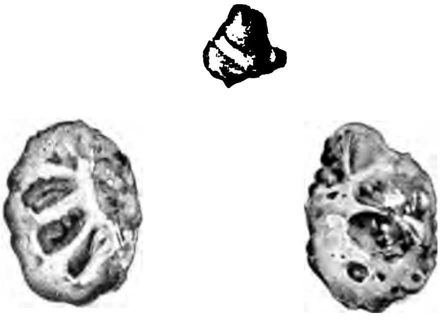
The most thorough and certain way of curing the disease is, however, by excision. This should be done with the tonsillotome, and it is best to etherize the child for the operation. It is considered wiser to amputate the tonsil than to enucleate it. After the operation the child should be made to gargle with a solution of borate of sodium for two or three days, and should be given only milk for its food.

As a result of attacks of acute inflammation the hypertrophied tonsils may have become adherent to the anterior or the posterior pillars of the fauces to such an extent that the guillotine either cannot be used, or not without danger of wounding these pillars. In such cases it may be necessary to revert to dissection or to the galvano-cautery to remove the tonsillar tissue.

I have an interesting case of hypertrophy of the faucial tonsils and of the pharyngeal tonsil to show you to-day.

The child (Case 401), a boy, seven years old, has for the past two years been under my observation. He was a healthy infant and well developed until his fifth year, when he did not continue to grow normally and lost much in weight. He became rather dull, and although he did not have any especially severe symptoms, yet he frequently had mild attacks of rhinitis, pharyngitis, and tonsillitis. After a number of recurrent attacks of tonsillitis the tonsils remained enlarged, and for the past two years he has presented in his throat the condition of hypertrophied tonsils. Various applications have been made to the tonsils without any favorable result. The specialist who first saw the child told the parents that the tonsils would probably decrease in size as the child grew older, and that operative treatment would not be advisable. The child is now beginning to breathe with his mouth open at night, and to be very restless when asleep, and has become very nervous. The chest, as you see, is decidedly flattened, and the sides of the nose are beginning to look a little pinched. The child is evidently suffering from obstruction to the entrance of air to the lungs, and I have therefore decided that operative treatment is indicated.

Fig. 101.



Part of the hypertrophied pharyngeal tonsil in the upper part of the figure. Excised surfaces of the faucial tonsils in the lower part of the figure. Male, 7 years old.

Dr. Farlow will now examine the case before you and operate on it. On passing the finger up into the naso-pharynx it is found that the pharyngeal tonsil is markedly enlarged, and as the result of the obstruction the breathing of the child is interfered with, and also its development.

The child is now etherized, and Dr. Farlow has first, as you see, excised both tonsils rith the tonsillotome and has then removed the pharyngeal tonsil with the forceps.

Here you will see the crypts and the intervening connective tissue of the excised ucial tonsils, and also the soft spongy tissue of the pharyngeal tonsil. (Fig. 101, p. 814.)

(Subsequent history.) Within six months after the removal of the pharyngeal and ucial tonsils the boy presented an entirely different appearance from what he did before he operation. His chest had developed, he had a better color and a good appetite, he had eased to be nervous, slept with his mouth closed, and showed no tendency to the recur-ence of the rhinitis from which he formerly suffered continually.

Peritonsillar Abscess.—In some cases an inflammatory process esulting in suppuration occurs in the cellular tissue around, above, or ehind the tonsil, constituting an abscess which is called peritonsillar. It s rather rare in early childhood. The disease is usually preceded by a ertain degree of simple tonsillitis, and when it develops the temperature ises, perhaps to 40° or 40.5° C. (104° or 105° F.), and the child evidently uffers much pain.

On inspecting the throat in these cases a unilateral swelling is seen in he neighborhood of the tonsil, as a rule, pushing the soft palate forward, nd the tonsil towards the median line.

The prognosis is in almost every case favorable, except those which ave been neglected or improperly treated. There is sometimes extensive urrowing of the pus, and hemorrhage, or even œdema of the larynx, may ccur.

The treatment is to locate the abscess by careful palpation and to open under strict antiseptic precautions with a guarded bistoury.

Pharyngitis.—An inflammatory condition of the posterior wall of the harynx is rather rare in infancy, but is not uncommon in children. It is sually coincident with an inflammatory condition of the naso-pharynx or f the tonsils, but in a certain number of cases it is so much more pronounced the posterior wall than elsewhere that it can be described as a separate isease. The diseases of the posterior wall of the pharynx are either (1) a mple catarrhal condition of the mucous membrane or (2) an inflammatory rocess in which the follicles are especially affected. In addition to these nditions, pus may form behind the mucous membrane, producing a retroharyngeal abscess. The inflammatory lesions of the pharynx may be ute or chronic.

The conditions which give rise to pharyngitis are the same various orbid processes that involve the parts in the neighborhood of the arynx, such as the naso-pharynx and the tonsils. These causes I have ready mentioned. There also seems to be a connection between certain ritations arising in the gastro-enteric tract and the pharynx. Where this curs it is usually the chronic form of pharyngitis which is met with, and e causal connection between these two distant parts of the economy is robably of a reflex nature.

Acute Simple Pharyngitis.—The pathological condition which is und in the simple acute form of pharyngitis is an acute inflammation characterized by a slightly heightened temperature, a hyperæmic condition of the blood-vessels of the posterior wall of the pharynx, and a certain amount of swelling and of serous exudation.

The symptoms are discomfort in swallowing, and at first a feeling of dryness in the throat, followed later by an exudation of viscid mucus. The child does not seem especially sick with this disease, and the length of the attack varies according to the influences which are causing it.

Acute Follicular Pharyngitis.—The acute follicular form of pharyngitis does not differ materially in its symptoms from the simple form, and in fact both forms are so frequently combined that a clinical distinction need scarcely be made between them. On examining the pharynx in this form of pharyngitis, in addition to the appearances which are seen in the simple form, the follicles will be found enlarged and raised above the surface of the mucous membrane.

TREATMENT.—The treatment of both forms of pharyngitis is essentially local, and is, as a rule, by applications to the inflamed mucous membrane in the form of a spray, either directly, or indirectly through the nose. The spray is essentially for the purpose of cleansing and thus soothing the inflamed mucous membrane, and should consist of mild alkaline solutions such as this one (Prescription 76):

PRESCRIPTION 76.

Metric.	Gramma.		*Apothecary.*	
℞ Sodii chloridi	0	3	℞ Sodii chloridi	gr. v;
Sodii bicarb.,			Sodii bicarb.,	
Sodii boratis āā	0	9	Sodii boratis āā	gr. xv;
Aquæ rosæ	30	0	Aquæ rosæ	℥i;
Aquæ	90	0	Aquæ	℥iij.
M.			M.	

Sig.—Spray for pharyngitis.

The chronic form of pharyngitis is usually accompanied by an irritating cough, which is most pronounced at night and in the morning. You should not think that these children who are coughing continuously and often losing in weight and looking weak and anæmic are necessarily affected with bronchitis. This series of symptoms is frequently looked upon as diagnostic of bronchitis, when it really arises from pharyngitis, and can be cured by treating the latter disease.

The treatment of chronic pharyngitis is to remove any hypertrophic condition of the tonsils, tongue, or nose, to regulate carefully the child's general nutrition, and to avoid undue exposure to inclement weather or to air vitiated in any way, as by arsenic or dust. Local applications of a one per cent. solution of nitrate of silver, followed immediately by thorough cleansing with sterilized water, are sometimes indicated in the more intractable cases.

Elongation of the Uvula.—Accompanying pharyngitis, usually in its chronic form, an elongation of the uvula is at times met with in children.

s condition may arise from a relaxed condition of the muscles of the palate and of the uvula, or may consist simply of a redundancy of the ous membrane at the tip of the uvula. The general irritated condition he uvula and the tickling sensation produced by its elongated tip touch- the base of the tongue cause a harassing cough, which by its persistence kens the child, gives rise to loss of appetite, and interferes with its ition.

Local applications of astringents, such as alum, are at times sufficient estore the uvula to its normal condition, but the disease can be cured kly by excising the end of the uvula with blunt-pointed scissors. amputation of the entire uvula is to be avoided, as it has been known to ken the muscular action of the soft palate.

A papillomatous growth is sometimes found attached to the tip of the la or to its side, which causes the same symptoms as elongation of the la. The treatment is excision, after which it does not recur.

RETROPHARYNGEAL ABSCESS.—Retropharyngeal abscess is a disease ch occurs usually during the first year of life and is very rare after this e. The disease may be secondary to injuries of the pharynx, to abscess he neck, and to disease of the cervical vertebræ, or it may be metastatic n septic processes such as occur in diphtheria. In a certain number of s it is idiopathic so far as we know.

PATHOLOGY.—The pathology of the disease consists in the formation n abscess in the tissues of the posterior wall of the pharynx, and is e apt to be on one side of the pharynx than in the median line.

SYMPTOMS.—The symptoms, whether the disease is primary or second- are very much the same. The first symptom is generally difficulty in llowing, which may go on to entire inability to swallow. The infant is t noticed to breathe in a peculiar way. It holds its head back and its th open. The breathing may be described as snorting, and at times as ost stertorous, differing markedly from the whistling sound which is d in obstruction of the larynx. On examining the throat the soft te is seen to be pushed forward and to be somewhat anæmic. The erior wall of the pharynx is bulging, usually unilaterally, is reddened, len, tense, and as the disease progresses is found to be fluctuating. In e cases the abscess burrows into the tissues of the neck and appears pear-shaped tumor behind the ear. I have met with two cases of this ety where the pus could be reached easily by an external incision.

DIAGNOSIS.—The diagnosis must be made chiefly from peritonsillar ess. This is, as a rule, not difficult unless the latter condition has eeded so far that the pus by burrowing has invaded the walls of the rynx. It is usually not difficult to determine the situation of the ess by passing the finger directly through the mouth to the posterior l of the pharynx. If there is pus in the tissues of the pharynx a e of fluctuation will be obtained. The position of the child in cases retropharyngeal abscess is also significant, and is not that which is assumed in peritonsillar abscess. It holds its head back, in order to allow a free passage for the air through the occluded pharynx into the larynx.

The diagnosis must also be made from œdema of the glottis, but this is not, as a rule, difficult, for inspection shows that in the latter disease bulging, redness, swelling, and fluctuation of the posterior wall of the pharynx are not present. The characteristic position of the head, also, is not seen in œdema of the larynx.

PROGNOSIS.—The prognosis in the cases where the abscess is secondary varies according to the nature of the disease which causes it. Thus, it is an exceedingly dangerous complication in diphtheria, and is one of serious import in cervical spondylitis. In those cases of undetermined origin which are spoken of as idiopathic the prognosis is very good if the proper treatment is carried out at once. We must, however, consider the possibility of the abscess bursting suddenly, the child suffocating by inspiration of pus into the larynx. This has been known to occur where the disease has been left untreated.

TREATMENT.—In the idiopathic cases the abscess should be opened at once. The method which I have found satisfactory in the cases which have come under my observation is to have an assistant hold the infant sitting upright in the lap, with a blanket tightly pinned around it so as to prevent it from moving its arms. Another assistant should hold the head. The mouth should then be opened, a guarded bistoury should be introduced into the pharynx and the abscess punctured. As soon as the opening has been made the bistoury should be removed quickly, and the infant's head should be immediately thrown forward and downward, so that the pus will be discharged from its mouth and not inspired into the larynx or swallowed. It is usually necessary after the operation to introduce the finger into the pharynx and to press the walls of the abscess, so as to empty any pus which may continue to collect there and also to keep the opening free. With this treatment, unless some complication should arise, the disease is usually cured in about a week.

Some operators prefer having the child placed in what is known as Rose's position, on its back with the head hanging over the end of the table.

Here is an infant (Case 402), seven months old, who has just been brought to the hospital.

Without examining the throat, you can almost diagnosticate a case of retropharyngeal abscess by the character of the breathing, which is snorting and labored. You will notice the very characteristic appearance produced by the obstruction in the pharynx. You see that the head is held back and the mouth open; that the infant's eyes are somewhat rolled upward, but that it is perfectly conscious. Its face is slightly cyanotic. On depressing the lower jaw and tongue you see that the soft palate is pressed forward, that its blood-vessels are almost empty, and that it is very pale. On throwing a strong light from the mirror on the posterior wall of the pharynx you see on the left of the median line a swollen, congested, bulging condition of the mucous membrane. On touching the most prominent point of the swelling with the finger you get a sense of fluctuation.

The infant is said to have been sick for two weeks with a cold in its head. Three days ago it began to breathe in this manner and to hold its head back rigidly and somewhat to the left. It also began to hold its mouth open. It was able to nurse only a few seconds at a time, when it would let go of the nipple and refuse to take it again. It has been growing very weak from lack of nourishment and from the exhaustion arising from the difficulty with its breathing.

Case 402.

Retropharyngeal abscess. Male, 7 months old.

(Subsequent history.) An opening in the abscess was made by Dr. Burrell, and a large amount of pus was evacuated. An hour later the child began to choke, and it seemed as though tracheotomy would have to be performed, but pressure with the finger on the walls of the abscess from time to time, surrounding the infant with an atmosphere containing steam, and free stimulation, proved eventually to be all that was necessary for its recovery.

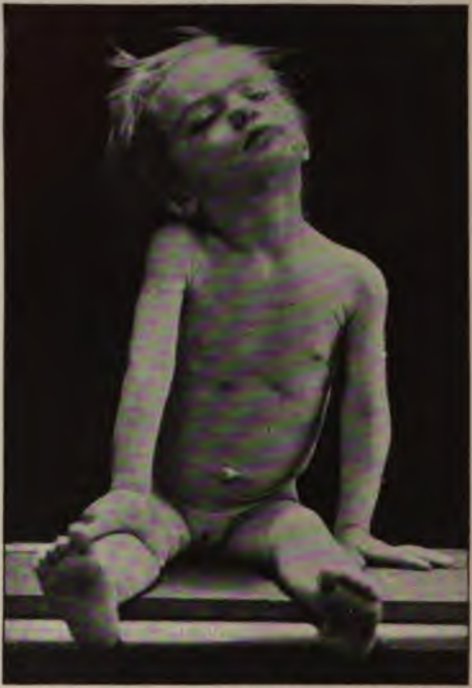
I shall now report to you a case (Case 403) of retropharyngeal abscess which came under my observation some years ago.

An infant thirteen months old, and always strong and healthy, had an attack of acute hinitis for several days. The rhinitis apparently caused considerable swelling and occluion of the nares, and the infant after four or five days began to hold its mouth open when breathing and to have difficulty in swallowing. This difficulty in deglutition increased, and it was then noticed that her head was held back. On examining the throat a tense fluctuting swelling was detected in the posterior wall of the pharynx very nearly in the median ine. This swelling was incised by Dr. Hooper. A considerable amount of pus was evacuted, and the infant immediately began to breathe more easily and was able to swallow withut difficulty. During the next twenty-four hours the abscess filled with pus a number of imes, and the pus had to be emptied by pressure with the finger. The infant made a perect recovery, and has had no return of the disease.

Cases such as this lead me to say a few words upon another method of drainage that I did not speak of when telling you about opening these abscesses that are found in the mouth. In the ordinary case, when the pus has come from the breaking down of glandular material in the posterior wall of the pharynx and already contains pyogenic bacteria, the dangers rom reinfection from a wound in the mouth are not serious. But when he pus has come from a tuberculous focus in the cervical vertebræ and ontains no other organisms than the bacilli of tuberculosis, and is morever in direct communication with an active pathological process in the bone, the risks of a secondary septic infection are considerable. It is, herefore, the practice of many surgeons, despite the difficulties of the operation, to attempt to reach the abscess by a careful dissection from the outside of the neck, as it is far easier to keep the wound in that situation septic. If there is any sign of the abscess pointing externally, the outside operation should always be preferred.

I happen to have here another case of retropharyngeal abscess to sho you, in which the condition is secondary to cervical spondylitis.

CASE 404.



Retropharyngeal abscess secondary to cervical spondylitis.

This child (Case 404) was being treated for cervical spondylitis by Dr. Bradford, when in addition to the drawing back of its head, which you see, it began to have increased difficulty in swallowing and to breathe with its mouth open.

On examining the pharynx a bulging, tense, fluctuating abscess of moderate size was detected.

LECTURE XLII.

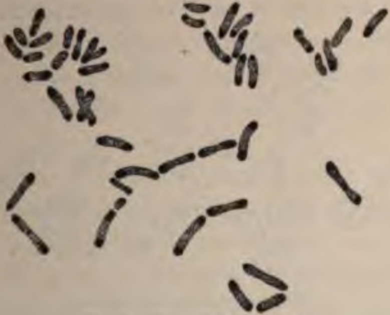
DIPHTHERIA.

DIPHTHERIA is an acute, highly infectious disease, due to the Klebs-ffler bacillus. It is primarily a local affection, the constitutional ptoms being due to the absorption of toxines.

There is frequently a concurrent infection with pyogenic cocci. These anisms produce the secondary inflammations occurring in the disease, and , by their toxines, give rise to additional constitutional symptoms.

ETIOLOGY.—The Klebs-Loeffler bacillus, first described by Klebs and r more fully identified by Loeffler, is a small organism, 2.5 μ to 3 μ in th and 0.5 μ to 0.8 μ in thickness. Its most striking features morphologi- y are its variation in form and the irregularity in its manner of staining. ends of the organism are frequently clubbed, and in most cases, when it tained, it shows a series of clear spaces with intensely stained particles. form and size vary greatly under various circumstances. I have here ecimen (Fig. 102) from a blood-serum culture from the throat of a child the diphtheria wards of the Boston City Hospital, which shows the rphology of this bacillus in its typical form.

FIG. 102.

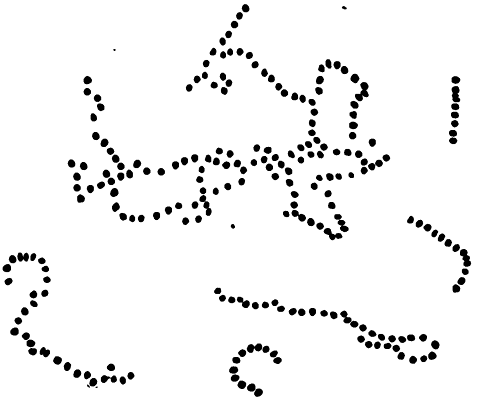


The bacillus of diphtheria.

It grows readily on a variety of culture media, and most readily on the dified blood-serum first introduced by Loeffler. It does not form spores. lch and Abbot have shown that in fluids it may be killed by an exposure ten minutes to a temperature of 58° C. (136.4° F.). Under favorable ditions it may remain alive for weeks, or even months, in fragments of d membrane. The bacillus of diphtheria is best stained with Loeffler's aline methylen-blue solution.

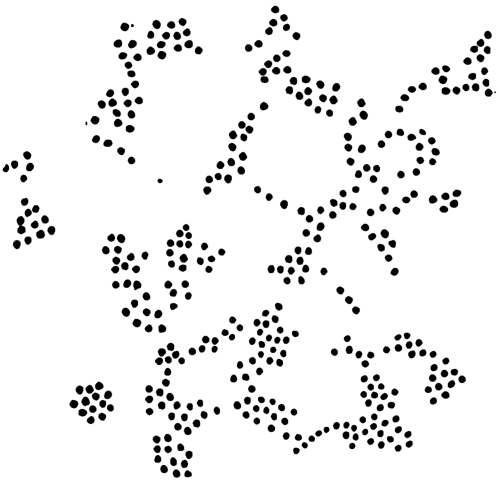
The pyogenic cocci most frequently found in the concurrent infections the streptococcus pyogenes, alone or associated with the staphylococcus pyogenes aureus, the former being the more important in its results. I have here some specimens (Figs. 103, 104) of these organisms, showing their morphology.

FIG. 103.



Streptococcus pyogenes.

FIG. 104.



Staphylococcus pyogenes.

You will notice that they both appear as dots, the streptococcus showing a tendency to arrange itself in chains, while the staphylococcus is irregularly bunched.

There is no true diphtheria where the Klebs-Loeffler bacillus is not present, but its presence in a healthy throat does not constitute the disease diphtheria, although the individual may be the source of infection to others.

The contagium of diphtheria is contained chiefly in the secretions of the throat and nose, and is communicated usually by direct or indirect contact, and, as a rule, not by the air.

An unhealthy condition of the mouth, nose, or throat predisposes to the disease, as a lesion of the mucous membrane is necessary for its entrance. Sewer-gas and confined, impure air of any kind may act by weakening resistance to the bacillus, or, by producing a benign lesion in the throat, may offer a suitable nidus for the invasion of the bacillus. Although clinically it has been supposed that animals have primary diphtheria, this has not been conclusively proved bacteriologically.

Although dipththeria may occur at any age, it is rarely met with in early infancy. It is most commonly seen from the second or third to the fifth or sixth year. It may occur more than once in the same individual.

PATHOLOGY.—The most constant lesion in diphtheria is the presence of a pseudo-membrane in the upper air-passages, due to a combination of inflammation and coagulation necrosis.

It must be remembered, however, that the same anatomical condition may be caused by other bacteria and by irritants, and also that the process may be simply a catarrhal inflammation which does not go on to the formation of a pseudo-membrane. It is evident, therefore, that there is no pathological condition characteristic of the action of the Klebs-Loeffler bacillus.

The adjacent lymph-nodes are apt to be swollen, and on microscopical examination they often show small foci of cell-necrosis; similar smaller necrotic foci may be found in other parts of the economy, such as the liver and the kidney, and are due to toxic absorption. There is also a general lymphatic hyperplasia, which is relatively greatest in the abdomen. The kidneys ordinarily show only parenchymatous degeneration, but in a few cases of concurrent infection they may present acute lesions. Hemorrhages into the serous membranes are often met with, and the organs in general show degenerative changes due to toxic absorption. Endocarditis is rarely seen. Catarrhal bronchitis and broncho-pneumonia frequently complicate diphtheria, and are caused by the inspiration of the pyogenic cocci. This was demonstrated by Prudden and Northrup in a very able paper which appeared in 1889.

INCUBATION.—The time which elapses after exposure to the infection until the first symptoms develop may be only twenty-four hours or may be two or three days. This period, however, is a very indefinite one, since the interval between the access of bacteria to the mucous membrane and the time when they invade the membrane with their toxic effects depends upon whether the tissues of the mucous membrane are vulnerable. Thus, it is probable that the bacillus diphtheriæ may exist in the mouth for an indefinite time without infecting the individual.

SYMPTOMS.—The prodromal symptoms of diphtheria are not especially characteristic. They may be acute in character or very mild and of a subacute variety. There are apt to be a sensation of chilliness, some heightening of the temperature, and more or less pain in the back and limbs. There is nothing, however, to distinguish this stage of the disease from many of the other affections of children, such as a simple tonsillitis. The child may often complain of discomfort on swallowing, and on examining the throat the fauces are found to be reddened. In a short time, however, more characteristic appearances will be found in the throat. A pseudo-membrane, white or grayish white, and commonly appearing on the tonsils first, develops, and on the second or third day usually extends to the soft palate and uvula. It may also appear in the pharynx. During this stage the throat becomes much swollen and the tonsils considerably enlarged, sometimes so as almost to meet in the median line. The membrane is usually firmly adherent to the mucous membrane, and as the case progresses it assumes a brownish-gray or yellowish-gray color. In addition to these lesions in the throat, the cervical glands are usually involved and become considerably swollen. The child, as a rule, shows grave constitutional symptoms and loses its appetite. The temperature in diphtheria is not characteristic, and is usually not especially high, 38.3° or 38.8° C. (101° or 102° F.). The pulse is somewhat increased in rapidity, and is weak in proportion to the severity of the disease. In cases of a mild type the symptoms abate towards the end of the first week, the pseudo-membrane separates, leaving a raw surface behind it, the throat becomes less swollen, and the child feels much better. It is, however, usually left much prostrated for a number of weeks, and even in these mild cases the toxic effects of the disease may show themselves in the form of a neuritis, with its accompanying paralysis, many weeks after the diphtheria itself has run its course. There may also even in mild cases be a slight discharge from the nares, owing to the involvement of the posterior nares, and a slight albuminuria.

I have brought you into the diphtheria ward to-day to show you one of these mild cases of diphtheria.

This boy (Case 405), five years old, has been sick for four days. His pulse is somewhat rapid, but of good strength. His respirations are slightly increased, but there is no retraction. There is a slight discharge from the nose, and the cervical glands are somewhat enlarged. He takes his nourishment well, and is in a very fair condition. A culture made on Loeffler's blood-serum of a shred of membrane taken from the throat showed the presence of the Klebs-Loeffler bacillus and a large number of streptococci. The urine contains a small amount of albumin.

I show you this case as especially illustrating the typical appearances of diphtheria in the throat, and in order that you may compare it with the typical appearances of the throat in follicular tonsillitis, which I showed you in a previous lecture (page 781).

On examining this boy's throat (Plate VIII., facing page 781, Diphtheria) you will see small patches of grayish-white pseudo-membrane on the upper part of the left tonsil and spreading to the left arch of the soft palate. The membrane has also involved the right side of the uvula, the right arch of the soft palate, and the side of the right tonsil pointing towards the median line. There is also a patch on the right tonsil and one on the posterior wall of the pharynx. The tonsils are moderately enlarged and reddened, and the mucous membrane of the soft palate is also considerably reddened.

When lesions of this character and having this distribution are seen in the throat you need have no doubt regarding the clinical diagnosis of diphtheria, and should at once have a bacteriological examination made.

VARIATIONS IN TYPE.—There are a number of variations which occur both in the severity of the disease and in the locality which is at first attacked or principally invaded.

In some epidemics the Klebs-Loeffler bacillus seems to be far more virulent than in others, and in some individuals it produces much more serious symptoms than in others. The severity of the attack does not always depend upon the extent of the pseudo-membrane. In general the severity of the cases depends on three factors: (1) the virulence of the bacteria, (2) the local resistance, and (3) the general resistance. A number of what may be called atypical cases have been observed and carefully studied, especially by Koplik, where no pseudo-membrane was detected and where the morbid appearances in the throat were those of a simple catarrh or follicular tonsillitis. The virulent Klebs-Loeffler bacillus was detected in these cases, and other children infected by them presented the typical local lesions of diphtheria.

In addition to these mild cases, the Klebs-Loeffler bacillus at times produces a most malignant form of diphtheria. In these cases the child either shows a fairly mild form of the disease for a few days and then suddenly develops the severe form, or it may be attacked at once by very severe symptoms. It becomes dull; the temperature is either slightly raised or may rise to 39.4° or 40° C. (103° or 104° F.), or higher; the pseudo-membrane spreads rapidly; there may be a dusky efflorescence on the skin, simulating closely that which I have described in the malignant form of scarlet fever. There may also be a purpuric condition of the skin. The picture of these septic cases is very characteristic. There is a peculiar, sweetish odor of the breath. There are cyanosis and a marked waxy pallor. There are hemorrhages from the throat and nose, with a profuse muco-purulent discharge from the latter. The cervical glands are often enormously enlarged. The membrane has been known to extend in all directions, and sometimes even through the Eustachian tubes to the external ears. All degrees of severity are met with between the mild and malignant types of diphtheria. The membrane, instead of extending upward to the naso-pharynx, as occurs in the malignant cases just spoken of, may spread downward, attacking the epiglottis and the larynx, and cause serious obstruction.

I have already told you that the pseudo-membrane most commonly appears first on the tonsils, thence spreading to the soft palate and to the uvula. The disease may, however, begin in the mucous membrane of any part of the mouth, nose, or throat.

The Nose.—Diphtheria sometimes begins in the nose and spreads no farther. In these cases the disease is usually of a mild type, but it is infectious. These cases are especially liable to be overlooked, as the child for one or two days may show merely the symptoms of fever, malaise, loss of appetite, and a discharge from the nose. On examining the nose carefully, however, a pseudo-membrane will often be found. It is, therefore, very important in cases of this kind to have a bacteriological examination made, and to isolate the child until it is determined that the Klebs-Loeffler bacillus is not present. These cases are probably a prolific source of infection to the community at large.

Where the naso-pharynx is affected, either primarily or secondarily through the nares or the pharynx, the constitutional symptoms are, as a rule, marked. This is in all probability accounted for by the great mass of absorbents in the naso-pharynx, where absorption takes place so easily that general septic poisoning quickly follows. Where the naso-pharynx is attacked by diphtheria, we usually meet with the most fatal results.

The Larynx.—In some cases the Klebs-Loeffler bacillus produces its effects first on the mucous membrane of the larynx. In these cases the mucous membrane of the nose and pharynx may never show any evidence of a pseudo-membrane. The first symptom, as a rule, is a cough of a harsh, ringing nature. The temperature may or may not be raised. As the toxic absorption is slight, on account of the locality affected, the constitutional symptoms are correspondingly mild. The child's symptoms are those resulting from laryngeal obstruction. There is dyspnœa, with retraction of the intercostal and supraclavicular spaces, and later of the epigastrium and the lower chest. This is accompanied by an increasing cyanosis. The child is very restless, is forced to sit up in order to breathe, and, for the same reason, bends forward with its head back. In these extreme cases, unless relief is speedily afforded, the child soon dies of suffocation. In another set of cases a slower form of suffocation may result from the extension of the membrane downward to the bronchi, while in still another set death may result from a complicating broncho-pneumonia.

A very prominent symptom in all forms of diphtheria may be cardiac weakness. In some cases the child may die suddenly without having presented any previous symptoms, or death may have been preceded by attacks of semi-collapse. In other cases there may be a weak, fluttering, intermittent pulse throughout the disease, which persists during convalescence. Under these circumstances the child should always be considered to be in a critical condition, as death, sometimes sudden, is liable to occur.

COMPLICATIONS AND SEQUELÆ.—There are a number of complications which arise in diphtheria besides those of laryngeal stenosis and cardiac weakness. The most serious of these are broncho-pneumonia and acute nephritis.

The form of pneumonia which complicates diphtheria is broncho-pneumonia, which, I have already told you, is produced, not by the Klebs-Loeffler bacillus, but by pyogenic cocci which have been inspired. Broncho-pneumonia is most frequent and most fatal in laryngeal cases which have been operated upon.

Albuminuria is so commonly met with in both the mild and the severe cases of diphtheria that it should be considered as a part of the disease rather than as a complication; as a rule, the greater the amount of albuminuria the more severe the case. Where acute nephritis complicates diphtheria it is not usually accompanied by œdema or anasarca.

Dysphagia may from the very beginning of the disease produce a profound impression upon the general nutrition. Otitis media occurs frequently.

Among the more common sequelæ are anæmia and chronic catarrh.

The most common and serious sequela of diphtheria is a peripheral neuritis, with its accompanying paralysis. This paralysis often does not appear until convalescence has been established,—perhaps in the third or fourth week from the time of the beginning of the attack. The paralysis may sometimes be merely of the muscles of the soft palate, in which case the fluids taken by the mouth are regurgitated through the nose; or it may have a general distribution, such as is seen in multiple neuritis. In the more severe cases of paralysis arising from this multiple neuritis, the lower extremities are affected and the knee-jerks are absent. The electrical reactions where the limbs are involved are the same as in peripheral neuritis from other causes.

The prognosis in these cases of post-diphtheritic paralysis is good.

DIAGNOSIS.—Recognizing that the same pseudo-membranous condition in the throat may occasionally be produced by the pyogenic cocci, as well as by the Klebs-Loeffler bacillus, the clinical diagnosis of a typical case of diphtheria is not difficult. A provisional diagnosis of diphtheria should be based upon the appearance in the throat of a pseudo-membrane, which usually appears first on the tonsils and has a tendency to spread to the uvula, soft palate, and pharynx. When in addition to this a nasal discharge is present and the glands of the neck are much enlarged, you have a picture which is not shown by any other disease. The most common difficulty met with clinically is in distinguishing between cases of acute follicular tonsillitis and diphtheria.

As I have already stated, the local lesions produced by the Klebs-Loeffler bacillus may be merely a catarrhal inflammation or those of a follicular tonsillitis. All such conditions, therefore, should be looked upon with suspicion until the absence of the Klebs-Loeffler bacillus has been demonstrated bacteriologically. Although a membranous laryngitis may be due to other causes than the Klebs-Loeffler bacillus, yet this is so rare that every case of primary membranous laryngitis should be considered to be diphtheria until it has been proved that it is not. A decisive diagnosis of diphtheria in any case can, therefore, be made only by determining the presence of the Klebs-Loeffler bacillus.

PROGNOSIS.—Diphtheria is an extremely fatal disease, especially in the septic and obstructive cases. The mortality varies decidedly in different epidemics and according to the age. Children under two years of age rarely recover. The rate of mortality seems to have lessened in cases where the antitoxin treatment has been thoroughly used. The symptoms which make the prognosis especially unfavorable are the extension of the membrane to the naso-pharynx or the larynx, extensive glandular enlargement, hemorrhage from the nose or into the skin, a high grade of albuminuria, broncho-pneumonia, and a weak heart. Morse, in an extensive study of the leucocytosis of diphtheria, has shown that it is of no prognostic value. The cases of neuritis invariably recover. The prognosis in all cases is uncertain, and should be given with caution, as death from heart-failure is liable to occur at any stage of the disease.

A child who has had diphtheria is liable to suffer from the deleterious effects for months or even years.

PROPHYLAXIS.—All patients with diphtheria should be isolated until the Klebs-Loeffler bacillus has disappeared from the nose and throat. The time when this occurs varies from a few days to a number of weeks.

In order further to protect the community, all cases of sore throat should be examined, and if the Klebs-Loeffler bacillus is found the individual should be isolated. It is especially necessary to carry out this precaution in schools, where the conditions are so favorable for the spread of the disease.

The throats and noses of all persons exposed to diphtheria or caring for diphtheritic patients should be repeatedly examined for the Klebs-Loeffler bacillus, and if this is found they should be given immunizing doses of antitoxin, the amount and frequency of the doses to be modified as our knowledge increases. If in the future it is proved that the antitoxin may produce serious effects in certain individuals, these views must be modified to correspond to this additional knowledge. If the Klebs-Lœffler bacillus is found in these individuals, they should be isolated so long as the bacillus is present. To shorten the period of isolation, mild antiseptic gargles or douches should be employed. Whether the isolation of healthy persons who have the Klebs-Loeffler bacillus in their throat or nose is advisable or not is still a mooted question. Much confusion has arisen because of the so-called pseudo-diphtheritic bacillus. The weight of evidence at present, however, goes to show that it does not exist, and that the bacteria described are merely Klebs-Loeffler bacilli of diminished virulence. At any rate, even if the pseudo-diphtheritic bacillus exists, it is so rare that it may be safely excluded in clinical work. The fact that the Klebs-Loeffler bacilli found in healthy throats may not be virulent is not an argument against isolation, because it is well known that a non-virulent form may become virulent when transferred to a different soil. Examinations of many healthy throats have shown that the Klebs-Loeffler bacillus is a very rare inhabitant of the normal throat, and that when it is present diphtheria often develops later. Theoretically, therefore, although it may be impossible or inadvisable practically, it would seem wise to consider the Klebs-Loeffler bacillus virulent until it has been proved to be non-virulent, and to consider its presence a source of danger to the community until it is proved not to be.

In addition to what I have already said, I must impress upon you the importance of keeping the teeth in good order as a prophylactic measure, as well as keeping the mucous membrane of the nose and throat in a normal condition.

TREATMENT.—The treatment of diphtheria consists (1) in attending to the hygienic conditions; (2) in the administration of remedies, either by the skin or by the mouth, to combat the toxine which produces the constitutional symptoms; (3) in local applications to the nose, throat, or larynx, and in measures directed to the general condition; (4) in operative measures to relieve obstruction in the larynx.

One of the most important parts of the treatment of diphtheria is the management of the room in which the patient is kept during the progress of the disease. It is well known that pathogenic organisms, such as the Klebs-Loeffler bacillus, do not thrive where they are exposed to sunlight and fresh air. The room should be thoroughly ventilated, and fresh pure air should be allowed to come continuously into it. It should also be one which has a sunny exposure.

In any treatment directed to the cure of diphtheria in young children we must remember that the disease is so exhausting that the treatment, as a rule, should be forced upon the child as little as possible. Any physical exhaustion produced by the treatment is to be considered serious in young children.

It is necessary perhaps to call your attention to the fact that much care should be taken both by the physician and by the nurse not to become infected themselves by the secretions from the mouth and nose of the patient. These secretions are especially dangerous if they happen to get into the eyes. It is probable that with extreme care there is not much danger of the spread of diphtheria in a household, as we know its tendency is not to disseminate itself in the surrounding atmosphere. Hence it is likely that with proper precautions it can be limited to the room in which the child is sick, and that if it extends beyond this room it has been carried directly by the hands or clothing of the nurse or the physician.

According to the knowledge of the present time, the most promising of all these forms of treatment is the second. This treatment is essentially comprised under what is called serum therapeutics. By serum therapeutics is meant the treatment of disease by injecting into the patient the serum of an animal which has been rendered immune to the especial disease, which is being treated, by means of inoculation with the toxine of that disease. The serum taken from the animals which have been rendered immune against diphtheria is called antitoxin serum. The serum is injected under the skin, usually in the thigh, and the place selected should always be one on which pressure is not exerted when lying in bed. The dose should be from 500 to 1000 antitoxin units, or 10 to 20 c.c. ($\frac{1}{3}$ to $\frac{2}{3}$ ounce) of the 1 to 50,000 serum, according to the age of the child and the severity of the disease.

The beneficial results of antitoxin are decidedly greater if the injection is made in an early stage of the disease than if made in the later, although even when administered late in the disease it sometimes produces wonderfully curative effects. When given early, within the first forty-eight hours of the disease, even where the membrane is spreading rapidly and inflammation of the glands with general systemic poisoning has taken place, one injection will often arrest the disease. Where improvement does not take place within twenty-four hours, a second dose, and, if necessary, a larger one, should be used. The sign by which we know that the antitoxin serum is beneficial is the improvement in the general condition of the patient. The effect of the antitoxin on the pseudo-membrane is characteristic. The pseudo-membrane ceases to spread, frequently whitens, shrinks, shows a line of demarcation, and usually within the next three or four days becomes detached from the mucous membrane. The temperature usually rises after the injection, but in a few days falls to the normal by lysis. In the more severe cases a single injection of the serum does not work so quickly. In these cases the temperature falls usually by lysis after the second or third dose. The pulse becomes normal two or three days after the temperature has fallen. The irregularities of the pulse are not so frequent in diphtheria since the antitoxin treatment has been employed. The effect of antitoxin on the albuminuria is still *sub judice*, but it probably does not increase the likelihood of its occurrence. When there is a concurrent infection the antitoxin serum is less effective, since it does not counteract the toxic absorption due to other bacteria than the Klebs-Loeffler bacillus. It is not safe to assume, however, that there is a concurrent infection because other bacteria are found in the throat.

When the larynx is involved, with accompanying stenosis, the antitoxin serum is found to be very valuable, and has reduced the number of operative cases.

In connection with the antitoxin treatment no specific drugs given internally by the mouth are indicated. Stimulants should always be given freely in diphtheria. Of course, symptomatic treatment of any kind is not contra-indicated.

The antitoxin has been found to have but little effect on the length of time during which the bacteria remain in the throat after the disappearance of the membrane.

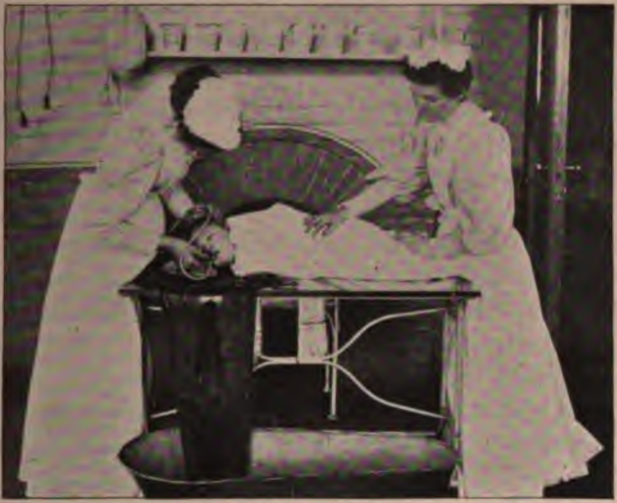
Too few cases have as yet been observed to estimate the relative frequency of the occurrence of neuritis since the treatment by antitoxin has been introduced. Various skin and joint complications, accompanied by fever, occur in a certain proportion of the cases in which antitoxin is used. Albuminuria has been attributed to its use; but, as already stated, this question must still be considered as *sub judice*. Antitoxin is also said to cause serious and even fatal results in some cases. How much danger there may be in its use cannot as yet be estimated, but must be left to the future to decide. Careful clinical observations and autopsies on fatal cases in which it has been used can alone enlighten us.

The local treatment of diphtheria consists in thoroughly cleansing the mouth and nose with warm, non-irritating solutions, such as normal salt solution, or boracic acid four per cent. All strong and irritating applications to the throat in diphtheria are harmful.

The technique of the local applications to the throat and nose is important. The most simple, efficacious, and safe, and that which produces the least discomfort, is by irrigation. The same method—namely, by means of a fountain syringe—should be employed for either the throat or the nose, except that in the former a larger hard-rubber nozzle should be used than for the nose, and one which is sufficiently long to pass over the base of the tongue.

Here is an illustration (Fig. 105) of the method of irrigation as employed in the Boston City Hospital and at the Willard-Parker Hospital in New York.

FIG. 105.



Irrigation of nose in diphtheria.

The child should lie on its side, and the water should be made to pass up one nostril and down the other until the stream runs clear. In some cases the child prefers to sit up while the irrigation is done. Ordinarily, the irrigation should be used once in two or three hours, perhaps with longer intervals at night. If the child resists this treatment, it may be advisable, in order to save its strength, to omit it for a time. This rule applies to all forms of local treatment.

Considerable suffering is at times occasioned by the enlargement of the cervical glands. Some patients prefer the application of ice poultices, others of hot flaxseed poultices. Either may be used if they produce the desired effect of reducing the discomfort.

Nutritive enemata made of peptonized milk, with stimulants, may, when retained, be an important adjunct to the treatment. Enemata, however, are often not retained. Digitalis may be used in cases where heart-failure is anticipated. In cases where there is a faucial paralysis the child may often, with success, be fed through the nostril by means of a soft-rubber catheter passed into the œsophagus; this method may also be used after intubation where there is unusual difficulty in swallowing.

Where measures are found to be necessary to reduce obstruction in cases of stenosis of the larynx, the child should be placed in an atmosphere of steam, and if this does not relieve the stenosis the sublimation of calomel should be employed. In either case, however, we must remember that the child should not be kept in this atmosphere continuously, and should be watched carefully to see if it is speedily relieved of the stenosis; for if it is not, the continuous inhalation of steam in the comparatively small area of breathing space which exists in the tent that is used for this purpose may of itself be detrimental to the child's recovery, from lack of sufficient oxygen. When tracheotomy has been performed an atmosphere of steam is especially valuable.

The tent, as described by Dr. Northrup, who has used it so extensively in the Willard-Parker Hospital in New York, contains about fifty cubic feet of air. To extemporize a tent, a sheet is thrown over supports above the crib and allowed to fall over the four sides of the crib. The main point is to have a fairly large and tight enclosure. The apparatus for furnishing the steam or sublimation must be free from the danger of upsetting and of setting the tent on fire. For sublimation, a deep vessel, such as a wash-bowl, should have an alcohol lamp placed in it, and over its top a tin strip. Over the space where the flame of the alcohol lamp touches the under side of the strip a little, compact pile of calomel, sufficient for a single sublimation, is placed. Eight or ten minutes are required to volatilize the calomel, and the tent should be kept closed about fifteen minutes. A safe and satisfactory method is to volatilize in an ordinary crib-tent 0.3 gramme (5 grains) of calomel every two hours for two days and nights, and then prolong the intervals to three hours on the third day, four hours on the fourth day, and later three times a day, according to indications (O'Dwyer). It has been Dr. Morse's experience that 0.6 gramme (10 grains) every half-hour for four or five times will sometimes produce good results where the smaller doses have failed.

The nurse may easily become salivated from inhaling the calomel fumes, and should be cautioned in regard to this. The child should not be exposed to a sudden change of temperature when the sublimation is over. The room should be thoroughly aired after opening the tent, and it is well to remove the child to another room while this is being done. Young children do not, as a rule, suffer from ptyalism following this sublimation. Older children, after a number of days' treatment, may show a mild stomatitis, and sometimes diarrhœa. Chemically pure calomel is essential, as the impure drug may cause conjunctival irritation. If the fumigation has to be very prolonged, anæmia may be caused. This should be combated by iron, and if there is prostration, a little whiskey should be given before the sublimation.

The sublimation of calomel is indicated where the symptoms of laryngeal obstruction are urgent, and may be used alone or in conjunction with steam.

Where the antitoxin does not relieve the symptoms of stenosis, and where the progressive dyspnœa is not quickly controlled by steam or calomel sublimation, it is well not to delay operative interference. The operative means of relieving stenosis of the larynx is by intubation or by tracheotomy. The indications in either case are, according to Northrup, a progressive, unremitting dyspnœa, when the labored breathing begins to produce sensible exhaustion, and when the supraclavicular and lateral thoracic retraction is marked. It would not be within my province to speak of the relative advantages of intubation and tracheotomy. Each operation has its strong exponents, and so much has been said in favor of both operations that the question as to which is best must be decided by the individual surgeon in the especial case. It is probable that the antitoxin treatment will increase the field for intubation in operative cases.

In the treatment of post-diphtheritic paralysis strychnine is the most valuable drug. Electricity, especially faradism, is also indicated.

The subsequent anæmia, which I have already referred to, should be treated in the usual way.

DIVISION XIII.

DISEASES OF THE ŒSOPHAGUS, STOMACH, AND INTESTINE.

LECTURE XLIII.

INTRODUCTION.

Before speaking in detail of the diseases of the stomach and intestine, a few general remarks are necessary to explain how limited is our knowledge of these diseases. Those diseases, however, which affect the œsophagus can easily be classified on a pathological basis, and are so few in number that they can be included in these general remarks.

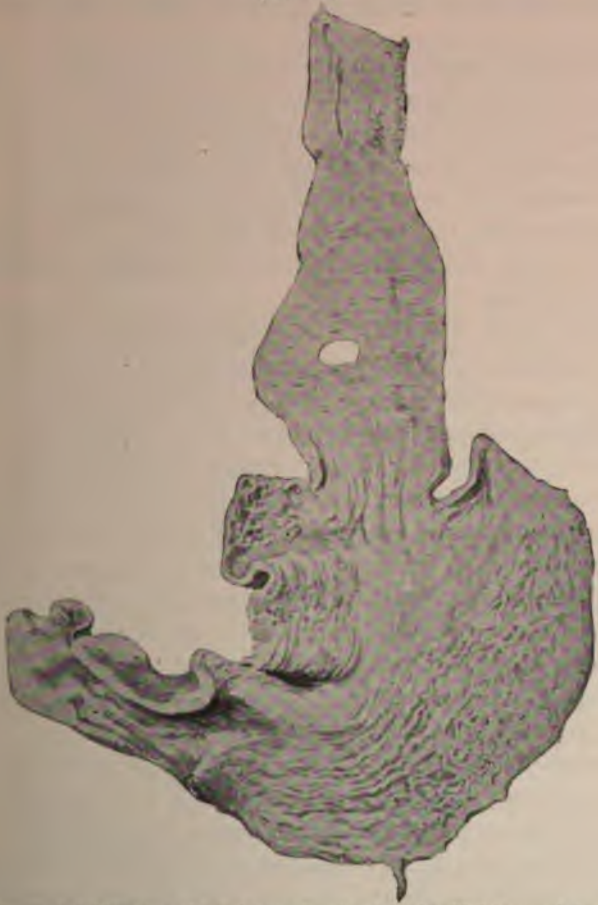
ŒSOPHAGUS.—The diseases of the œsophagus are rare in infancy and early childhood. There may be congenital malformations, such as narrowing or dilatation. The swallowing of hot or corrosive liquids may cause obstruction, which is occasioned by a cicatricial stricture. Œsophageal stricture may also occur as a result of congenital syphilis. Pressure outside of the œsophagus may cause obstruction. These strictures, especially those of cicatricial origin, are accompanied by a great deal of muscular spasm, which at times is constant, and again relaxes. Thus, the child will swallow with comparative freedom at intervals, while at other times the obstruction appears to be complete. In addition to the inability to swallow, and the consequent regurgitation of the food, the secretion of saliva and mucus is often very profuse, and causes symptoms of distress and choking.

The diagnosis and treatment of these cases are effected chiefly by means of bougies; but, as much harm may come from these instruments, and as especial surgical knowledge is required to use them and to decide whether œsophagotomy should be performed, I shall not dwell on this class of cases.

An inflammatory condition of the œsophagus is said to occur in young infants, and is spoken of as *œsophagitis*. It is rare. The symptoms, as described by Billard, are unwillingness to nurse, crying, immediate regurgitation after beginning to suck, and often some tenderness about the neck on pressure. The prognosis is bad.

It is quite common for children to swallow various *foreign bodies*, such ıttons and pins. These bodies may either be caught in the back of the ıt or lodged in the œsophagus, instead of passing through to the stomach. reful examination of the throat with the finger should first be made, if the foreign body is not detected in the throat the œsophagus should xplored carefully with a bougie, and the foreign body is then usually ed through into the stomach, unless it is thought wiser to remove it the bristle probang. The diet for the following twenty-four to forty- hours, or until the body has been passed through the intestine, should ıch as will give sufficient consistency to the fæces to protect the intes- from injury while the body is being passed over its surface. Various

Fig. 106.



Congenital dilatation of œsophagus, female, 10 weeks old (⅗ natural size).

arations of the cereals are useful for this purpose. If necessary, a dose l can be given, but, as a rule, active treatment is contra-indicated.

have here a specimen of the œsophagus and stomach of an infant 406) ten weeks old which shows the condition of congenital dilatation ıe œsophagus (Fig. 106).

The infant was healthy at birth, and its mother had a plentiful supply of breast-milk. During the first two or three weeks of its life nothing abnormal was noticed about it, except that it vomited occasionally. When it was four weeks old it began to regurgitate, vomited the milk frequently, and lost in weight. The fæcal discharges showed that the milk which reached the stomach and intestine was fairly digested, but the discharges were infrequent and small in number. It was weaned when it was nine weeks old, and small amounts of milk, carefully modified in various ways, were given to it. No improvement in the symptoms followed this treatment, and although at times a small quantity of milk would be retained, yet, as a rule, after a few minutes the milk was regurgitated. The infant had no other symptoms, but rapidly lost in weight, and finally died of exhaustion.

The post-mortem examination was made by Dr. Whitney, and the only pathological conditions found were, as you see, in the œsophagus. The last two inches of the œsophagus were dilated into a more or less cylindrical swelling, with marked thinning of the walls and atrophy of the mucous coat. A dilatation had been formed in which evidence of a small area about to perforate into the mediastinum was found. The entire stomach, as well as its cardiac and pyloric orifices, was markedly contracted, apparently from lack of use.

STOMACH AND INTESTINE.—Our knowledge of the diseases of the stomach and intestine is exceedingly limited, and is especially so where infants and young children are concerned. The classification of these diseases on a pathological basis has been proved to be inadequate, and in like manner a classification on the basis of symptoms is insufficient. Bacteriological investigations, however, have advanced our knowledge to such an extent that we may hope in the future to be able to classify these diseases on an etiological basis. The terms dyspepsia, dysentery, diphtheritic, croupous, and others have become almost unmeaning, and should be replaced by terms more closely connected with the etiology of the disease.

Accordingly, the American Pediatric Society requested Dr. Holt and myself to prepare a nomenclature which would correspond more nearly to our present knowledge of this exceedingly difficult subject. I wish especially to emphasize the value of Dr. Holt's work, which has aided me so much in my own studies on this subject. The classification finally adopted by the Society was one which especially relates to infants and young children, and you must remember that in what I am about to say concerning this important class of diseases I am dealing especially with this early period of life. The diseases of the gastro-enteric tract as they occur in older children resemble so closely those of adults that they need not occupy a prominent place in lectures on children, especially as the pathology and symptoms of this later period of life differ very materially from those of the earlier periods. These differences are still more strongly marked from the fact that children succumb much more readily to the early stages f a disease than do adults, and may die before the later and more characristic lesions and symptoms of the disease have developed. There are rtain known facts resulting from the anatomical and physiological peculiities existing in infancy which play a significant part in all these diseases. is well, therefore, first to explain the general principles which influence e symptoms and prognosis of these diseases before attempting to describe ch disease separately. In many cases we can arrive at only approximate nclusions as to the actual lesion which exists and the prognosis which ould be given. A practical clinical diagnosis should be made according the region where the stress of the lesion exists, rather than to the pathogical lesions which are present.

General Etiology.—In the present state of our knowledge it is not racticable to discuss in detail the various supposed causes of gastro-enteric isturbances. We can suppose that these disturbances may be due to nerous conditions which may act alone or may render the tissues vulnerable bacteria. Some of these diseases are caused by specific organisms, while thers are due to a number of organisms. These bacteria act either of hemselves or through their products.

In a general way, these diseases can be classified as functional and rganic. The organic class may be divided into inflammatory and non-nflammatory diseases, although the boundary-line between these two conitions is at times very doubtful. A prominent and important peculiarity f these diseases as they occur in infancy is, as would naturally be expected t this early period of development, a variety of symptoms which are prouced by reflex causes. By the term reflex we mean peripheral irritation ith a resulting action. By functional we mean a disturbance of the function f the organ without a known lesion. By organic we mean a known lesion.

In addition to these cases are others which, as yet imperfectly undertood, seem to be produced by certain morbid products eliminated from the lood by the gastro-enteric tract, as, for example, urea. This etiological ctor can be spoken of under the term *eliminative.*

General Pathology.—The general pathological anatomy of the astro-enteric tract of infancy and early childhood is essentially that of the eum and colon. In those cases in which the more severe lesions are resent the stress of the lesion is usually in the lower ileum and the colon, nd very frequently in the colon only. For this reason the terms ileo-litis and colitis seem more descriptive than ileo-enteritis and enteritis. he pseudo-membrane in ileo-colitis is often extensive, but sloughing and rforation are exceedingly rare in young children. It is at present believed at not all ulcers of the gastro-enteric tract are necessarily inflammatory. he great number of lymph-nodules and the abundance of the lymphatic exuses are the principal anatomical conditions which influence the patholgy of the enteric tract in early life.

General Bacteriology.—The knowledge of the different bacteria hich occur in the gastro-enteric tract, and of the connection which they have with the different diseases, is at present, with few exceptions, uncertain and unreliable. There is little doubt that the bacteria may find their way, by means of the stomach, to the intestine, and that the acid secretion of the stomach which they meet in their way through it is not sufficient to prevent their arriving alive in the intestine. We know that these bacteria play such an important rôle in their etiological relations to the various diseases that full weight must be given to their presence when we are treating the disease. It would seem that the bacteria which are commonly found in the intestine when it is in a normal condition do not cause any abnormal conditions; but when the intestine has become irritated, from mechanical or thermic causes, the bacteria are able to penetrate its mucous membrane, become noxious, and produce abnormal symptoms, often of a serious nature.

GENERAL SYMPTOMATOLOGY.—Vomiting as a symptom is often very misleading in early life, so far as the differential diagnosis between the stomach and the intestine is concerned, as it frequently occurs from disturbance in any part of the gastro-enteric tract, and should not be considered as indicative of any one disease. Serious symptoms during life are often proved at the autopsy to have been produced by no pathological lesion, while grave lesions may be found at the autopsy where the intestinal symptoms during life were very mild.

Marked diarrhœa may exist during life and no lesions be present at the autopsy. Serious lesions may exist, and yet no blood appear in the dejections. Blood may appear in the dejections, and yet no serious lesion exist, the hemorrhage being only temporary, and comparable to epistaxis.

GENERAL DIAGNOSIS.—The observation of the temperature is very important for the diagnosis of these diseases. As a rule, an elevated temperature of short duration points towards functional and toxic disturbances, while an elevated temperature long continued points towards inflammatory lesions.

Intestinal discharges are often very misleading for diagnosis.

Having considered and accepted these general principles relating to diseases of the gastro-enteric tract in infancy, the American Pediatric Society adopted the following classification (Table 109), as presented to them by their committee. This classification must be understood to be merely provisional, and is for the purpose of aiding those who are interested in this subject to work with uniformity.

At the same time it is believed that it is a great advance upon the unmeaning and misleading nomenclature now current.

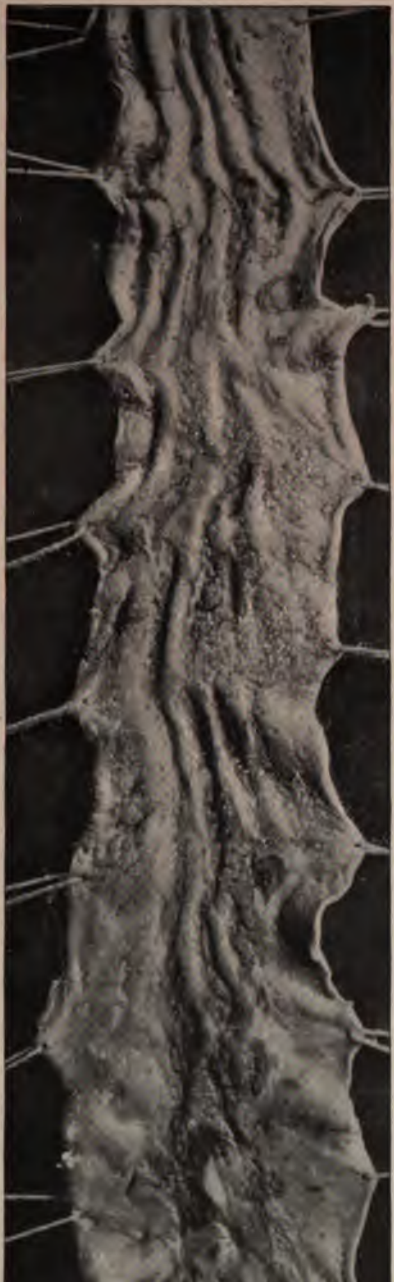
On examining this table (Table 109) you will see that whenever the etiology has been definitely determined it is made to designate the disease, but the true etiology is still unknown in so many cases that other terms have of necessity been used, the names simply representing the extent of the knowledge we have of the especial disease.

The diseases of the gastro-enteric tract may, on this basis, be divided into diseases of the stomach (gastric), diseases of the intestine (enteric), and

TABLE 109.

- *Gastric*
  - *Developmental*
    - Malformations.
    - Malpositions.
  - *Functional*
    - *Acute*
      - *Nervous* (vomiting)
        - CENTRAL. From many causes, as tubercular meningitis, heat, cold, fright, etc.
        - REFLEX. Irritation from foreign bodies, food, or otherwise.
      - *Indigestion.*
    - *Chronic* — *Indigestion.*
    - *Eliminative.*
  - *Organic*
    - *Non-Inflammatory.*
      - *Mechanical*, dilatation, etc.
      - *Ulcers.*
      - *New Growths.*
    - *Inflammatory.*
      - *Acute* — *Gastritis.*
        - *Catarrhalis.*
        - *Corrosiva.*
        - *Pseudo-Membranosa.*
        - Such as, according to Wollstein in *Archives of Pediatrics* for July, 1892, is met with in exhausting diseases, atelectasis, diphtheria, tuberculosis, etc.
      - *Chronic* — *Gastritis.*
        - *Catarrhalis.*
- *Developmental*
  - Malformations.
  - Malpositions.
- *Acute*
  - *Nervous* (exaggerated peristalsis causing diarrhœa).
    - CENTRAL. Heat, cold, fright, etc.
    - REFLEX. Foreign bodies, food, or otherwise
    - Both small and large intestine may be affected, and the condition of the mucous membrane is normal or hyperæmic.
    - Tubular.
  - *Indigestion* . . . Especially of duodenum · frequently some icterus and vomiting of bile.
    - [illegible]
    - A definite etiology and pathology.
    - *Typhoid bacillus.*
- *Chronic*
  - *Appendicitis.*
  - *Ileo-Colitis or Colitis*
    - Follows acute attacks, especially of ileo-colitis.
    - *Tubercular.*

FIG. 107.



Colon showing presence of bismuth which had been given by the mouth.

the disturbances which arise from animal parasites. The diseases are then divided into those which arise from developmental, those which arise from functional, and those which arise from organic causes. The organic diseases are subdivided into non-inflammatory and inflammatory, and the functional and organic diseases into acute and chronic.

GENERAL TREATMENT.—In the treatment of these diseases we should endeavor to carry out four general rules: (1) to combat the serious conditions already referred to; (2) to dislodge the bacteria as quickly as possible, perhaps by laxatives and irrigation; (3) not to introduce into the gastro-enteric tract for a certain period food which may prove a favorable culture ground for the bacteria, since it has been shown that where the food is sterile when it enters the gastro-enteric tract it is quite effective in reducing the number of bacteria in the intestine; (4) to introduce such drugs into the gastro-enteric tract as may, by their anti-fermentative and germicidal powers, diminish the action of or destroy the bacteria. This last rule is, however, very difficult to carry out, and, with our present knowledge of drugs and their administration, practically impossible. It is true that we know that subnitrate of bismuth is an anti-ferment, and that it reaches the part of the enteric tract which we know to be most affected in enteric disturbances characterized by fermentation. In proof of this I need simply refer you to this intestine (Fig. 107) of an infant, given to me by Dr. Holt, to illustrate this point, where bismuth had been given, and where at the autopsy the bismuth was found thickly coating the mucous membrane of the small intestine, and also appearing in the large intestine. It is, however, questionable whether in any case the attempt to kill the bacteria by the internal administration of drugs has been successful. Preparations, such as salol, which are known to be broken up into their carbolic acid components on reaching the intestine, cannot with safety be given to the infant in doses large enough to kill the bacteria, for in such doses there may be serious results from poisoning. We can, however, possibly, by means of these germicidal drugs, produce a condition in the intestine which, though not conducive to the death of the bacteria, may yet be so unfavorable for their growth as to aid our treatment when we are endeavoring to dislodge them. Nothing definite has, however, as yet resulted from using drugs for this purpose, and, so far as I can judge, the danger of treating infants or young children in this way is greater than the good that may result from it.

LECTURE XLIV.

DISEASES OF THE STOMACH.

FROM what I told you in the last lecture you will understand how difficult it is to make a differential diagnosis between gastric disease and gastro-enteric disturbance. The only symptom which definitely shows the stomach to be involved, whether from reflex, functional, or organic conditions, is vomiting, and, as we know that in many cases vomiting is caused primarily by disturbance of the intestine, we really have no symptom which represents gastric disease alone. The difficulty of locating disease in the stomach is rendered still greater by the fact that serious organic lesions may exist in the stomach without any symptoms whatever, whether of vomiting, pain, or tenderness. We must, therefore, be exceedingly cautious in making a diagnosis of diseases of the stomach.

Diseases of the stomach may arise from *developmental*, *functional*, or *organic* causes.

DEVELOPMENTAL.—Under developmental affections of the stomach are included *malformations* and *malpositions*. A malformation of the stomach may be represented by a narrowing of either the cardiac or the pyloric orifice, or by constrictions in various parts of the ventral cavity, which are known as hour-glass contractions. A malposition of the stomach may be met with in various places, one of which is in the thoracic cavity. These malpositions, however, are exceedingly rare, and of pathological rather than of clinical interest, as the diagnosis can scarcely be made during life.

FUNCTIONAL.—The functional diseases of the stomach play a great rôle in infants and in young children. They may be of an *acute* or a *chronic* variety, or may be what I have spoken of as *eliminative*. The latter class, in which certain morbid and irritating substances are supposed to enter the stomach as though it were an excretory organ, may in the future explain many of the rather obscure gastric symptoms which arise in early life, but at present our knowledge concerning this class of cases is so slight and indefinite that they need merely be alluded to. Acute functional gastric symptoms may be produced by a number of causes, but in general they are to be understood as arising from a *nervous* condition represented by vomiting or by a disturbance of the function of digestion, which had best be spoken of, until more is known of the subject, as *simple indigestion*.

NERVOUS (Vomiting).—Vomiting may arise from gastric or from intestinal irritation in many diseases, such as tubercular meningitis, from heat, cold, fright, and from other causes. Direct irritation, from foreign bodies, food, or otherwise, may produce a reflex form of vomiting. In these cases the cause, if possible, should be removed, and the stomach given a complete rest until the nervous disturbance has subsided. As a rule, no internal remedies are indicated in these cases, except an emetic where the vomiting arises from the reflex causes just described, or, if necessary, avage.

There is one form of vomiting, however, which is of such importance that it must be spoken of as a disease by itself. There is no name which can be given to it except that of *persistent vomiting*, as no single definite cause nor any pathological lesion has ever been proved to produce it. It has not even been shown that it is a primary disturbance of the stomach. In fact, in many cases it is possible that the source of irritation is entirely outside of the stomach, and perhaps connected with the great sympathetic ganglia, such as the solar plexus.

ETIOLOGY.—The inciting cause of the vomiting in most cases is obscure, but is evidently very varied. It does not seem to be produced especially by errors of diet, but, on the contrary, occurs in children whose diet has been most carefully regulated. Undue exposure to cold, fright, and excitement all appear to me to have sometimes an etiological influence on the disease. This form of vomiting may occur at any age. I have met with cases in young infants and throughout the whole period of childhood. The attacks may occur not only in delicate, nervous children, but also in those who are quite vigorous.

SYMPTOMS.—The attack is very apt to come on suddenly, the child being previously in good health and not having shown any digestive disturbance. The period over which the disease extends and the intervals of the vomiting during the attack vary considerably, but in those cases which have come under my notice they are somewhat as follows. The child, without any especial warning, begins to vomit, and at first the vomitus will simply be the remains of food which still happen to be in the stomach. It will continue to vomit quite regularly every fifteen or thirty minutes. This may last for ten or twelve hours; the intervals then grow longer, and sometimes the vomiting will cease for twelve or fifteen hours and then begin again. Occasionally a little mucus appears in the vomitus, but not to any great extent. As the disease progresses, a slight amount of bile usually appears in the vomitus. A very prominent symptom is thirst, the child crying continually for water, and vomiting it soon after it is taken. As a rule, the temperature in these cases is normal or subnormal. The pulse varies, but is very apt to be slow, sometimes intermittent, and may become weak. After the first twenty-four hours the child emaciates rapidly, looks very ill, and becomes apathetic.

Unless the disease is unwisely treated by endeavoring to introduce food or drugs into the stomach, it will usually prove to be self-limited, and will run its course in two or three days. In some cases the length of the attack is much shorter, being comprised within twenty-four hours, while in others it may last for many days. The recovery is often as sudden as was the onset of the disease. As soon as the vomiting has stopped, the appetite returns; there are no special symptoms of indigestion; the child takes its food well, and the emaciation disappears rapidly. Relapses occasionally take place.

DIAGNOSIS.—The diagnosis of *persistent vomiting* is often difficult, more on account of a lack of sufficient knowledge concerning the disease than from much evidence of the existence of the diseases which it is supposed to simulate. In these cases an examination of the abdomen should be made at once, including a rectal examination. This is necessary in order to exclude such sources of vomiting as intussusception and appendicitis. The absence of any marked increase in the temperature and a careful examination of the thorax will in most cases exclude the sudden onset of some pulmonary disease or of the acute infectious diseases. The disease which is most commonly suspected in these cases is tubercular meningitis. In some instances, after the disease has lasted for two or three days, the resemblance to tubercular meningitis may be quite striking; but if the whole course of the affection is taken into consideration, the diagnosis soon becomes clear. In persistent vomiting the face and general appearance of the child indicate nausea rather than the apathy which would be present in tubercular meningitis. The mind also, in contradistinction to what takes place in the latter disease, is clear, the child remaining quiet merely because it is exhausted. The great thirst which I have already mentioned as being present in persistent vomiting also aids materially in the differential diagnosis from tubercular meningitis. The sudden onset of the vomiting in a previously healthy child is quite different from the slow progress and the occasional vomiting of a cerebral type met with in tubercular meningitis.

After the first twenty-four hours, persistent vomiting is readily differentiated from attacks of simple indigestion, as where the vomiting arises from indigestion the stomach is speedily relieved, and the vomiting does not continually recur without apparent cause, as is the case where persistent vomiting is present.

Persistent vomiting is also very commonly diagnosticated as acute gastric catarrh, but in the latter disease the heightened temperature, coated tongue, pain, and tenderness in the epigastrium will, after the first twenty-four hours, allow us to differentiate the two diseases.

PROGNOSIS.—The prognosis of persistent vomiting varies according to the age of the individual affected. In young infants, especially in those whose vitality is weak, it may prove to be a very serious disease, from the exhaustion which invariably arises in the first twenty-four hours. The rule is that the younger the individual the more prostrating and serious is the disease. Even older children are at times so prostrated by the continuous vomiting that grave doubts are often entertained as to their ultimate recovery. In general, however, the prognosis in these cases is good, and, although I have met with a number of them, I have never seen the disease result in death.

TREATMENT.—The treatment of persistent vomiting is essentially starvation during the first twenty-four hours. The child should be kept perfectly quiet in a darkened room. If after twenty-four hours the vomiting still continues, or even before if there appears to be much exhaustion, or if the child is restless and sleepless and has an intermittent pulse, hydrate of chloral and bromide of potassium, dissolved in brandy and water, should be given by the rectum. These are intended to procure sleep and to stimulate the nervous centres. As a rule, however, the child is quiet, and sleeps in the intervals of the vomiting, and, as the disease usually attacks an infant or a child who has been perfectly well, cardiac weakness is not commonly shown in the first forty-eight hours. No food and no drugs should be given by the mouth. After forty-eight hours, small enemata of peptonized milk can be given, and when the disease appears to have run its course, as it often does in three or four days, small quantities of a carefully modified alkaline milk can be tried cautiously by the mouth. A mistake is usually made in the treatment of the disease in feeding by the mouth too early.

I shall speak of a few illustrative cases of this disease which have come under my notice, as a knowledge of them will be of great use to you in your practice.

The first (Case 407) was an infant, eight months old, strong and healthy, whose food had always been the milk of a wet-nurse. Without any previous symptoms the infant began to vomit, and continued to vomit every fifteen minutes for twelve hours. The intervals then became longer, and the vomiting ceased entirely on the third day of the attack. During the attack the infant emaciated rapidly, so that it looked as though it were in the last stages of some wasting disease. It lay perfectly quiet and slept in the intervals of the vomiting. Its mind was clear. Its temperature was subnormal, and its pulse weak and intermitting. It was treated by rectal enemata of brandy, peptonized milk, and bromide of potassium.

The infant had several attacks of this kind in each of the following years of its life until it was five or six years old, when it would sometimes go for six months or a year without an attack. As it grew older the attacks became less severe, and when it was ten years old they ceased entirely.

The next case (Case 408) was that of a girl, twenty-two months old, whom I saw in consultation with Dr. Joseph Stedman. She was perfectly well before the vomiting began. Her temperature was normal: the pulse was slightly accelerated at first, and later became slow and intermittent. During the first four days of the attack the vomiting was almost continuous, and she became so weak and exhausted on the fourth day that it was feared she might die suddenly. There were great restlessness, dilated pupils, throwing of the head backward, slow pulse, and normal respirations. The emaciation was rapid. The urine was scanty. On the fifth day, the vomiting having continued, she fell into a state of collapse, the pulse was hardly perceptible, her countenance was ghastly, and her extremities were cold. At one time after a severe attack of vomiting she became cyanotic, and was almost stifled by tenacious mucus. This, when vomited, appeared to invade the larynx, so that it seemed as though her life was saved a number of times by the prompt action of an experienced nurse. On the sixth day the vomiting grew less, and on the seventh day it ceased. She was not, however, able to be up and about until the eleventh day, and was not entirely well until the third week from the time that she was attacked. The treatment in this case was the same as in the previous one.

A third case, a boy (Case 409), nine years old, was seen by me in consultation with Dr. F. B. Harrington. This boy was attacked suddenly with vomiting as described in the previous cases. The duration of the attack was about two weeks. The prostration was extreme, and the boy's strength was supported solely by enemata, as at no time during the two weeks could anything be retained by the stomach.

These last two cases were unusually protracted.

Acute Gastric Indigestion (Acute Dyspepsia).—By indigestion we mean a disturbance of the gastric secretions interfering with the function of the stomach to such a degree as to cause morbid symptoms. Exactly what this disturbance is in infants and young children has not yet been clearly proved. The cause of acute indigestion in infants, and in almost every case in young children, is the food which is given to them. This is especially noticeable in the first year. The ages at which indigestion most frequently occurs in this period are, first, in the early days of life, when the equilibrium of the breast-milk has not been established; second, in the middle of the first year, when the breast-milk is so apt to be replaced or supplemented by some other food; and, third, at the end of the year, when entirely new articles of diet are usually given to the infant.

Symptoms.—The symptoms of acute indigestion are extreme pallor, nausea, eructations of gas, a general appearance of discomfort, due probably to the pain induced by the development of gas in the stomach, with its resulting distention, and finally vomiting. If the diet is exclusively of milk, the vomitus will usually contain large curds of the coagulated proteids. In connection with the gastric disturbance there is commonly constipation, although sometimes there may be a relaxed condition of the bowels. The fæcal discharges accompanying these attacks are of an abnormal color, usually a mixture of green, white, and yellow, and of sour odor. There is little or no fever. At times the symptoms are so severe that the infant looks as though it were going to die. In rare cases also reflex symptoms of a serious aspect may arise, such as I have already described when speaking of asthma dyspepticum (page 750).

Diagnosis.—Sometimes the diagnosis is obscured by the absence of vomiting, but the pallor and nausea are usually of sufficient prominence to allow us to decide that the seat of the disturbance is the stomach. An emetic, such as one-half to one teaspooonful of wine of ipecac, usually relieves the symptoms promptly and makes the diagnosis clear.

Treatment.—The treatment of acute indigestion is to empty the stomach, to give a mild laxative in order to clear away the undigested food, and to regulate the diet. The laxative may be one or two teaspoonfuls of castor oil, an eighth to a tenth of a grain of calomel for four or five doses, or a teaspoonful of liquid magnesia. If the food has been breast-milk, an analysis of the milk should be made at once, and the proper modification of the milk, according to the rules which I have already given you, should be carried out. If the infant is being fed on an improperly modified milk, or if improper food of any kind has been given to it, a recurrence of the attacks can easily be obviated by a modification of the elements of the food which seem to have produced the disturbance.

Thus, in a number of cases I have found that whenever the infant's food was modified so as to raise the percentage of the sugar above 5, acute indigestion followed. In like manner in certain cases the percentage of the fat had to be reduced to 3, or perhaps 2.5, and the proteids even as low as 0.45, for a number of weeks until the digestive function of the stomach became normal.

In older children the symptoms are similar to those which I have just described, and the diagnosis and treatment the same as in the infant, for there is no way by which an attack of acute indigestion can be so surely prevented from recurring as by at once placing the child for several days on an exclusive diet of a milk modified in such a way as to contain a percentage of from 2 to 5 of fat, 5 to 6 of sugar, 1 to 2 of proteids, and 10 of lime water.

CHRONIC GASTRIC INDIGESTION (Chronic Dyspepsia).—If the attacks of acute indigestion are allowed to occur frequently from lack of proper treatment, a subacute or chronic form of the disease develops.

SYMPTOMS.—In infants the symptoms of chronic indigestion are much less severe than those of the acute form. The infant is apt to vomit after taking its food, to be restless, fretful, and either to lose in weight or not to gain. Its sleep will be very much disturbed, apparently by pain from flatus. In chronic indigestion the bowels are apt to be constipated, but this is not always the case. The chronic indigestion of older children presents a somewhat different aspect. The temperature is at times somewhat heightened. The tongue is apt to be coated, and the breath to have an odor. These children do not vomit so frequently as do infants. They lose in weight, become fretful, and get tired easily.

TREATMENT.—I have seldom found the use of any especial drug to be of much benefit in these cases of chronic indigestion. In quite a number of cases of both acute and chronic indigestion, before any food is introduced into the stomach it is often wise first to wash out the stomach thoroughly (lavage). This procedure is especially indicated if the indigestion has produced continuous vomiting.

The technique of washing out the stomach is very simple. A soft rubber catheter with a double eye, No. 21 French scale, as recommended by Dr. Holt for infants under six months, and No. 25 for older children, is attached by means of a piece of glass tubing 7.5 cm. (3 inches) long to another rubber tube which is 50.5 cm. (20 inches) long attached to a funnel, preferably of hard rubber, and capable of holding from 90 to 120 c.c. (3 or 4 ounces). The infant is seated upright in the nurse's lap, with its head inclined forward and resting on the nurse's arm. Its arms are controlled by a towel pinned around them. The catheter, having been wet with warm water, is easily passed over the base of the tongue into the stomach. As there is often considerable gas in the stomach, the funnel should be raised as high as possible above the infant's head, in order that the gas may pass out from the stomach. From 90 to 120 c.c. (3 or 4 ounces) of sterilized water should be poured into the stomach by means of the funnel. The funnel is then depressed below the level of the stomach, and the gastric contents will in this way be siphoned out. As the curds are often too large to pass through the eye of the catheter, a number of washings will often be necessary to break them up. By washing out the stomach not only are the irritating substances which are producing the indigestion removed, and the mucous lining of the stomach left free to recover its normal condition, but it is also possible to have a chemical examination of the contents made. Clinically, however, the latter is not necessary, although it is of great interest physiologically. No food should be introduced into the stomach for at least two hours after the washing. The washing of the stomach is almost entirely free from danger, and, in addition to being an important part of the treatment of indigestion, is often of great use where poisonous substances have been swallowed.

This method of treating disturbances of the stomach is more valuable in young infants than in older children, because the latter resist so vigorously that the remedy is often of more harm than good. The tube can, however, usually, even in older children, be introduced by aid of the ordinary gag which is used for intubation. Two assistants are usually necessary in introducing the tube in older children, while in infants one assistant is sufficient. In some cases it is found necessary to introduce the tube through the nose. The tube should be passed into the throat rapidly, since the gagging and vomiting occur chiefly when the tube touches the pharynx. There is usually an escape of gas or gastric contents as soon as the tube enters the stomach.

When the inflow of water through the tube is shown to be too rapid, by the fact that the infant holds its breath too long, or by its crying, vomiting, or coughing continuously, the flow should be stopped for a short time. Care must also be taken not to introduce the catheter too far into the stomach, as it may bend on itself and interfere with the flow of the returning water and gastric contents. If the gastric contents are expelled along the side of the tube rather than through it, the tube should be withdrawn until the vomiting has ceased. There seems to be no danger of passing the tube into the larynx, or of perforating the stomach with it.

Lavage is contra-indicated where there is cardiac disease or any severe pulmonary disturbance, and when the introduction of the catheter continues to excite vomiting it should be used with extreme caution. The fact that the infant is in a feeble condition is not a contra-indication to this treatment.

In connection with lavage it is well to speak of forced feeding (gavage) in the treatment of infants and young children. In cases of acute and chronic indigestion, and also where a catarrhal condition of the stomach is present, the infants at times refuse to take any food whatever. This does not occur merely where the disturbance is in the stomach: I have frequently met with it in severe cases of all kinds of disease. In a number of instances, where the infants would probably have died of starvation had not gavage been employed, this means of providing for their nourshment has been very successful. Forced feeding may sometimes have to be employed for a number of days, and even weeks, before the child will of itself swallow again.

The technique of gavage is similar to that of lavage. The same apparatus is employed, but the child should be placed flat on its back in bed, and its head held by an assistant. The catheter should be passed into the stomach rapidly, the funnel raised up in the air for a few minutes in order that the gas may escape, and the amount of food adapted to the age of the child should then be poured into the funnel. As the last of the food disappears from the funnel, the catheter is pinched tightly and quickly withdrawn. This precaution is important, in order that the pharynx shall not be irritated either by the slow withdrawal of the catheter or by the trickling of the remains of the fluid, as vomiting may in this way be excited.

One of the advantages which has resulted from the use of the stomach-ube is the knowledge we have acquired of the time which the food remains n the stomach at different ages. Thus, it has been found that during the arly weeks of life the stomach is nearly emptied in an hour, while in older nfants two hours are required for the same process. This knowledge is specially valuable when we are regulating the intervals of feeding in premature infants, and in infants during the first six months of life. These ntervals I have already given in my lectures on Premature Infants and on Feeding.

Where other means can be employed, they are preferable to the stomch-tube. I have found in most instances where infants or children refuse o take their food that the simplest way of forcing it upon them is to pinion he arms with a towel and have the nurse hold the child half reclining n her lap. Sometimes an assistant is needed to hold the head, but this is ften unnecessary. Simply pressing the child's nostrils with the thumb and nger will cause it to open its mouth, and the food can then be poured in with a spoon, or, as I have done in a number of cases, by means of a dropper with a large end. A child two and one-half years old, who has recently een under my care, for several weeks would not take any food without eing forced to do so. Although this child was very ill with pneumonia, nvolving both lungs, it was fed every two or three hours, night and day, y this method. After the first two or three feedings it did not resist, and he nose did not have to be pinched, all that was necessary being to threaten o do so. 120 c.c. (4 ounces) of milk were, after a little practice, introuced by means of the dropper into the child's stomach in five or six minutes.

I have found that the most speedy cure of chronic indigestion is to give he child a carefully modified alkaline milk. In some cases it will be ecessary to reduce the fat or sugar, in others the proteids, but in every ase, as soon as it is determined which of these elements in full strength does not suit the individual digestion, an improvement in the symptoms will soon follow the reduction of the percentage of that element. After the indigestion has been relieved by this means, other articles of diet adapted to the age of the child can gradually be added.

In addition to the direct treatment of the stomach, the intestinal disturbance which almost always accompanies the gastric indigestion should be relieved by occasionally giving a dose of some mild laxative, preferably one of the salts of magnesia. This latter treatment is indicated not only for children, but for young infants, because, when there is gastric indigestion, the undigested food which passes into the duodenum is a prolific source of intestinal disturbance. This, by adding to the discomfort of the child, weakens it, and tends to prolong the gastric indigestion.

ORGANIC.—The organic affections of the stomach may be divided into non-inflammatory and inflammatory. They are, in my experience, very rare in comparison with the functional diseases which I have just described.

NON-INFLAMMATORY.—The non-inflammatory conditions of the stomach comprise a diminution in the size of the organ, mechanical dilatation, ulcers, and new growths.

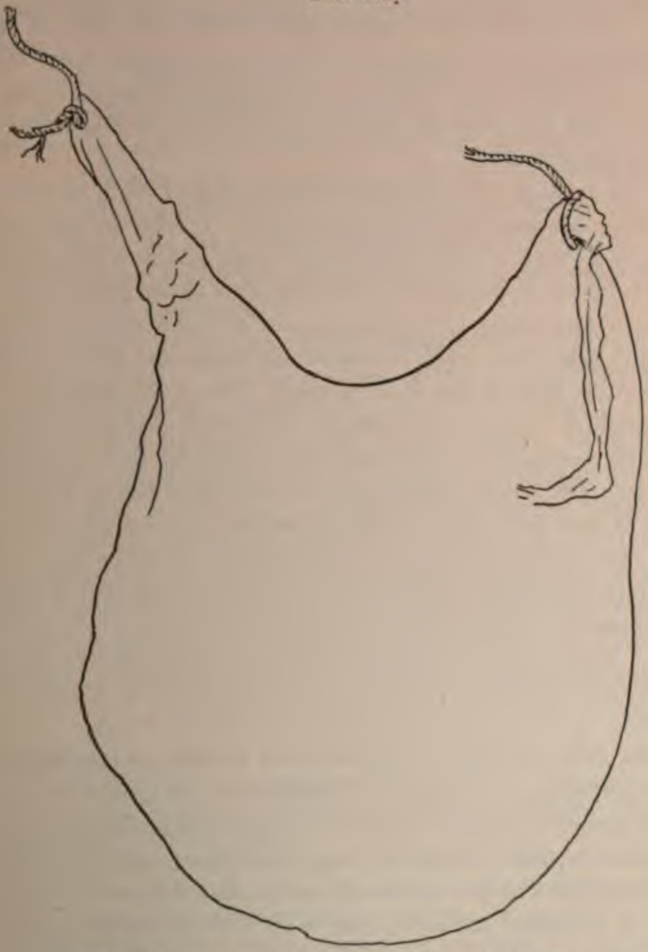
Contraction of the Stomach.—In certain cases the gastric capacity of the stomach is decidedly diminished. This diminution in the size, as a rule, depends upon a lack of use, such as occurs in infantile atrophy. Sufficient food to fill the stomach is not taken, and in this way the stomach is not called upon to perform its normal work. In cases, also, where there is continuous vomiting, this same lack of use may produce a diminution in the size of the stomach. These cases are of pathological rather than of clinical interest, as they can seldom be diagnosticated, and their treatment is essentially that of the special disease to which they are secondary.

Dilatation of the Stomach.—Dilatation of the stomach is rather more common in infancy than in older children. It may rarely arise from some malformation, such as a stenosis of the pylorus, but in most cases is the result of errors in feeding. It is more apt to occur where the infant is not nursed, unless especial care is taken to give the infant the quantity of food which is adapted to its age and gastric capacity. When the infant is nursed, the breast seems to provide the amount of food which is suitable. Dilatation from errors in feeding may be caused by the fact that the food is not adapted, either in quality or in quantity, to the age of the individual infant. Where the quality is at fault, the nutrition of the tissues of the stomach is interfered with, and its walls become weak, and are thus more easily distended by the gas which results from the abnormal changes in the food. In this way dilatation occurs. This class of cases is notably represented in the disease rhachitis, where dilatation of the stomach takes place very readily.

Where the quantity of the food is not properly adapted to the size of the stomach, dilatation can take place in even a healthy infant. I have already demonstrated to you, in my lectures on Development and Feeding, he size of the stomach at different ages, and the amount of food which it ormally holds. I shall, therefore, not repeat what I explained to you o fully at that time, but shall merely impress upon you the great importance of carefully regulating the amount of food which is given at each eeding during the first year of life.

PATHOLOGY.—The pathological condition which exists in cases of astric dilatation is well represented in this stomach (Fig. 108).

FIG. 108.



Dilated stomach. Rhachitic infant, 7 months old. (Natural size.)

It was taken from an artificially fed rhachitic infant (Case 410), seven months old, ho died under my care at the Boston City Hospital. The gastric capacity in this case as 300 c.c. (10 ounces), which corresponds to the gastric capacity of an infant twelve onths old. You will notice the shape of the stomach, which is very significant of the mptoms I shall presently describe.

You see that the lesser curvature is not much altered, while the greater curvature is ry much increased. The pathological condition of the tissues is such as would be expected from general malnutrition. In such diseases as rhachitis there is a stretching of the muscular fibres, as well as an atrophied condition of the entire gastric walls.

SYMPTOMS.—The symptoms of dilatation of the stomach are essentially those of chronic indigestion. Vomiting is quite frequent, and continues until the stomach has been entirely emptied, when a period of relief comes, to last until fresh irritation arises from another supply of food. Abdominal pain, flatulence, and general discomfort are prominent symptoms. Emaciation and rapid loss in weight also occur. In some cases, in young infants, convulsions may arise, apparently due to the reflex disturbance which is produced. There are usually considerable thirst and loss of appetite. When the dilatation is of a high grade, the vomiting may occur only after considerable intervals,—twenty-four to forty-eight hours,—during which time the food does not pass out through the pyloric orifice to any degree, but collects in the stomach.

If you will look at this dilated stomach (Fig. 108), you will readily understand the mechanism of these symptoms. Under normal conditions the stomach, as I have already shown you (page 85), is somewhat tubular in shape and oblique in position. The food thus easily passes through the cardiac to the pyloric orifice. In dilatation of the stomach, on the contrary, the greater curvature is so much increased and depressed below the level of the pyloric orifice that a pouch is formed. The food, collecting in this pouch as though it were at the bottom of a well, has to be practically pumped, by the contraction of the muscular walls, up to and through the pyloric orifice. The already weakened stomach thus has to perform work for which it is not fitted, and finally is relieved by spasmodic vomiting. When only the small amount of food adapted to their normal gastric capacity is given to young infants whose stomachs are dilated, a large space of empty stomach is left above the level of the liquid which has entered the stomach. This creates a feeling of emptiness and general discomfort, so that the infant appears to be hungry when, in fact, it is only suffering from the feeling of incomplete filling of the stomach.

DIAGNOSIS.—On inspection the abdomen is seen to be distended and tense, and on percussion to be highly tympanitic in its upper part. Succussion is not an especially valuable diagnostic sign in dilatation of the stomach. Succussion is so frequently found in many conditions, and is so likely to be confounded with that which occurs in the colon, that it cannot be relied upon. The outlines of a normal stomach when somewhat distended vary so much in infancy that the results of percussion are often very misleading. When, however, the tympanitic resonance is found to extend below the line of the umbilicus, we may suspect that we are dealing with gastric dilatation. In infancy the cardiac end of the stomach is so slightly developed that any great increase in the area of gastric percussion to the left is an important aid in making the diagnosis.

The differential diagnosis is to be made chiefly from dilatation of the colon. In many cases when the colon is dilated it is impossible to determine whether the stomach is also dilated, since under these circumstances the colon can almost completely cover a largely dilated stomach. In older children, in cases where the diagnosis is uncertain I have found a valuable means of determining the presence of dilatation to be artificial distention. This can be done without harm or discomfort to the child by giving it first one half of a seidlitz powder and then the other half, so as to allow the chemical combination to take place in the stomach. Except in certain cases where it is very necessary to determine whether the stomach is really dilated, this is not a procedure which I am in the habit of adopting. In most cases in infants and children clinically satisfactory results can be obtained by percussion.

PROGNOSIS.—If the dilatation is due to congenital stenosis of the pylorus the prognosis is very unfavorable. In other cases the prognosis depends upon whether the condition arises from improper amounts of food or from some disease, such as rhachitis. In the former class the prognosis is good, and the stomach under a proper regulation of the diet soon resumes its natural size. In the second class it is not so good, and, as a rule, the stomach will remain more or less distended until the disease which causes the dilatation has been cured.

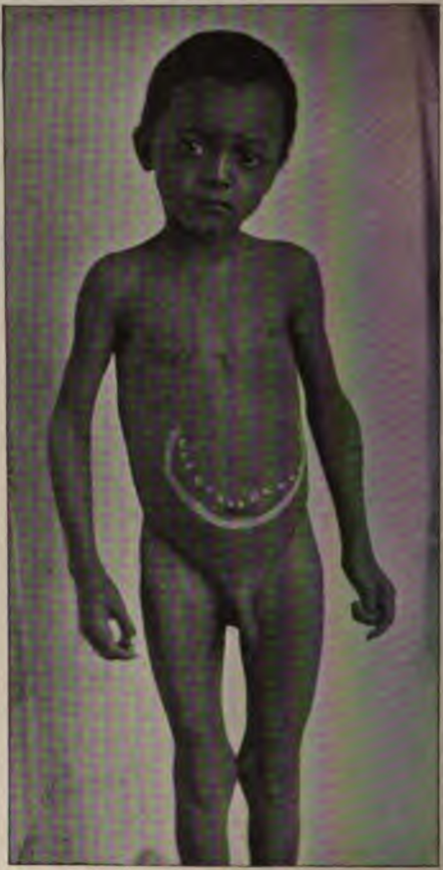
TREATMENT.—If the case is an obstinate one, lavage is an important part of the treatment. In many cases, however, good results are obtained simply by regulating the quality and quantity of the food. In both infants and children carefully modified milk is the food from which the best results are obtained. When the food is first given in the proper amount it will, as I have just told you, not fill the stomach nor satisfy the demands of the infant. Under these circumstances the infant will be very restless, and will often cry almost continuously from the time of one feeding until the next. You must impress upon the nurse that these signs of discomfort are liable to last for a number of days, until the stomach has more nearly resumed its normal size, and that an additional supply of food must not be given to it.

I shall report to you the case of an infant (Case 411), four months old, which illustrates dilatation of the stomach as it occurs in the first year of life. This infant, a male, was well and strong at birth. It was not nursed, but was fed on a mixture of milk, cream, and water. It was an unusually vigorous infant, and is reported to have never been satisfied with the small quantities of food suitable to its age. When it was three weeks old it was given 150 to 180 c.c. (5 or 6 ounces) at each meal. Somewhat later, in its second and third months, it gradually developed symptoms of indigestion, and when I was called to see it was in a very serious condition. It had been having frequent and prolonged convulsions. At times when it was in the convulsions it would fall into a state of collapse, the pallor of its face would be extreme, and it would look as though it were dying. On examination, nothing abnormal was found in the thorax. The entire abdomen was found to be distended, especially in the upper part, where the gastric tympany was pronounced and easily marked out by percussion. The percussion showed the stomach to be dilated, and to extend below the line of the umbilicus and far to the left of the median line.

The infant was given small amounts of food at frequent intervals. For the first two or three days it cried and screamed for more food, but the convulsions ceased, its general condition improved, and by the end of the week the distention of the stomach had subsided very markedly and the infant had become tranquil. From this time there was no recurrence of the symptoms.

I have here in the wards to show you a colored boy (Case 412), six years old.

CASE 412.



Dilatation of stomach. Age, 6 years.

This child is markedly rhachitic. He is reported to have been in fair health, though delicate, until one month ago, when he began to have persistent vomiting. He has lost greatly in weight, has been very restless at night, and has had continual borborygmi.

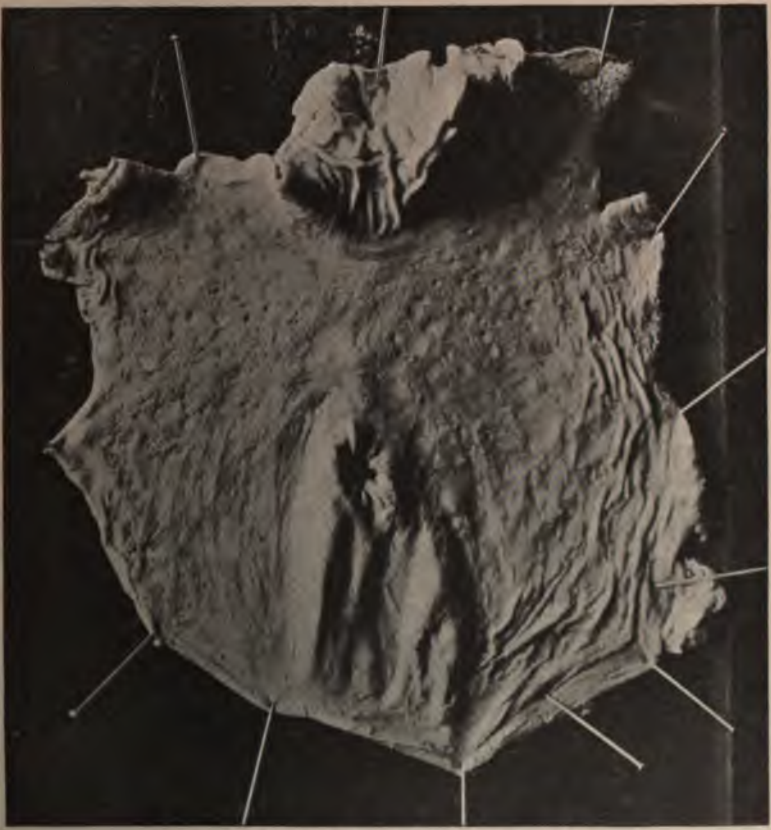
Physical examination shows marked abdominal enlargement. On percussion the gastric tympany is found to extend downward as far as the umbilicus, 7.8 cm. (3 inches) to the right of the median line and 10.4 cm. (4 inches) to the left. I have marked this percussion line, which represents the greater curvature of the stomach, with spots. As the resonance of the colon is also exaggerated in this case, and as its differentiation from that of the stomach is somewhat difficult, because it evidently overlaps the lower border of the stomach, I shall endeavor to eliminate this obstacle to diagnosis by mechanical means. You see that the child readily takes half of this seidlitz powder which has been dissolved in water. The other half, which has also been dissolved in water, is next swallowed. As the combination of the two salts takes place in the stomach you can easily hear with the stethoscope the chemical action which is resulting. The child shows no signs of discomfort, and says that he does not feel any pain or any more tenderness in the epigastrium than before the powders were taken. The outline of the upper part of the stomach can now be fairly well seen, and on percussion the line of the greater curvature is found to be 2.5 cm. (1 inch) below the ne of the umbilicus, the colon having been pushed out of the way by the distended omach. I have indicated the line of greater curvature by a broad white line, and in this ay we determine that the stomach is really dilated.

(Subsequent history.) In this case it was not found necessary to wash out the stomach ore than once or twice, for as soon as small amounts of food were given at frequent inter- ls the vomiting ceased and the stomach gradually resumed its normal size. At the end two months the child left the hospital free from any abnormal gastric symptoms.

Ulcers.—Ulcers of the stomach in infancy and early childhood are very re, but cases have been reported. They may be non-inflammatory or flammatory, the distinction between the two often being very difficult to ake.

Through the kindness of Dr. Northrup I am enabled to report to you ch a case, occurring in a female one year old who was under his care. I so have here the stomach (Fig. 109) to show you.

FIG. 109.



Follicular ulceration of stomach. Female, 1 year old.

The infant (Case 413) was under treatment for one month. It had vomiting and diar- rea. During the first week that it was in the hospital its temperature varied from 38.8° 39.4° C. (102° to 103° F.), after that being normal or subnormal. The respirations ried from 40 to 50, and the pulse from 120 to 140. In the second week it began to refuse high. The first class, or afebrile form, is by far the more common, and is what is usually spoken of as gastritis catarrhalis. It is subacute rather than acute. According to my experience, it is difficult and almost impossible to state definitely the symptoms of the afebrile form of acute gastric catarrh. They so nearly approach those which occur in cases of indigestion, where we believe no gross pathological condition exists, that we should always be guarded in our use of the word catarrh. Pain is so common a symptom in all gastric disturbances, the existence of tenderness is so difficult to determine in infants and young children, and a hypersecretion of mucus is so often known to occur without the presence of an inflammatory condition, that there does not seem to be any one symptom upon which we can rely. The general picture of the disease which is supposed to represent acute gastric catarrh is that of fever, nausea, vomiting of food mixed with mucus and at times of mucus alone, and a sense of tenderness, uneasiness, and discomfort in the epigastrium. There may be frontal headache, a rather swollen, coated tongue of somewhat glassy appearance, and often a slight follicular pharyngitis. There is loss of appetite, with, at times, hiccough and eructations of gas. The bowels are usually constipated at first, but after three or four days diarrhœa may result.

Where the infant or child seems prostrated for a few days, and sick beyond what would naturally be expected in an acute attack of indigestion, and where, in combination with a somewhat heightened temperature, frequent vomiting of mucus occurs, we are justified in supposing that we are dealing with a catarrhal condition.

TREATMENT.—The treatment of cases of this kind is the same as that which I have described in speaking of indigestion. Food should be withheld from the stomach for many hours, for, as I have just told you in speaking of the pathological conditions which occur in gastritis catarrhalis, the food remains so long in the stomach that a fresh supply at short intervals will act as an additional source of irritation. In those cases which do not respond readily to long intervals of rest and to feeding with small quantities of a modified alkaline milk, lavage will prove of value. Much judgment should be used as to the time when the food is to be increased in strength, for unless great precautions are taken relapses will frequently occur, and as a result the disease may finally become chronic. After convalescence has been established the child will begin to gain in weight. Some simple tonic, such as nux vomica, is usually indicated for a week or ten days until the child has recovered its strength. During the beginning of the attack, when food is being withheld, if the child is made very restless by extreme thirst, teaspoonful doses of iced soda water can be given, but with caution and as seldom as possible. The second or febrile form of acute gastric catarrh is rare, but is of much more serious import than that of which I have just spoken. It is characterized by high fever, 39.4°, 40°, 40.5° C. (103°, 104°, 105° F.). The invasion is very acute. It may last for two or three weeks and show severe and alarming symptoms. There may be active vomiting, delirium, and sopor in the beginning, so that it will be impossible to determine whether or not one of the other acute febrile diseases is developing. The characteristic symptoms of gastric catarrh develop later, and then the differential diagnosis is easily made. Instead of the cessation of the vomiting in the first twenty-four or forty-eight hours, as in scarlet fever, and of the continuance of the cerebral symptoms, as in meningitis, or of the development of pulmonary symptoms, as in pneumonia, the vomiting continues, though not quite so frequent as in the beginning, the mind becomes clear, and the symptoms point to the abdomen rather than to the head or the thorax. The onset of pneumonia in some cases, though in my experience rarely, simulates this disease. The pulse is rather irregular. There is usually constipation at first, followed by diarrhœa.

The prognosis is good, except in very debilitated children.

The child should be placed in a darkened room, soothing applications applied to the abdomen, and small quantities of iced soda-water given. The food should be given as I have just described in the other form of gastritis catarrhalis; that is, in very limited quantity and at long intervals. If there is much exhaustion, stimulants are indicated.

Gastritis Corrosiva Acuta.—Corrosive lesions of the mucous membrane of the stomach are at times produced by swallowing irritants, such as arsenic, carbolic acid, and caustic fluids. In these cases the lesions are usually found on the summits of the rugæ.

The treatment is by washing out the stomach with large quantities of water, administering the proper antidote, and feeding the child on a liquid diet so modified as to be as little irritating as possible to the injured mucous membrane.

Gastritis Pseudo-membranosa.—The membranous form of gastritis is extremely rare in infancy and childhood. Cases have been reported, notably those of Wollstein. In these cases the congestion of the rugæ was very marked, and along the greater curvature extended over an area of a number of inches. There was a thick grayish-green membrane, with some erosions. The gastric walls were much thickened.

The *symptoms* of gastric disturbance in these cases are often almost entirely absent, but there may be vomiting, pain, and tenderness in the epigastric region, and insatiable thirst. A pathognomonic symptom would be the vomiting of shreds of membrane, with or without an admixture of blood. This symptom is, however, extremely rare, because the membrane is usually adherent, so that a differential diagnosis is often impossible.

The *prognosis* is very unfavorable, and the *treatment* is purely symptomatic.

Gastritis Catarrhalis Chronica (Chronic Gastric Catarrh).—Chronic gastric catarrh, as I have already stated, is not usually met with in infancy, but occurs in later childhood. It is especially common in the summer months, and is generally the result of neglect or of improper treatment of the acute form of the disease.

PATHOLOGY.—The pathological condition which is found in chronic gastric catarrh is the result of long-continued hyperæmia. There is often a slaty discoloration of the mucous membrane, with cellular infiltration of the submucosa. In addition to this there is usually found a considerable quantity of tough mucus.

SYMPTOMS.—The symptoms are not so clearly defined as in the acute form of the disease, but are variable and of a rather sluggish type. The tongue is apt to be much coated and the breath to have a disagreeable odor. There is considerable abdominal distention after meals, so that the children complain that their clothes feel uncomfortable.

Frontal headache is apt to occur. The children gradually grow thin and anæmic. They vomit at irregular intervals, and are usually constipated. There is often a slight cough, and the symptoms, so far as the stomach is concerned, may form so small a part of the general picture of the disease that the child is not infrequently brought to the physician on account of its cough and because it is supposed to have some pulmonary affection.

PROGNOSIS.—Although the disease is often somewhat intractable, the prognosis under proper treatment is good. It may last for three or four months; but in many cases which are usually considered chronic gastric catarrh it has seemed to me there is no organic lesion, but that the disease is functional in its character, and the prognosis consequently very good.

TREATMENT.—It is often necessary in these cases to precede the treatment by carefully washing out the stomach. We must remember, however, that a considerable quantity of mucus may be in the stomach which cannot be removed by washing, so that if the symptoms continue after one or two washings, even though no mucus is returned by the tube, we should repeat this treatment from time to time. The diet should be an alkaline modified milk, with a low percentage of proteids, if necessary peptonized, and a moderate percentage of fat and sugar. The percentages of the different elements should be increased as improvement in the gastric symptoms takes place, and later broths and milk can be tried. Symptomatically in certain cases pepsin, dilute hydrochloric acid, and bismuth are occasionally indicated. A valuable tonic in the after-treatment of these cases is nux vomica.

This specimen, which is numbered 16 (Plate III., facing page 112), is what is usually spoken of as clay-colored. This clay color may be due to a diminution in the amount of bile which enters the intestine, or to undigested fat. This color is abnormal, and is usually met with in intestinal diseases of a subacute or a chronic type. It does not necessarily indicate a serious condition, however, as even a small plug of mucus may interfere with the flow of bile into the duodenum.

This specimen, which is numbered 17, is the light green color, which may be simply a change that has taken place after the fæces have been passed, and which often is not significant of any especial pathological condition. It may, however, show that the changes which have taken place in the food during its passage through the intestine have not been entirely normal. It is the least important of the changes which take place in the color of the intestinal contents. The colors in these next two specimens, numbered 18 and 19, are what may be seen in a more serious disturbance of the enteric tract. These colors may appear in any of the intestinal diseases which are accompanied by diarrhœa, but are significant of no especial disease. They are merely to be considered pathological in contradistinction to the normal colors in these other specimens, 3, 4, 6, 7, 8, 9, and the beginning abnormal condition represented in 17.

Besides these shades of green there are a great many varieties of color produced by the mixture of green, yellow, white, and brown. These are valuable merely as instructing us whether we are dealing with a normal or an abnormal condition of the intestinal contents, and, as I have already told you in my general remarks on diagnosis, are not significant of any one disease, either functional or organic. Much variety in the color also arises from the admixture of blood, mucus, and shreds of membrane. In this connection it is well to remember that the yellowish-white lumps seen in undigested fæces are often made up of fat as well as of proteid material.

The color of the intestinal contents may also be changed by the administration of various drugs, such as iron, which causes a more or less black color. Bismuth gives the colors which you see in these three specimens numbered 12, 13, and 14. Number 12 is the color which was produced by giving to an infant 0.18 gramme (3 grains) of bismuth every two hours for six doses; number 13, where 0.24 gramme (4 grains) of bismuth was given every two hours for six doses; and number 14, where the latter dose had been omitted for twenty-four hours. The size of the dose and the intervals between its administration will of course produce different shades of color.

Where the solids of the intestinal contents are much reduced in proportion to the serum, as in cases of acute and frequent diarrhœa, the discharges become more and more fluid, and sometimes almost entirely lose their color and look like water.

CONSISTENCY.—In the first year of life, or while the infant is having only milk for its food, the consistency of the fæcal discharges is intermediate between solid and fluid, and the discharge, as a rule, is smooth and free from lumps. As the infant begins to take other forms of food and a mixed diet, the fæcal discharges gradually become more solid. The consistency of the fæcal discharge is abnormal when it becomes liquid, as in diarrhœa, or when it is too solid, as in constipation.

COMPOSITION.—In addition to the various substances which make up the food which enters the intestine, the fæcal discharges contain bile, mucus, epithelial remains, and many bacteria. In diseased conditions they may also contain certain morbid elements, such as blood, pus, and membrane. In intestinal diseases of both an acute and a chronic type the mucus may be very largely increased, but it cannot be considered to be especially characteristic of an inflammatory condition, as the secretion of mucus apparently may be very much increased in purely functional conditions. The bacteria are very numerous and of many varieties, but in most cases the detection of any especial form of these organisms does not aid us in diagnosticating the especial disease. Notable exceptions to this statement are where one finds the typhoid bacillus, the comma bacillus, and the amœba coli.

ODOR.—While in the normal fæcal discharges of infants fed entirely on milk the odor is comparatively slight, it becomes much stronger as other articles of food, either of a starchy or of a proteid nature, are added to it. Where an abnormal condition exists, various changes take place, as in acid fermentation, where the odor is sour, and in albuminous decomposition, where the odor is very foul. Although these conditions can scarcely as yet be considered of great diagnostic importance, they are sufficiently so for us to make use of them in the diagnosis and treatment of intestinal diseases. Thus, where acid fermentation is supposed to be present, a reduction in the percentage of the sugar, and perhaps of the fat, is indicated, while where albuminous decomposition is suspected a reduction of the proteids in the food is called for.

AMOUNT.—In estimating the amount of the fæcal discharges we must consider the total amount in twenty-four hours, and not the large or small amount which may occur at one movement. The total amount in twenty-four hours is of much importance in both the acute and the chronic diseases of the intestine. In the acute diseases, the more frequent the diarrhœa and the larger the amount the greater is the exhaustion and the worse is the prognosis. In some chronic diseases the total amount of fæcal discharges may be very large. In these cases the larger the total amount the less has been the absorption and the worse is the prognosis, for this condition is an indication that the child is being starved from a lack of power to absorb the food which has been given to it.

FUNCTIONAL.—The functional diseases of the intestine may be classed as acute, chronic, and eliminative.

ACUTE (Simple Diarrhœa).—The acute functional disturbances of the intestine may be of nervous origin, or they may arise from intestinal indigestion.

Nervous.—In certain infants and children whose nervous system is easily affected exaggerated peristalsis causing diarrhœa may arise from a number of causes without any known lesions, fever, or gastric disease. Among these causes may be cited heat, cold, and fright. In like manner in these individuals foreign bodies, food or otherwise, may by simple reflex irritation cause such a nervous disturbance as to produce diarrhœa. In these cases either the small or the large intestine, or both, may be affected, and, so far as we know, the mucous membrane is either normal or simply hyperæmic. There is more or less serous exudation. These cases are rare in comparison with the other forms of diarrhœa, such as those which are caused by bacteria, and in them intestinal decomposition and intestinal inflammation are not present primarily.

SYMPTOMS.—The symptoms of simple diarrhœa are very apt to appear suddenly. There is usually abdominal pain, not, as a rule, of great intensity. At first there are two or three rather liquid yellowish-brown discharges occurring at intervals of perhaps one-half or one hour, and often accompanied by considerable flatus. There is a certain amount of restlessness, pallor, and exhaustion. Vomiting is rare. The temperature, as a rule, is not raised, or is raised very slightly. The pulse is rather weak and somewhat quickened. The number of the discharges may be eight, ten, or twelve in the twenty-four hours, and these soon become watery and of a lighter color, but are seldom green. The odor is somewhat increased, but not excessively. These symptoms, unless they are exaggerated by improper food or by bad treatment, usually disappear in a few days.

TREATMENT.—If there is a known cause, such as some intestinal irritant, a dose of castor oil or calomel may be given, but, as a rule, this is not necessary. The child should be kept in bed. Food should be withheld for some hours. A few drops of tinctura opii camphorata and hot applications to the abdomen are indicated for pain. If there is a tendency for the diarrhœa to continue, the subnitrate of bismuth may be given, and in some cases where there are signs of exhaustion a stimulant may be needed. For several days the diet should be simply milk heated for twenty minutes at 75° C. (167° F.) and containing ten per cent. of lime water.

I must impress upon you that these simple diarrhœas, especially in hot weather, should never be allowed to continue, as they render the intestine vulnerable to the more serious diseases, which may at any moment gain an entrance in this way.

Tubular.—In addition to these more common intestinal affections of nervous origin is one that is called *tubular*. This disease is so rare before the age of puberty that it need only be alluded to. It is a condition of the mucous membrane of any part of the intestine in which an exudation of mucus takes place in such a way that masses closely simulating a membrane may form on the surface. When discharged through the rectum they are sometimes found to have formed a cast of the intestine. These masses are mostly made up of mucus, and may occur in shreds of greater or less extent as well as in the tubular form.

The disease is supposed to be of nervous origin. The symptoms are pain, tenderness, and tenesmus. The temperature is usually normal.

The *prognosis*, as a rule, is good, although in some cases the disease may be much prolonged.

The *treatment* is to be directed essentially to improving the general health and the nervous condition, the local treatment being merely symptomatic.

Indigestion.—Disturbances arising from intestinal indigestion in most cases may be located in the duodenum. It has yet to be proved that any pathological lesion is present in these cases, and for the present they must be looked upon as functional.

These cases vary in their symptoms with the individual and according to the part of the duodenum which is most involved. In one set of cases the disturbance of digestion is shown simply by the increased peristalsis, such as I have just described in the nervous cases, but here the evidence points to an undigested condition of improper articles of food which have been given and which appear in the discharges.

The treatment of this class of cases is very simple, and consists in first giving a laxative and then regulating the diet according to the age of the individual.

What is usually spoken of as a "bilious attack" represents another class of cases. This condition is very rare in infancy, and usually occurs in middle and later childhood. In these cases, in addition to the increased peristalsis and evidence of undigested food, there are frequently icterus and vomiting of bile. In addition to these symptoms there may be headache and excessive nausea. The icterus is usually slight in degree, but often is marked and is noticeable in the conjunctivæ and in the urine. Here is a specimen (Plate III., facing page 112, No. 11) which I have numbered 11, and which shows the staining of bile on the napkin of an infant during an acute attack of indigestion involving the duodenum and accompanied by icterus. The temperature in these cases is usually slightly raised for a few days and then becomes subnormal. In a certain number of cases the fæcal discharges become clay-colored. This color is often produced mechanically, as I have already explained.

Although the symptoms in these acute attacks may often appear quite serious, the prognosis is always good. In some individuals they are liable to recur even when the diet and the general health are well attended to.

In the treatment of this class of duodenal disturbances we must consider that the fats in the food are in all probability especially liable to prolong the disease by not being properly digested so long as the function of the duodenum is involved. We should, therefore, in treating these cases, lessen the amount of fat given in the food. I have found that the treatment which most speedily shortens the attack is (1) total restriction from food or twelve hours, with the administration of small quantities of cold, sterilzed water if the thirst is excessive, and (2) the administration of small quantities of milk modified as in this prescription (Prescription 77):

PRESCRIPTION 77.

Fat	0.16
Sugar	6.00
Proteids	3.00
Lime water	10.00

The mixture to be heated to 75° C. (167° F.) for twenty minutes; from 120 to 180 c.c. (from 4 to 6 ounces), according to the age, to be given every three hours.

Under this treatment the icterus usually passes away in a few days, and the child can then soon be given its ordinary food.

CHRONIC.—The chronic functional diseases of the intestine may be the result of acute nervous disturbances, or they may arise from a number of prolonged attacks of acute indigestion. Incontinence of fæces and constipation are also forms of chronic functional intestinal disturbances. Under this same heading we can class infantile atrophy.

Nervous.—In the chronic form of nervous functional intestinal disturbance either the small or the large intestine may be affected, and, as I have already stated in describing the acute form, the condition of the mucous membrane, so far as we know, is either normal or hyperæmic. The causes are the same as in the acute form. This class of cases is not especially common, as they are merely a prolongation of the symptoms which I have already sufficiently described in speaking of the acute cases. The treatment of these cases is essentially with stimulants and care of the general health.

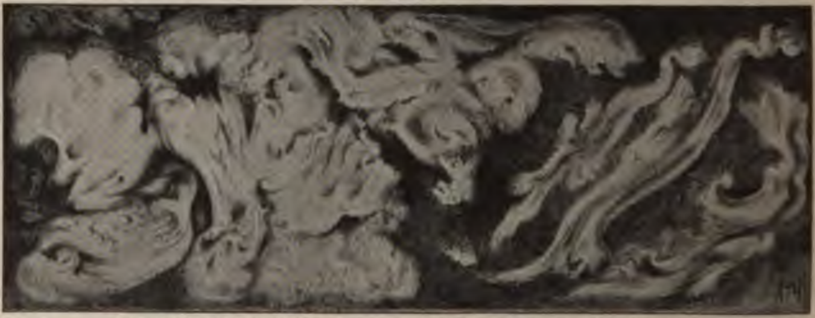
Indigestion.—The chronic form of functional intestinal indigestion plays an important part in intestinal diseases, especially when it is located in the duodenum. Chronic indigestion of the duodenum constitutes a disease of itself, and is one of the most difficult to cure which we meet with. It has usually been spoken of under the names of chronic gastro-duodenal catarrh and mucous disease. We at present, however, have no proof that either a catarrhal or any other pathological lesion of the mucous membrane is present in these cases, and the weight of evidence is in favor of the view that the disease is purely functional.

ETIOLOGY.—The etiology of chronic duodenal indigestion is in many cases obscure, but in a large number of cases it is produced by the continual administration of food which is not adapted to the age or digestive capabilities of the child. It is at times met with as a sequela of some exhausting disease, such as typhoid fever, pneumonia, or one of the acute exanthemata. It very rarely occurs in early infancy, being usually met with during the middle and later periods of childhood.

SYMPTOMS.—The symptoms are at first somewhat varied. The disease may be preceded by a number of attacks of gastro-enteric indigestion of a subacute character. A tendency to nausea and vomiting extending over a number of months may sometimes precede the full development of the disease. The gastric disturbance, however, is not marked, and is probably a reflex condition depending upon the functional disturbance of the duodenum. At first the fæcal discharges show merely the various changes which occur in ordinary indigestion, sometimes manifesting a tendency to diarrhœa and sometimes to constipation. The color of the discharges at this early period is not significant of anything beyond ordinary indigestion, and is usually a mixture of yellow, white, and green. As the disease progresses, certain characteristic symptoms arise and definitely mark its presence. Mucus begins to appear in the fæcal discharges, and soon becomes quite large in amount.

I have here a specimen (Fig. 110) of the shreds and masses of mucus which appear in the discharges, and which, in combination with the other symptoms which I am about to describe, are so significant of the disease that it will be well for you to examine them closely.

FIG. 110.



Mucus from fæcal discharges in a case of chronic duodenal indigestion.

With this hypersecretion of mucus, which I shall again impress upon you is not necessarily an indication of an inflammatory condition, the child begins to be fretful, to be wakeful at night, to grind its teeth, and to lose in weight. The skin becomes dry, and there is usually a coexisting follicular pharyngitis which causes a short, dry cough. The child gets tired easily, and complains of pain in the epigastrium after eating. The abdomen is apt to be distended and tympanitic. There are frequently frontal headache, a coated tongue, and a disagreeable odor to the breath. The fæcal movements now begin to become clay-colored, and the skin to assume a sallow tint, with at times a slight amount of icterus. Sometimes an exacerbation of all the symptoms takes place, resulting in an acute attack of indigestion. These symptoms, varying in intensity, and sometimes ceasing to be prominent for days or weeks, usually continue for months, and in intractable cases may last for years. The temperature in this disease is usually normal, sometimes subnormal, but may of course, where an exacerbation occurs, be somewhat raised. The pulse is usually moderately slow. Sometimes a subacute form of bronchial catarrh accompanies the disease, but it does not appear to be a part of it. There is often a craving for large quantities of sugar.

DIAGNOSIS.—When all the symptoms are present, the diagnosis of chronic duodenal indigestion is not difficult. The appearance of the child is characteristic. Its eyes are dull and heavy; its skin is dry and harsh and sometimes slightly icteric, while the loss of flesh, the distended and tympanitic abdomen, and the coated tongue are more marked than in any other disease. Where, in addition to this picture, an examination of the fæcal discharges shows them to be clay-colored and to contain a large amount of mucus, the diagnosis is quite evident. The disease which is most commonly mistaken for chronic duodenal indigestion is pulmonary tuberculosis. The short, dry cough, the emaciation, and in some cases the bronchial catarrh, often make parents and physician fear that this serious disease is present. If, however, the entire history of the case is studied carefully, pulmonary tuberculosis can soon be eliminated.

PROGNOSIS.—The prognosis of chronic duodenal indigestion is in most cases good. Even in those cases which last for a period of years the health is usually entirely restored. Where, however, the disease has lasted for a long time, and the child is in a very debilitated condition, the prognosis becomes more serious.

TREATMENT.—The treatment of this disease is essentially by diet, and not by drugs. Such articles of food should be given as will be chiefly digested by the stomach and will not tax the duodenal digestion. This of course indicates a proteid diet, and contra-indicates the administration of starches, sugars, and fats. In order not to tax the disturbed duodenum by overloading it in its weak condition, small amounts of food at shorter intervals than usual are found to produce a better result than the regular three or four daily meals. The diet which I have found most valuable in treating these cases is a milk so modified as to have a low percentage of sugar and fat, a high percentage of proteids, and ten or fifteen per cent. of lime water. Soups of various kinds, and meat, can also be given, and the crust of French bread in limited quantity. A valuable adjuvant to this treatment, as a mild astringent and stimulant, is a small amount of claret, preferably given in seltzer water. The meals should be five in the twenty-four hours. It is exceedingly difficult in most cases to keep the child on this diet, but if it is rigorously enforced the duration of the disease will be decidedly shortened. As the epigastric pain and the amount of mucus in the discharges grow less, the diet may be somewhat varied by giving fish and eggs, and the percentage of lime water in the milk may be reduced to five. As relapses occur very easily, however, it is generally best to continue with this rigid diet until the fæcal discharges have become normal in color and have not shown the presence of mucus for a number of weeks. In mild cases where there is much constipation, small doses of calomel, or any mild laxative, are indicated. Podophyllin can also be given, as in this prescription (Prescription 78):

PRESCRIPTION 78.

Metric.	Grammæ.	*Apothecary.*	
℞ Podophyllin.	0 06	℞ Podophyllin.	gr. i;
Alcohol.	3 75	Alcohol.	ʒi.
M.		M.	

Sig.—From 3 to 5 drops, according to the age of the child, in the morning and evening, lessening the dose if it causes more than two discharges daily.

Where there is a tendency to diarrhœa, small doses of bismuth are found to be valuable.

Tincture of nux vomica freely diluted in water and given in doses of a few drops after each meal seems in some cases to be of value.

The remainder of the treatment is essentially symptomatic, and if the children are weak and anæmic tartrate of iron and potassium can be given.

During the whole course of this disease cod-liver oil is contra-indicated, but where the disease has been cured and the child is left weak and emaciated it may in some cases be beneficial. Its administration, however, should always be carefully supervised, as it may cause a relapse.

I have here a child (Case 413*a*), three years old, who has an attack of chronic duodenal indigestion, and who represents very well the general picture of this disease. I wish you to notice especially in this case the distended abdomen, the dry, harsh skin, which is slightly icteric, and the evident loss of flesh. She has been affected by the disease for the past two months. The prominent symptoms have been epigastric pain and clay-colored movements with a hypersecretion of intestinal mucus.

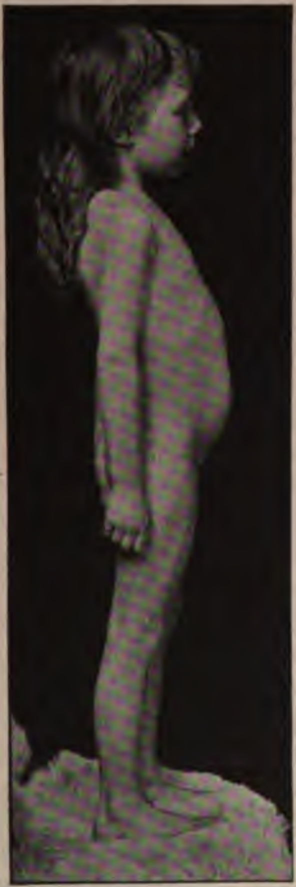
This little girl (Case 414), six years old, represents also a case of chronic duodenal indigestion.

She was healthy at birth, and was nursed until she was thirteen months old. During her first year she had an attack of bronchitis, and since then she has been subject to cough. Previous to this attack she has never had any intestinal disturbance. Her abdomen is said to have been always rather prominent. From her earliest infancy she has been a nervous child, has not slept well, has talked much in her sleep, and has occasionally walked in her sleep. Six months ago she began to lose in weight, and two months ago her cough became quite severe. She then had an attack characterized by vomiting for twenty-four hours, followed by anorexia, fever, languor, and apathy; the bowels became constipated, the skin icteric, the urine dark-colored, and the fæcal movements light-colored. She had an intense craving for sugar, and ate all that she could lay her hands on, so that she had to be watched very closely to prevent her satisfying this morbid appetite. Her appetite for other articles of food was poor. You will notice that the abdomen is distended and tympanitic, and that the tongue is coated. The breath has a disagreeable odor, and there is loss of flesh. There is a follicular pharyngitis, which is evidently the cause of the cough, as nothing abnormal can be detected in the lung or the nose.

(Subsequent history.) The child was placed on the following diet. Her first meal was milk so modified as to contain fat 2, sugar 3, proteids 4, lime water 10. With this meal she was allowed to have a small amount of the crust of French bread. The second meal consisted of broth and the crust of French bread, and one ounce of claret in half a tumbler of seltzer water. The third meal consisted of meat, the crust of French bread, claret, and seltzer water; the fourth, of soup, the crust of French bread, claret, and seltzer water; the fifth, of the modified milk and the crust of French bread. After each meal three drops of tincture of nux vomica were administered.

This diet was carried out rigorously for one week. At the end of that time the child looked much better, the urine was clear, the fæcal movements began to resume a more natural color, the mucus in the discharges was very much lessened, and the abdomen was not so much distended. The tongue was less coated, and the cough had almost disappeared. It was also found that the craving for sugar had much decreased. The diet was then slightly increased in variety. At the end of a month the child had recovered entirely, and some weeks later an ordinary diet at the usual times was given to her.

CASE 414.



Chronic duodenal indigestion. Female, 6 years old.

Incontinence of Fæces.—Incontinence of fæces is a condition in which there is a loss of power of the sphincter to control the movements. It may be due to organic or to functional causes. The organic causes are very rare in childhood, and will best be spoken of in connection with the diseases in which they occur. Functional incontinence may arise from nervous influences, such as excessive mental fatigue, or from stretching of the rectum from habitual constipation.

This boy (Case 415), eleven years old, represents the nervous type of the disease. He has been much overworked at school, has been made to study a number of languages, and has been allowed to take only a very limited amount of exercise in the open air. He has completely lost control of the sphincter ani, and, as you see, is very anæmic and weak. Nothing abnormal has been found on a physical examination.

(Subsequent history.) The boy was taken from school, relieved entirely from his studies, and kept in the open air most of the day. Under this treatment, in addition to the administration of tartrate of iron and potassium and claret, he improved rapidly, and in two months was entirely well.

This boy (Case 416), seven years old, came to the Children's Hospital, during the service of Dr. Lovett, with a history of incontinence of fæces lasting over a year. He illustrates the condition of incontinence from habitual constipation, as the incontinence was found to depend on stretching of the rectum by impacted fæces.

The rectum was emptied by a dose of castor oil and an enema each day. At the end of a week the boy had ceased to have involuntary fæcal movements, and he has since continued well.

Constipation.—By constipation is meant a condition in which the movements of the bowels do not take place as often as is normal for the individual, and in which the consistency is abnormally increased. Constipation is a symptom, and not a disease. It is a relative term, as what would be normal in one individual may be abnormal in another. During the first year of life two or three daily discharges may be considered normal; in the second year two discharges; and in the third and fourth years one discharge is the usual number. The causes of constipation are varied, and in many cases rather obscure. Mechanical obstruction may produce constipation. Thus, as the sigmoid flexure is proportionately long in infancy, flexions may occur, with resulting obstruction. The usual cause of constipation, however, is of a functional character, and may be *spasmodic* or *atonic*.

Spasmodic.—The spasmodic cases are rare, but should be recognized, as they frequently cause much disturbance of the child's general health. In these cases the fæcal movements are usually much increased in size and consistency. This condition produces so much pain and irritation in the rectum that the child endeavors not to have a movement.

Atonic.—The atonic is the most common form of constipation, and simply represents a sluggish condition of the intestinal peristalsis. It is usually caused by food which is not adapted to the digestion of the special child. Thus, in some cases cereals, such as oatmeal, seem to produce this condition, although in a large number of cases they relieve it.

As a rule, constipation can be easily cured, but some cases are extremely intractable and last for a number of years. When the intestine has become more developed and assumed the relative proportions found in adult life, the constipation is very apt to pass away, so that we may in almost every case give a favorable prognosis. Constipation can usually be cured by strict attention to the regulation of the diet by the use of fruits, vegetables, and cereals. In young infants an increase of the fat in the milk will in quite a number of cases relieve it. Variation in the percentage of sugar is occasionally found to be efficient in curing the constipa-

on. Many drugs have been employed in the treatment of constipation,
it, as a rule, we should endeavor not to use them, as they are very apt
be only temporary in their action. In connection with the diet, I place
ost reliance upon enemata and laxative suppositories, such as those made
glycerin or of gluten.

Infantile Atrophy (Marasmus).—Infantile atrophy is essentially a
sease of infancy and early childhood. It is a condition in which extreme
rophy of all the muscular tissues takes place without demonstrable dis-
se of any of the organs. It is apparently due to a vice of absorption,
though this has by no means been clearly proved.

The primary cause of infantile atrophy is unknown. In a number of
ses the disease seems to be secondary to grave intestinal disturbances,
hether of toxic or of organic origin.

PATHOLOGY.—The pathological conditions which are found in cases of
fantile atrophy are exceedingly unsatisfactory, and have not given us
uch information concerning the disease. There is an atrophic condition
all the muscles. Nothing abnormal is found in the various organs
hich can be especially attributed to this disease. It is supposed by some
thologists that the lymph-glands are enlarged; but this enlargement does
ot seem to be a prominent feature. No pathological condition of the
esenteric lymph-glands has been found, and the atrophy of the mesentery
ound them is so great that their increase in size may be seeming rather
an real. In the intestine, although in some cases there is considerable
rophy of the mucous membrane and the submucous tissue, no character-
tic lesion has been proved to be present.

SYMPTOMS.—The symptoms of infantile atrophy are those of starva-
on. The infant begins to emaciate, and extreme loss of weight is the
rominent feature of the disease. The food is apparently digested well,
d the fæcal movements are often of a normal character; in many cases
e total amount in the twenty-four hours is abnormally large. The appe-
te is, as a rule, lessened, the temperature is normal or subnormal, the pulse
weak, and the respirations are generally normal. Usually the infant
ems not to suffer from pain, being sometimes quite apathetic, but in some
ses extreme fretfulness and restlessness occur. Vomiting, apparently of
reflex nature, is at times a prominent symptom. The weight continues
diminish, and without any other symptom the infant may die from
xhaustion.

DIAGNOSIS.—The diagnosis of infantile atrophy is chiefly to be made
om ordinary starvation and from general tuberculosis. From the former
is soon differentiated by its lack of response to good food. In the
rdinary cases of starvation which result either from improper food or from
ck of food, a diet carefully adapted to the age of the infant or child is
on followed by rapid improvement. The differential diagnosis from
eneral tuberculosis is at times exceedingly difficult. I have had under
y care in the hospital in adjoining beds an infant with infantile atrophy

and one with general tuberculosis. In these two cases the symptoms and course of the diseases were so identical that it was impossible to differentiate the two diseases except at the autopsy. On physical examination nothing abnormal could be found in either case except extreme emaciation. In both cases the temperature was slightly raised.

PROGNOSIS.—The prognosis of infantile atrophy is bad, especially during the first year of life. Even under the most careful treatment it is always a very intractable disease. Under special forms of treatment, however, which I shall presently mention, the prognosis is much better than when these cases receive the old and routine treatment of cod-liver oil internally and by inunctions.

TREATMENT.—The treatment of infantile atrophy is essentially by such a modification of the constituents of the milk as to promote intestinal absorption, and without drugs. Although, as I have already stated, it is not entirely proved that the morbid condition is that of a lack of absorption, yet my clinical results are most favorable when the disease has been treated on this principle. After experimenting in a large number of cases by modifying the different constituents of the milk in various ways, I have arrived at the following conclusion: a mixture should be given which contains a low percentage of fat, a high percentage of sugar, and a moderate percentage of proteids. The low percentage of fat is given on the supposition that the infant will increase in weight and thrive on a small proportion of fat, provided it is absorbed. I have found that when higher percentages of fat are given the infant continues to lose in weight. The administration of cod-liver oil is not indicated in these cases, for it is only by a precise adjustment of the percentage of the fat in the food to the individual power of absorption that good results can be obtained. The sugar of high percentage and the proteids of normal percentage seem to be digested and absorbed provided they are combined with a low percentage of fat, since by this combination the nutritive properties of the sugar and of the proteids are made use of. The prescription which I usually write in the beginning of the treatment of these cases, where they occur in the first year of life, is the following (Prescription 79):

PRESCRIPTION 79.

Fat	0.45
Sugar	6.00
Proteids	1.00
Lime water	5.00

After the infant has begun to gain in weight I usually increase the percentage of the fat, but for a number of weeks I do not raise this percentage above 1 or 2. When the infant has once begun to gain steadily the power of absorbing fat is rapidly regained, and percentages such as are in this prescription (Prescription 80) can then be given:

PRESCRIPTION 80.

Fat	3.00
Sugar	7.00
Proteids	2.00
Lime water	5.00

The same treatment can be carried out when the disease occurs in children in their second and third years, but in these cases it is usually possible o increase the percentages of the different elements more rapidly, and after two or three weeks to begin with other articles of diet, such as beef juice, broths of various kinds, and finally, with caution, cereals.

These special modifications of the milk do not, of course, suit every ndividual infant or child, and when the treatment with them is not successful, each of the elements of the milk must be carefully changed and different combinations of these elements tried until the individual idiosyncrasy of absorption in the special case has been discovered.

I have here a case of infantile atrophy of high grade to show you.

This infant (Case 417) is nine months old.

CASE 417.

Infantile atrophy. Female, 9 months old.

She has been fed on foods of various kinds, all of which have contained a considerable percentage of starch. She is said to have been healthy and plump at birth and during the early months of life while she was nursed. After she was weaned and placed on these tarchy foods she began to lose progressively in weight, and she is now, as you see, in an extremely emaciated condition. Physical examination shows nothing abnormal. She has four teeth. Her temperature is slightly subnormal, her pulse is regular but weak, her respirations are normal. On first entering the hospital the bowels were constipated and the fecal movements were brown and looked poorly digested. Since being placed on a diet of modified milk the movements have become well digested and of normal color, but the total amount in twenty-four hours is greater than normal. She has been very fretful, and at times vomits, but since her diet has been regulated she is less fretful and is somewhat apathetic. On entering the hospital she weighed 2966 grammes (6½ pounds). She has been in the hospital two weeks, and has gained in that time 1000 grammes. The food which has been found to suit her powers of absorption contains fat 1, sugar 5, proteids 1, lime water 5, and 0 to 120 c.c. (2 to 4 ounces) have been given every two hours.

This is a case in which it is uncertain whether recovery will eventually take place, as the emaciation is so extreme, but the prognosis is rendered somewhat favorable by the fact that she has already gained 1000 grammes.

(Subsequent history.) The infant did not increase progressively in weight, but sometimes lost considerably, and at one time it seemed as though she could not possibly live. After the food had been modified in various ways, she finally began to improve, and when she was able to digest and absorb 150 c.c. (5 ounces) of milk so modified as to contain fat 3.5, sugar 6.5, and proteids 1.5, she improved rapidly, and finally recovered entirely. Her temperature, with few exceptions, was normal or subnormal through the whole course of the attack.

Infantile atrophy is so exceedingly intractable a disease, and so greatly taxes the patience and skill of the physician, that it may be of interest and encouragement to you in treating these cases to see this child whom I have had brought to the hospital to show you.

This boy (Case 418) is three years old. He was healthy at birth, and remained strong and well during the early months of his life. He was then fed on a number of starchy foods, and soon began to lose progressively in weight. He was in the hospital for five or six months, and was a typical case of a very high grade of infantile atrophy such as I have just shown you. It seemed at one time as though he could scarcely live from day to day, but finally the proportions of the food were so adjusted that he began to absorb a small amount of nutriment. He then began to gain in weight, and recovered entirely. To-day, in his third year, he is, as you see, a remarkably strong, well-developed, and robust child, and, so far as I can detect, is in a perfectly normal condition.

CASE 418.

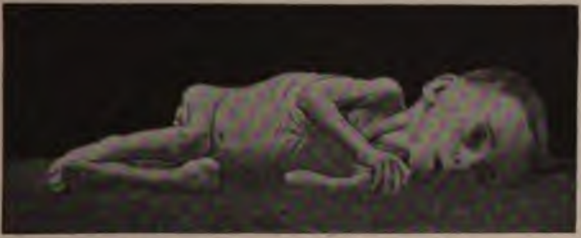
Recovery from infantile atrophy of high grade. Male, 3 years old.

This next infant (Case 419) is also a case of infantile atrophy of high grade.

his infant entered the hospital one week ago, with a history of having been fed on s foods containing starch from the earliest months of its life. It is said to have been y at birth and of average weight. On entering the hospital it weighed 2593 grammes unds). It is, as you see, extremely emaciated, and illustrates the more advanced stage ntile atrophy. It is unable to raise its head; it is apathetic; its skin is cool and dry; pirations are shallow; its pulse is weak, and its temperature is slightly subnormal. It as though it could not live many days. A physical examination shows nothing mal in any of the organs. The fæcal movements are rather large in amount, and, its food has been carefully regulated, are fairly well digested. On entering the al they were still larger in amount and were of a brownish color. It weighs to-day grammes (about 5½ pounds), which is slightly less than its weight on entering the al. This is a case in which the prognosis is very grave, and unless we can soon the food to the digestive tract so as to have it absorbed the infant will die in a short It is being fed on a modified milk in which the percentage of fat is 2, sugar 6, pro-, lime water 10. Although the skin is cool, it is not so cold as in this next child (Case whom I am about to show you.

Case 419.

I.

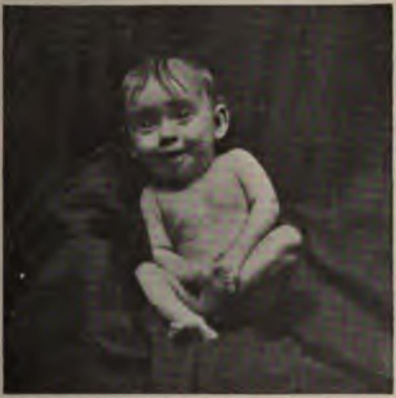


Infantile atrophy. Female, 10 months old.

Subsequent history.) In another week the infant began to gain in weight and evidently orb its food. Although it had a number of relapses, in which it lost considerably in t, it finally began to gain steadily. At the end of three months it had recovered y, and, as is seen in this picture (Case 419, II.), was quite plump.

Case 419.

II.



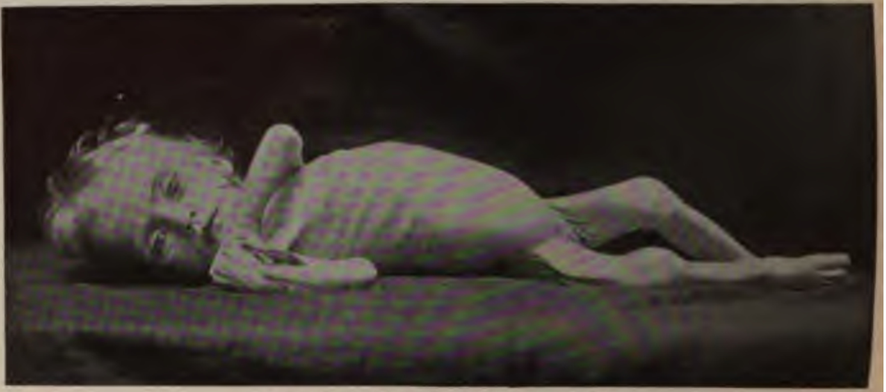
Infantile atrophy. Recovery after three months.

this case the percentage of the fat was finally raised to 4, and that of the sugar to 7, e proteids had to be kept at 1: the lime water was reduced to 5.

his infant (Case 420, page 874), a female, one and a half years old, entered the hospital two weeks ago. She then weighed 4281 grammes (9½ pounds). She is said to have weighed but 900 grammes (2 pounds) at birth. She was nursed by her mother, who apparently had plenty of good breast-milk, and who had two other children whom she had nursed that were healthy and strong. As the infant did not gain, she was nursed for only a short time, and was then fed on various artificial foods. She began to lose in weight, and this loss has continued ever since, so that now, as you see, her emaciation is extreme.

CASE 420.

I.

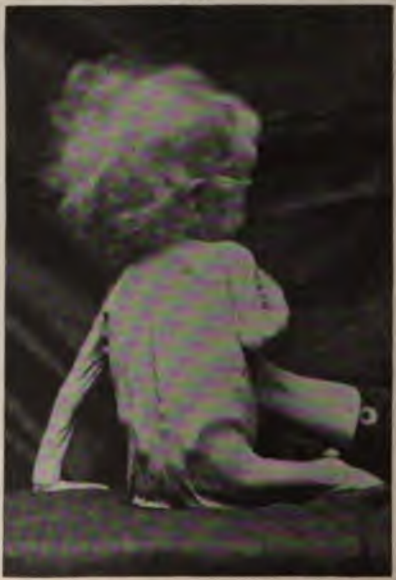


Infantile atrophy. Female, 1⅓ years old.

On physical examination I find that the anterior fontanelle is widely open. There is no enlargement of the epiphyses of the ankles or wrists, but there is a slight rhachitic rosary. Nothing abnormal can be detected in any of the organs. She has four upper and two lower incisors. She is very apathetic, and seems hungry, but when food is given to her she

CASE 420.

II.



Infantile atrophy, showing extreme emaciation of arms, back, and hips.

vomits. Since entering the hospital she has lost 519 grammes (1¼ pounds). Her skin is dry, harsh, and at times quite cold. It has seemed to me ever since she entered the hospital that there was no hope of saving her life, and, as she is losing in weight and does not respond to the various modifications of the food which have been given to her, the probability is that she will soon die. The fæcal movements in this case are very large in amount, but since entering the hospital have been fairly digested. When she is lifted and placed so that you can see her back (II.) you will appreciate the atrophic condition of her muscles, the bones seemingly being covered only by skin. The cervical and inguinal glands are slightly enlarged, and she has a slight cough.

(Subsequent history.) The infant lost steadily in weight during the following week, when it died.

The post-mortem examination, made by Dr. Councilman, showed the following conditions:

There was extreme atrophy of all the muscles. There were no changes in the mesenteric glands, and they were not enlarged, although the extreme atrophy of the mesentery around them made them look so. The liver was normal, and its tissues showed little evidence of atrophy. The spleen was normal. Sections made from various places in the stomach and the intestine showed no changes beyond considerable atrophy of the mucous membrane and of the submucous tissue. The thymus gland was atrophied. There was an extensive bronchitis in the posterior portions of the lungs, while in some parts there was a partial and in others a complete atelectasis.

ELIMINATIVE.—Under the term eliminative disturbances of the intestine are included a number of unexplained and obscure symptoms which we at present are unable to classify elsewhere. It is probable that they will be more fully understood in the future. It seems as though the intestine often acts as an organ for the elimination of various morbid products from the economy. The diarrhœa which results from the irritation of these foreign elements is not distinguishable from that which occurs when the irritation is primarily in the intestine itself. Our knowledge of this class of disturbances is, however, so small that I shall merely refer to its possible occurrence.

ORGANIC.—The organic diseases of the intestine may be divided into *non-inflammatory* and *inflammatory*.

NON-INFLAMMATORY.—The non-inflammatory diseases of the intestine may be divided into *mechanical*, *fermental*, *cholera infantum*, *cholera Asiatica*, and *new growths*.

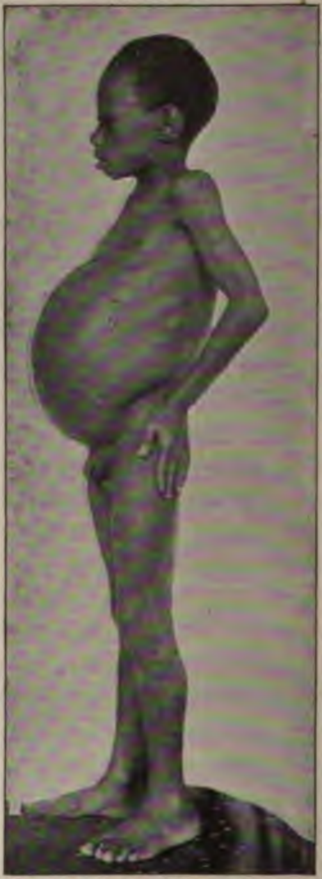
Mechanical.—The mechanical diseases of the intestine are quite numerous, but, with a few exceptions, are not of especial importance medically, and belong rather to the province of surgery.

Dilatation of the Colon.—I have already spoken of dilatation of the colon so far as it relates to the diagnosis of dilatation of the stomach. In comparison with dilatation of the stomach, dilatation of the colon is very rare, except as a temporary condition which is liable to occur at any time from an over-production of gas.

I have here an illustration (Case 421, page 876) of dilatation of the colon which was seemingly caused by a congenital stricture, and in which an artificial anus was made by Dr. Halstead. The child recovered from the operation, but later, owing to still further obstruction, he had to be operated upon again, and died.

I show you this case so that if you happen to meet with this rare pathological condition you will recognize its presence. The extreme distention of the abdomen, which was tympanitic through its whole extent, the evident obstruction to the fæcal discharges, and the absence of symptoms pointing towards gastric disease, would suggest a dilatation of some part of the intestine, presumably of the colon.

CASE 421.



Dilatation of colon. Male, 12 years old.

Volvulus.—By volvulus is meant a twisting or bending of the intestine. This condition is more apt to occur in early life than later, possibly because of the greater proportionate length of the mesentery at this time, which allows the intestine greater latitude of motion. It occurs either by itself or in connection with the next disease of which I shall speak, from which it is to be differentiated by the absence of blood and mucus in the discharges.

Intussusception.—Intussusception or invagination is a condition in which a part of the intestine has passed down into another part. Under these circumstances there is an outer layer of intestine within which is the part of the intestine forming the invagination. Only a small portion of the intestine may be invaginated, or it may extend from the ileo-cæcal valve to the rectum. Small invaginations are frequently found at the post-mortem examinations of infants and young children. These probably take place during the death-struggle, as no pathological condition is found in connection with them. This form is usually multiple and in the small intestine. The form of intussusception which occurs during life is very rare under three months, and is most common from the third to the sixth month. At this age the large intestine is shorter in relation to the small intestine than in the adult, while the mesentery is relatively wider, and thus allows much greater latitude for misplacement, especially of the cæcum and colon. The etiology of intussusception is obscure, but it is probably directly due to increased local peristalsis.

The pathological condition depends upon the tightness of the constriction and the length of time from the beginning of the obstruction. In some cases the incarcerated portion of the intestine is so little constricted that the bowel remains pervious. In other cases the constriction is so great that the tension of the intestinal capillaries quickly becomes so extreme that hemorrhage occurs, and inflammation, with resulting adhesions, is apt to follow rapidly. The intestine may not only be invaginated, but may be bent on itself, an important point to remember in regard to treatment.

SYMPTOMS.—The symptoms of intussusception are usually more acute in infants than in older children. In infants they are often at first rather obscure. Paroxymal pain and discharges of blood from the rectum occur. Later the blood is mixed with mucus and looks like currant jelly. There is usually vomiting, which may be stercoraceous. The mind is clear, and in young infants the face is often tranquil between the paroxysms of pain, so that on looking at the infant it would scarcely be supposed that a serious condition was present. Later, however, the face grows haggard and the eyes become sunken. During the first twenty-four to forty-eight hours, and even longer, the infants will often take their food quite readily. Tenesmus is at times present. There may be fever, especially when inflammation has occurred. The pulse is usually quickened. These symptoms all vary, and depend on the amount of the invagination. In some cases these are the only signs which indicate that there is abdominal disturbance. In many instances, however, either at once or within a few hours, a tumor can be felt in the abdomen.

DIAGNOSIS.—The chief points in diagnosticating intussusception are the occurrence of discharges of blood, vomiting, abdominal pain, and the detection of an abdominal tumor, usually on the left side of the abdomen. In these cases a careful rectal examination should always be made, for a tumor can often be found in this way where an external examination has failed to detect it.

PROGNOSIS.—Without treatment the prognosis is unfavorable, though there are a certain number of recoveries by spontaneous reduction, or rarely by sloughing of the invaginated portion of the intestine, which is then passed by the rectum. If death takes place, it usually occurs about the third or fourth day, or at any rate within a week, after the incarceration is complete. Where the incarceration is not complete the infant may live for many weeks, and in older children in rare instances the disease may become chronic.

Treatment.—The treatment of intussusception when the diagnosis has been definitively made should be immediate, as in no other disease does a delay result in more serious consequences. Food and cathartics or laxatives are contra-indicated. If the infant shows signs of collapse, small quantities of brandy-and-water should be given. In the early hours of the attack an attempt should be made to reduce the intussusception by hydrostatic pressure. This can be easily done by having the infant's buttocks somewhat raised and introducing water under a pressure of about 200 cm. (6⅔ feet) by means of a fountain syringe. The water should be lukewarm, and should have dissolved in it salt in the proportion of one teaspoonful to a quart. The abdomen should be gently rubbed at the same time. In some cases this procedure results in a reduction of the intussusception.

Even where inflammation has not begun and adhesions have not formed, the pressure of the column of water may fail to reduce the intussusception, because the invaginated portion may be bent on itself, so that the hydrostatic pressure increases the obstruction rather than relieves it. Where adhesions have taken place and where there is great congestion, as sometimes occurs during the first twenty-four hours of the attack, hydrostatic pressure is usually unsuccessful and may be dangerous. If this method has failed, the infant should be placed at once in the hands of a surgeon, as under these circumstances an early laparotomy will give the most favorable results.

I shall report to you one of the cases of intussusception which have come under my care.

A male infant (Case 422), six months old, nursed by its mother, and previously perfectly healthy, after a slight loss of appetite for several days began to have abdominal pain in the morning, and in the middle of the day had a discharge of blood from the rectum unmixed with fæcal matter or mucus. The bowels had been thoroughly moved on the previous day, and there had been no tendency to constipation. During the afternoon there were five or six discharges of blood. In the evening the infant looked well and did not show any signs of discomfort except occasional slight attacks of abdominal pain and an indisposition to nurse. The rectal temperature was 39° C. (102.2° F.). An examination of the abdomen externally and by the rectum revealed nothing abnormal. The infant had a restless night, vomited several times after nursing, and had six discharges of blood. The temperature was 38.3° C. (101° F.), the pulse 135, strong and regular, and the general appearance good. The abdomen was soft and not tender on pressure, but towards the umbilicus, under the left costal border, a rather ill-defined cylindrical tumor could be detected.

Hydrostatic pressure was employed to reduce the intussusception, but failed. The surgeon who saw the infant on the second day decided to wait twenty-four hours before performing laparotomy. On the following day the infant died suddenly.

At the post-mortem examination nothing abnormal was found except an ileo-cæcal intussusception. An examination of the invagination showed that the retained cæcum was so twisted that the lower opening was directed to one side of the axis of the intestine, and the hydrostatic pressure from below must have simply packed the sac tighter and rendered reposition more difficult. The invagination involved 20 cm. (8 inches) of the intestine.

he serous surfaces were firmly adherent through their whole extent, and considerable force as required to reduce the invagination without tearing it. The reduction, however, was uccessfully accomplished, the adhesions giving way and the intestine being left uninjured nd apparently healthy. This case illustrates how necessary it is to employ the most killed surgical aid in these cases.

Hernia.—I have already spoken sufficiently of the pathological conition represented by hernia, in my lecture on diseases of the new-born page 430).

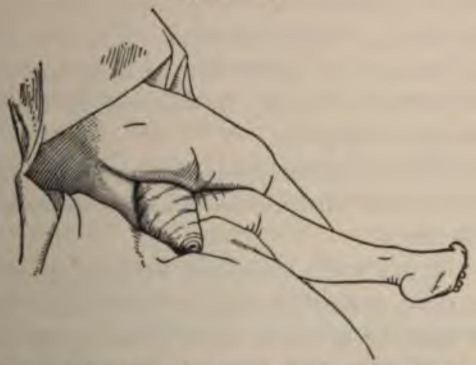
There are a number of lesions which occur about the anus in infants nd young children which, though somewhat rare, should be recognized for urposes of differential diagnosis. They are, however, so purely surgical in heir treatment that they need only be mentioned here.

Fissures.—One of these conditions is that of fissure, which occurs either t the anus or more commonly a little distance from the orifice. Pruritus nd reflex urinary symptoms are common. Defecation is often painful, and onstipation of the spasmodic type may thus result.

Prolapse.—Prolapse of the rectum is not uncommon in young children. t is usually produced by straining from various causes, especially in xtreme constipation. The wall of the rectum comes down through the nus, and is easily recognized by the appearance of the mucous membrane. he prolapse is ordinarily transitory, but in the more severe forms the ectum remains down.

The treatment is to remove the cause. Constipation should be relieved irst by enemata and then by keeping the movements of the bowels semiiquid by means of gentle laxatives. The child should be kept in bed for number of days, the protrusion being gently pushed back each time that it omes down. After reposition it should be kept in place by means of a pad nd a T bandage. Under this treatment a large number of cases recover. he more serious and intractable cases, however, should be referred to a urgeon.

Case 423.



Congenital prolapse of rectum. Female, 22 months old.

I have here a case of prolapse of the rectum to show you which has een under the care of Dr. C. B. Porter.

The infant (Case 428), a female, twenty-two months old, has had this condition of prolapse since birth. Lately the prolapsus has been increasing in size. The infant is not fretful, and seems very well. The movements of the bowels are normal through the prolapsed portion of the rectum. This is one of the more severe types of the disease. You see that the prolapsus forms a large rounded tumor covered with reddish mucous membrane projecting from the anus. It is about 7.8 cm. (3 inches) long and 4.0 cm. (1½ inches) thick. The tumor is not sensitive to the touch.

Polypi.—Polypus of the rectum is more common in early life than at any other period. Hemorrhage from the rectum, when not due to constipation, diarrhœa, or fissure, usually arises from polypi. A careful examination for this growth should be made where rectal bleeding is frequent or large. Rectal polypi are of various sizes, and may be myxo-fibromata or adenomata. The surface of the polypus is usually smooth, and the pedicle is often long and thin.

The diagnosis is easily made by a digital examination.

The treatment is simply to twist or cut off the polypi. The growth is not apt to recur.

Hemorrhoids.—Hemorrhoids are rarely met with in infancy or early childhood, but can occur as in later life, and should be treated by the same methods.

Fistulæ.—Fistula in ano is not a very common condition in infancy or early childhood, but is at times met with. The condition has the same characteristics as in the adult, and should be treated in the same way.

Fermental.—The non-inflammatory conditions of the intestine, which for want of a better term we speak of at present as fermental, include those which arise from acid fermentation and albuminous decomposition, which are produced by micro-organisms. The disturbances which arise from these causes represent the greater proportion of the diarrhœal diseases which occur during the warm months of the year.

ETIOLOGY AND PATHOLOGY.—The causes of fermental disturbance in the intestine lie in impure or improper foods and bad hygienic surroundings. In both acid fermentation and albuminous decomposition it is probable that the small intestine is most affected. The condition of the mucous membrane may be normal, or there may be desquamative catarrh. The process may go no farther, or it may be followed by inflammatory changes in the intestinal mucous membrane.

The fermental class of cases holds a position midway between the nervous forms of intestinal disturbance and the inflammatory forms with their pronounced lesions.

SYMPTOMS.—You can well understand from the great variety of causes which give rise to these fermental processes how varied may be the symptoms. The onset may be subacute, with little or no fever and without vomiting, or it may be acute and accompanied by a high temperature and active vomiting. After a variable period of general discomfort and restlessness, diarrhœa sets in, which varies so greatly as to its frequency, amount, color, and consistency that it would be impossible in the present state of our knowledge to divide these variations clinically. The onset of fermental diarrhœa is, however, so often characterized by the toxic symptoms of sudden rise of temperature, followed after a day or so by a normal temperature, that when we meet with this occurrence we are usually justified in eliminating the inflammatory and more serious intestinal lesions. In some cases the diarrhœa, although accompanied by much prostration and various nervous disturbances, disappears after a few days; in others, especially in the warm weather, it may last for a number of months. In this fermental diarrhœa the color of the discharges is commonly some shade of green or greenish yellow, and the odor is often very offensive, sometimes being the excessively sour one which is supposed to arise from acid fermentation, and at other times the extremely foul one of albuminous decomposition. The discharges are usually accompanied by considerable pain and a large amount of gas. The symptoms are often so severe that the disease has a serious aspect, but in a considerable number of cases after the intestine has been thoroughly emptied the temperature falls and the nervous symptoms subside. There is usually rapid and great loss of weight. In cases which are not prolonged by fresh irritation or by unwise treatment recovery often takes place quite rapidly.

DIAGNOSIS.—Where the attack is subacute, with slow onset, without vomiting, and with infrequent discharges, the diagnosis is not difficult, and is to be made from the nervous disturbances, which can usually soon be differentiated by the absence of fever and by rapid recovery. Where, however, the onset is acute and is accompanied by vomiting, the diagnosis must often be held in abeyance, as the symptoms of high temperature, vomiting, and diarrhœa may be present in infants and young children in the initial stage of a number of acute diseases. The disease from which it is to be especially differentiated is cholera infantum. In fermental diarrhœa the prostration is much less, and the temperature after the early hours of the attack is much lower. The serous discharges and the continuous vomiting which soon arise in cholera infantum are quite different from the greenish discharges and the less frequent vomiting which occur in fermental diarrhœa. We must remember, however, that cholera infantum and the acute inflammatory intestinal diseases are usually preceded for a number of days by this fermental form of diarrhœa, and that the special micro-organisms which produce the former disease gain an entrance for themselves and their toxines by means of the abnormal intestinal conditions produced by the fermental changes. You must also remember that gastro-enteric symptoms are often so pronounced during the early days of a pneumonia that they may mask the presence of that disease.

PROGNOSIS.—In previously healthy children the prognosis of fermental diarrhœa is good. It depends, however, upon the degree and kind of the fermental process which is causing the disease, and also on the amount of resistance to these processes which the individual possesses. It also depends upon the vulnerability of the individual to the other bacteria which may at ts administration. The peristalsis which occurs as the result of fermental rritation is a conservative process of nature, intended to carry away the norbid products which have resulted from the fermentation. Under these onditions the administration of opium prevents the elimination of the oison from the intestine and allows it to remain and produce still further rritation, or to be absorbed and give rise to still graver septic symptoms. In certain cases where the intestine has been thoroughly emptied, small doses f opium in the form of tinctura opii camphorata may be used with caution o diminish pain and control the excessive peristalsis which may result rom nervous exhaustion after the disease has run its course. In these ases, however, stimulants are more valuable than opium.

When a child in the warm weather has once had an attack of fermental iarrhœa, it is very apt to have a number of attacks: its diet should therefore be carefully regulated for a considerable period, and, if possible, it hould be taken to the sea-shore or the country until the return of cool veather.

As especial illustrations of the great variety of fermental diarrhœas which you are liable to meet with in warm weather, I shall call your attenion to these cases which have come under my notice.

A child (Case 424), three years old, and perfectly well, was attacked suddenly with bdominal pain, nausea, pallor, and prostration. He vomited once or twice, and was ound to have a temperature of 40° C. (104° F.). Within a few hours he began to have requent fæcal dejections of sour odor, lessened consistency, moderate amount, and a eculiar dark green color, a specimen of which (Plate III., 18, facing page 112) I have ere to show you. This green is one of the more common colors met with in fermental iarrhœa. At first the discharges took place every hour, and later every three or four ours. After the first twenty-four hours the temperature became normal, and in three or our days the diarrhœa ceased entirely.

I have here a case which is also illustrative of this form of fermental iarrhœa.

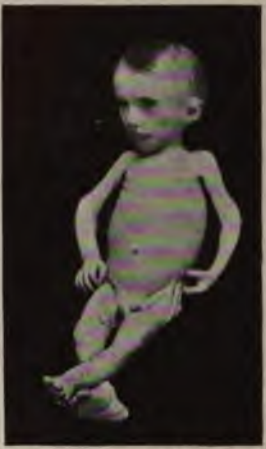
This infant (Case 425, page 884) is thirteen months old. On entering the hospital t was much emaciated, and had a slight diarrhœa, caused apparently by improper food. Its temperature was only slightly raised. On examining it nothing else abnormal was etected. The diarrhœa was infrequent, and was not accompanied by any other especial ymptoms. It soon began to improve, gained in weight, and had a normal temperature. After it had been in the hospital one week it suddenly began to have diarrhœa characterzed by large frequent discharges, of lessened consistency, of foul odor, and of the color which you see in this specimen (Plate III., 19, facing page 112). The discharge would eem from its foul odor to be an illustration of what is called albuminous decomposition. You will notice the mixture of yellow and light and dark green, which is so different from he dark-green specimen which I have just shown you (Plate III., 18, facing page 112). These colors are, however, only relative, and are not diagnostic. In this acute attack the emperature was raised at first, but soon fell to a little above normal.

The infant has lost greatly in weight, has become extremely emaciated, and looks as f it would die. The skin often becomes cold, and the prostration is extreme. These ymptoms have continued for three or four days, and the number of discharges in the wenty-four hours varies from seven to ten. This is the seventh day from the beginning f the acute attack, and you see the condition in which it has left the patient.

(Subsequent history.) The symptoms became less severe, and the diarrhœa abated. A few days later the diarrhœa stopped entirely, and the infant then gained rapidly in weight and strength. This picture (II.) shows the great improvement which occurred in a month.

Case 425.

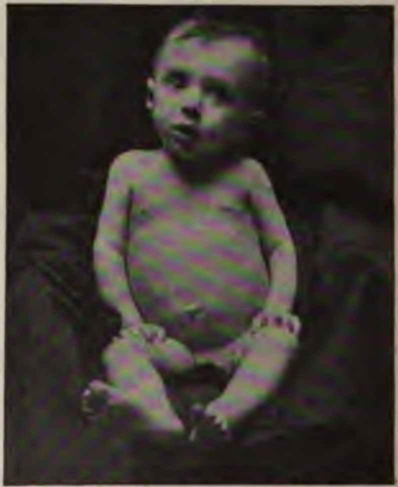
I.



Fermental diarrhœa. Male, 13 months old.

Case 425.

II.



Fermental diarrhœa. One month after recovery.

These cases of fermental diarrhœa at times are prolonged for many weeks or even months, and thus produce a chronic form of diarrhœa. This occurs especially in children who are the subjects of rhachitis, syphilis, and general tuberculosis; also in those with chronic broncho-pneumonia. I have already told you that the continuous administration of improper food may produce this condition; so also may improper exposure from insufficient clothing.

Cholera Infantum.—Cholera infantum is an acute gastro-enteric disturbance characterized by intense choleriform symptoms. The term cholera infantum should be exclusively restricted to this class of cases, and should not be used to designate the many acute and serious attacks of vomiting and diarrhœa which are so often designated cholera infantum. It is a rare disease in comparison with the fermental diarrhœas which I have just described to you.

ETIOLOGY.—There is not much doubt that cholera infantum is caused by a specific micro-organism, although this organism has not as yet been determined. It most commonly occurs in the first two years of life, and in its development is probably closely associated with the food, for it has been noticed that infants who are fed exclusively on pure and sterile foods, such as breast-milk, are not liable to be attacked by it. It is also significant that the disease occurs only in hot weather.

PATHOLOGY.—The pathology of cholera infantum has not yet been satisfactorily determined, but it seems to be a non-inflammatory disturbance of the whole gastro-enteric tract, without any gross lesion beyond a desquamative catarrh, and sometimes hyperæmia, of the mucous membrane.

SYMPTOMS.—The onset of cholera infantum may be sudden, but, as a rule, it is preceded by some form of gastro-enteric disturbance, which, by causing an irritation of the mucous membrane, renders the infant vulnerable. When, however, the disease has once gained a foothold, the development of the symptoms is very rapid.

After a variable but generally short period of restlessness and apparent abdominal discomfort, the infant begins to vomit. The vomiting is either accompanied or quickly followed by profuse diarrhœa. After the stomach and intestine have been emptied of the food which may happen to be in them at the time of the onset, the vomitus and the diarrhœal discharges are chiefly serous; and it is this watery consistency of the discharges which especially characterizes the disease. As a rule, the discharges are odorless, and consist of serum mixed with epithelial cells and many bacteria. Although the disease is more likely to attack weak and debilitated infants, yet it often attacks those who are healthy and robust. It may run its course to a fatal issue in from twenty-four to forty-eight hours. The extremities soon become cold, the skin is pallid or even cyanotic, and the face pinched. The abdomen may be a little distended, but is soft, and soon becomes rather retracted. The pulse is rapid and difficult to count. The respirations are somewhat quick and superficial. The temperature of the entire surface of the body is low, but the deep rectal temperature is high, 39.4°, 40°, or 40.5° C. (103°, 104°, or 105° F.). The thirst is great and is a very prominent symptom. The fontanelle very soon becomes depressed. The urine is suppressed, and nervous symptoms, such as twitching of the arms and great restlessness, are present. Rapid emaciation takes place, and all the symptoms increase in severity. At first the infant whimpers, but soon it becomes listless, falls into a stupor, or may have convulsions. The infant may die in this stage, which closely resembles the algid stage of cholera Asiatica. The disease appears to be self-limited, and if the infant survives the first two or three days a crisis comes, the skin becomes less cool and of a better color, the vomiting and diarrhœa grow less frequent, and finally it is left with a slight amount of simple diarrhœa and occasional vomiting. These symptoms may become chronic, in which case the infant finally dies of exhaustion or from an attack of one of the other gastro-enteric diseases, to which it is left very susceptible.

DIAGNOSIS.—The diagnosis of cholera infantum is not difficult if the characteristic symptoms are borne in mind; these are rapid onset, constant vomiting, frequent serous discharges, intense thirst, high rectal temperature, low surface temperature, collapse, depressed fontanelle, sudden loss of weight, and distressed, restless expression, suggesting speedy death, all developing in from twenty-four to forty-eight hours.

PROGNOSIS.—The prognosis is bad. The more violent the attack, the higher the temperature, the less the vitality, and the warmer the weather, the worse is the prognosis. When the infant has survived the very acute symptoms which appear in the first two or three days, the prognosis is much more favorable.

TREATMENT.—Cholera infantum is so formidable in its attack that it must be treated most energetically if we hope to succeed in saving the infant's life. The indications for treatment are (1) to assist the effort which nature is making to free the stomach and intestine from the poison which is in them; (2) to restore the surface circulation, which is so seriously interfered with; (3) to supply water to the tissues, which are being drained to so grave an extent; and (4) to support the strength until the disease has run its course.

The poison seems to act with especial virulence on those portions of the economy where it is most concentrated,—namely, the stomach and the intestine. We therefore have at first extreme irritation of these parts, which causes increased peristalsis, and later vaso-motor paralysis, with great transudation of serum. This condition of the gastro-enteric tract is to be especially borne in mind during the whole course of our treatment.

In this disease we should not attempt to use any remedy which works slowly. The condition of the mucous membrane is in all probability such that absorption of drugs does not take place readily. The administration of drugs is, therefore, contra-indicated, for they may later, when absorption is being restored, prove fatal by their cumulative action. During the acute stage of the disease the digestive functions fail to act, and therefore food of any kind will be only an additional source of irritation.

Early in the attack, and when the vomiting has not caused much prostration, the stomach should be thoroughly washed out with warm water and the intestine should be irrigated. If the rectal temperature is very high, ice-cold water may be used for irrigation. Where the vomiting has continued for some time and there is prostration with great thirst, the infant should be allowed to suck sterilized ice-cold water from the bottle. At first nothing else should be given by the mouth.

The infant should be placed at once in a warm pack. This should be done by wrapping it to the chin in sheets wrung out of water at least as hot as 38° C. (100.4° F.). It should then be enveloped in a hot blanket. This procedure should be repeated as often as the infant shows signs of collapse or much cyanosis and coldness of the skin. This is the best method that I know of to restore the surface circulation. In extreme cases the subcutaneous injection of salt solution can be tried.

While the infant is in the hot pack, water can be given freely by the mouth, and, if necessary, small and frequently repeated doses of stimulants, unless they appear to excite vomiting, in which case they should be given hypodermically.

If the vomiting and diarrhœa still continue excessive after this treatment, small doses of morphine, 0.0006 gramme ($\frac{1}{100}$ grain), and atropine, 0.00008 gramme ($\frac{1}{800}$ grain), for an infant a year old, can be tried hypodermically. The effect should be carefully watched, and the dose repeated if necessary, as recommended by Holt.

If, after the vomiting and diarrhœa have ceased, the heart's action continues very weak and does not respond to stimulants, small doses of digitalis should be given. The greatest caution should be employed in using drugs, however, as they generally do more harm than good.

If an absolutely fresh and sterile milk can be obtained, it can be used as a food, as in any of the other forms of gastro-enteric diseases which I have already described, but for some days the percentages of the elements in the milk must be much lessened, and the child's strength must be supported mostly by stimulants freely diluted with sterilized water.

Cholera Asiatica.—Cholera Asiatica is a highly infectious disease, caused by the comma bacillus of Koch, which manifests its most violent symptoms in the gastro-enteric tract. Its symptoms very closely resemble those of cholera infantum. The disease in infants should be diagnosticated from cholera infantum, which is done by finding the comma bacillus in the vomitus or in the discharges. There are no especial differences between cholera Asiatica in the adult and the same disease in the infant. It is exceedingly fatal during infancy and childhood, and young infants who are attacked by the disease during a cholera epidemic seldom live. The treatment is the same as that which I have just described for cholera infantum.

New Growths.—New growths in the enteric tract are very rare in infancy and childhood, and are mostly confined to myxomatous polypi of the rectum.

INFLAMMATORY.—The inflammatory diseases of the enteric tract may be *acute* or *chronic*.

Under *acute* inflammatory diseases may be included *appendicitis* and *ileo-colitis.*

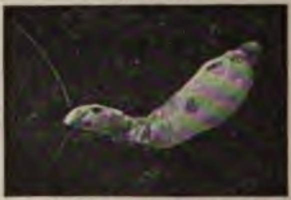
Appendicitis.—Inflammation of the appendix cæci is essentially a surgical disease, and is one which under all circumstances should be placed immediately in the hands of those who are skilled in abdominal surgery. From my observation of this disease I am so strongly impressed with this fact that I consider an extended description of it in medical lectures and by physicians out of place. I shall therefore confine my remarks on this disease to a very few words, which will aid you in making a diagnosis when you meet with one of these cases.

Under the term appendicitis we now include those inflammatory conditions in the neighborhood of the cæcum which were formerly called cæcitis and perityphlitis. The reason for this is that there is little doubt that in most instances the appendix is the part primarily involved. The disease occurs most commonly after the tenth year, and is rare in the early months of life, but it may occur at any age.

ETIOLOGY.—The cause of appendicitis is in most cases an inflammation of the lining mucous membrane of the appendix arising from fæcal concretions. It is seldom caused by any foreign bodies, such as seeds of any size.

I have here a specimen of the appendix (Fig. 111) which was taken from a girl nine years old forty-eight hours from the beginning of the attack, the first she had ever had. The operation was performed by Dr. S. J. Mixter, and was followed by complete recovery.

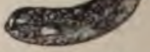
FIG. 111.



Appendix removed from female 9 years old. (Natural size.)

On opening the appendix this fæcal concretion was found (Fig. 112).

FIG. 112.



Fæcal concretion in appendix. (Natural size.)

PATHOLOGY.—The pathological lesions which occur in these cases vary from a simple inflammatory condition, with exudation, induration, and thickening, to gangrene and necrosis.

SYMPTOMS.—The symptoms of appendicitis are, as a rule, the more obscure the younger the individual. In infants and young children abdominal pain may be difficult to localize, and may be referred to some other part of the body. In like manner pain in the thorax may be referred to the abdomen, so that it is often impossible to be guided by the apparent seat of the pain. There are no prodromal symptoms which are especially characteristic or of much aid in determining whether appendicitis is present. The temperature is often very misleading. I have seen a child with a severe attack of appendicitis in whom the acute symptoms disappeared in a few hours and the temperature was raised very little above normal. Dr. Mixter, whose surgical knowledge was called upon to determine what should be done in this case, decided to operate, and on opening the abdomen the appendix was found in a highly inflamed condition: pus had formed and distended it, and perforation had almost taken place. There is nothing especially significant in the pulse or the respiration which will aid you in diagnosticating the disease. In a number of cases, however, vomiting, pain and tenderness in the region of the cæcum, and later a sensation of resistance and dulness on percussion, constitute a group of symptoms which should lead us strongly to suspect the presence of this disease. The vomiting, as a rule, is not stercoraceous, and in young infants diarrhœa is apt to occur as often as constipation.

In cases of appendicitis which recover after operation various inflammatory lesions are left, and the disease is liable to recur from time to time. This condition is known as chronic appendicitis. Where the symptoms continually recur, the patients lose in weight and strength, but often can be entirely cured by having the appendix removed.

Diagnosis.—The diagnosis is to be made chiefly from intussusception and volvulus, especially the former. In intussusception, as I have already told you, there is usually an absence in the beginning of pain and tenderness, and the tumor which is ordinarily found is to the left of the median line rather than to the right. The vomiting in appendicitis is not stercoraceous; in intussusception it is often so. Pain and tenderness to a varying degree are always present, but the tumor is often not felt until late in the disease. The temperature and pulse are generally slightly raised. The same anxious expression of the face occurs in appendicitis as in intussusception. You must not depend upon the locality of the tumor and the pain and tenderness in differentiating these two diseases, for in some instances the inflamed appendix may be found to the left of the median line, and in intussusception, especially if not of the ileo-cæcal variety, it may be on the right of the median line. All these questions, however, are for the skilled surgeon to decide; and when this group of symptoms is present we are justified in making a provisional diagnosis of appendicitis and in at once summoning surgical aid.

Prognosis.—The prognosis of appendicitis under judicious treatment, especially if operative interference is instituted early, is very favorable; but when operation has been deferred until perforation has taken place the prognosis becomes unfavorable. Even under these conditions, however, many cases recover. The prognosis of cases which are operated upon when inflammation is not present between recurrent attacks of appendicitis is in almost every instance favorable.

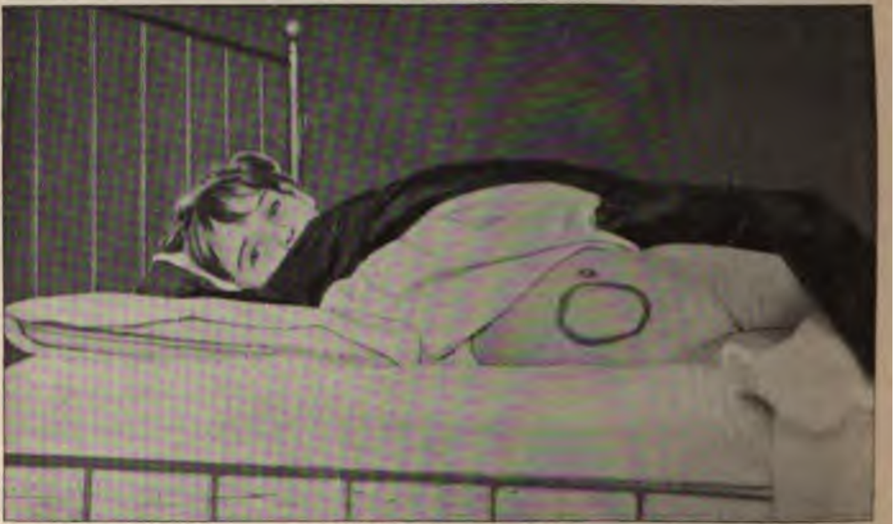
TREATMENT.—When you have made the diagnosis of appendicitis, you should at once place the child in bed, enforce absolute quiet, apply hot fomentations to the abdomen, and, if necessary, give sufficient opium to relieve the acute pain. Cathartics and laxatives should not be given. The food should be small quantities of peptonized milk, and should be given by enemata. It is almost needless to repeat that the best surgical aid should be called in at once to determine upon the next steps in the treatment.

I have to report to you a case of appendicitis which was under the care of Dr. Crocker and was operated upon by Dr. George Haven.

A child (Case 426), twenty-eight months old, had loss of appetite, sleeplessness, nausea, vomiting, slight diarrhœa, and abdominal pain. Two days before the operation a tumor had been found in the left lower part of the abdomen. The child's face had a pinched expression and showed much pallor. Her pulse was 170, her temperature 39.7° C. (103.5° F.). On the day when the operation was performed, in addition to the tumor which had first been found, the right half of the abdomen was filled by a tumor of somewhat irregular outline, with tense walls, and giving an absolutely flat note on percussion. Changes of posture produced no effect on the physical signs. An incision was made through the middle of the tumor, and about a pint of pus escaped, together with masses of detritus having a strongly fæcal odor. The child recovered entirely.

I happen to have here in the wards a little girl (Case 427), eight years old, who illustrates one of the mild cases of appendicitis which often recover without operation, and whom I have placed under surgical supervision in accordance with my strong opinions on this subject.

CASE 427.



Appendicitis. Female, 8 years old.

This child was well until four days ago, when she began to have severe pain in the right side in the region of the appendix. This was followed by headache, nausea, and vomiting. Marked tenderness soon appeared in the area where she complained of pain. The pain continued with slight intervals. The bowels were constipated. At first there was an almost constant desire to have a movement of the bowels. On entering the hospital the abdomen was tympanitic and not tender, except in the area which I have marked in black, which covers a space of 6.5 to 7.8 cm. (2½ to 3 inches). Within this line there have been pain, tenderness, and dulness on percussion. Her tongue has been coated. On close inspection you will notice that there is slight bulging in the area marked in black. The temperature has been about 39.4° C. (103° F.), the pulse 100, the respirations 28.

With symptoms of this nature there is not much doubt that we are dealing with a case of appendicitis. Morphine, 0.004 gramme ($\frac{1}{16}$ grain), was required a number of times to relieve the pain.

(Subsequent history.) On the day following the child's entrance to the hospital the temperature began to fall, the tumor became less distinct, and there was less tenderness and not much pain. It was decided not to operate, but to watch the case carefully. On the third day after entering the hospital, the seventh day of the disease, the temperature became normal, the pain and tenderness disappeared entirely, and the tumor became indistinct. The bowels moved naturally on the seventh day, and in the third week from the onset of the attack the child was perfectly well, and only a little resistance could be felt in the area which had been occupied by the tumor.

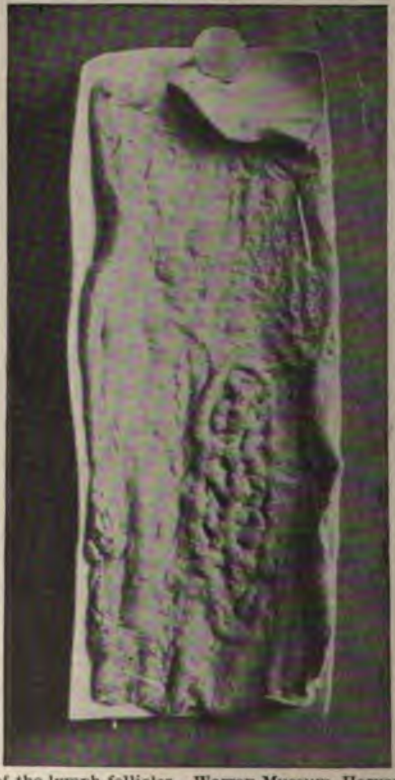
Ileo-Colitis.—Under the term ileo-colitis are included all the more marked and grave lesions of the intestine. These lesions are so varied that it would be impossible to classify them in detail, and practically we can divide them in only a very general way.

The divisions which have been adopted to simplify the subject are (1) simple catarrhal inflammation, which includes the non-ulcerative form of follicular inflammation, (2) follicular ulceration, (3) an inflammation characterized by a pseudo-membrane, (4) an inflammation caused by the typhoid bacillus, (5) an inflammation caused by the amœba coli, and (6) an inflammation caused by the bacillus tuberculosis. The first three of these divisions, *catarrhal*, *ulcerative*, and *membranous*, although differing essentially in their prognosis, are so often represented by the same symptoms that they can be differentiated only in the most general way. A symptom common to all these diseases is that the temperature, although not necessarily high, is, as a rule, raised through the whole course of the disease. In this way we can usually differentiate these diseases from the non-inflammatory conditions of which I have already spoken. There are so many varieties of pathological lesions found in connection with the catarrhal and non-ulcerative follicular and the ulcerative follicular inflammations that the clinical distinction between the two conditions, until our knowledge of these diseases shall have been greatly increased, must be very limited. In both the lesions are so varied that they probably arise from a number of organisms, and their pathology must for the present include all forms which cannot be classed under the pseudo-membranous, typhoidal, or amœbic forms of ileo-colitis. They may occur as acute primary diseases, but are usually secondary to the fermental diarrhœas, and sometimes to the infectious diseases, especially measles.

In the pseudo-membranous form of ileo-colitis the ileum and the colon are chiefly affected. The lesions are probably due to a number of organisms, but its pathology is more definitely known than that of the catarrhal and ulcerative follicular forms. It is characterized by the presence of a membrane on the surface of the mucous membrane, which extends into it, due to a combination of fibrinous exudation and necrosis. That is, there is a definite pathology. The disease may be primary or secondary. In the primary form it represents what is usually spoken of as epidemic or sporadic dysentery. The secondary form is that which follows certain infectious diseases, such as measles. All these forms are commonly spoken of as dysentery; but from what I have told you you will see that the word dysentery should no longer be retained in our nomenclature, as it has been used for so many different pathological conditions.

Before endeavoring to tell you what little is known regarding the symptoms of these diseases, I shall show you a few specimens illustrating some of the pathological conditions which occur in *ileo-colitis catarrhalis*, *ileo-colitis ulcerativa follicularis*, and *ileo-colitis pseudo-membranosa*. Much more extended studies of these conditions, both as to their pathology and their bacteriology, must be made before anything more than this general view of the subject can be used for clinical purposes. You will of course understand that these specimens which I am about to show you do not represent all the lesions which occur in these diseases, but illustrate some of the principal ones only. The notes in connection with these cases show how with our present knowledge it is usually impossible for us to diagnosticate the lesions during life.

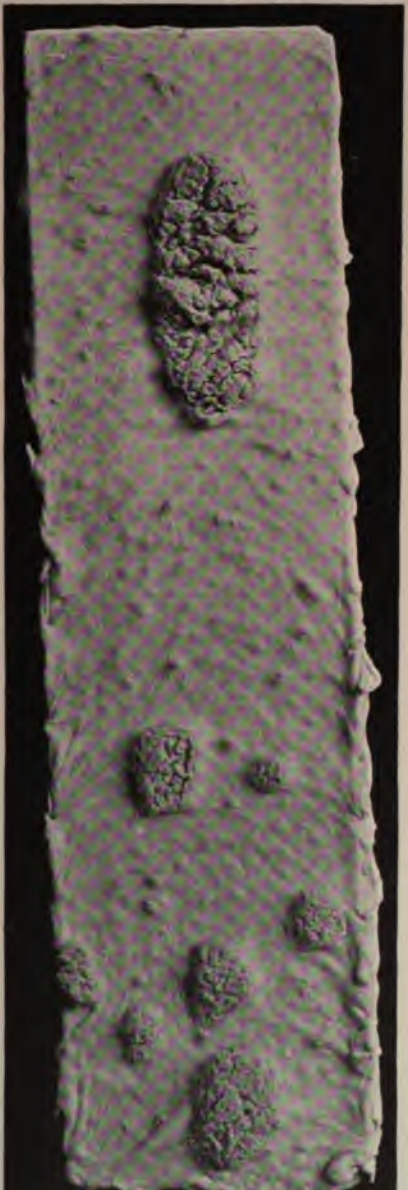
FIG. 113.



Hyperplasia of the lymph-follicles. Warren Museum, Harvard University.

This first specimen (Fig. 113) is a portion of the colon of an infant who during life had only a slight diarrhœa.

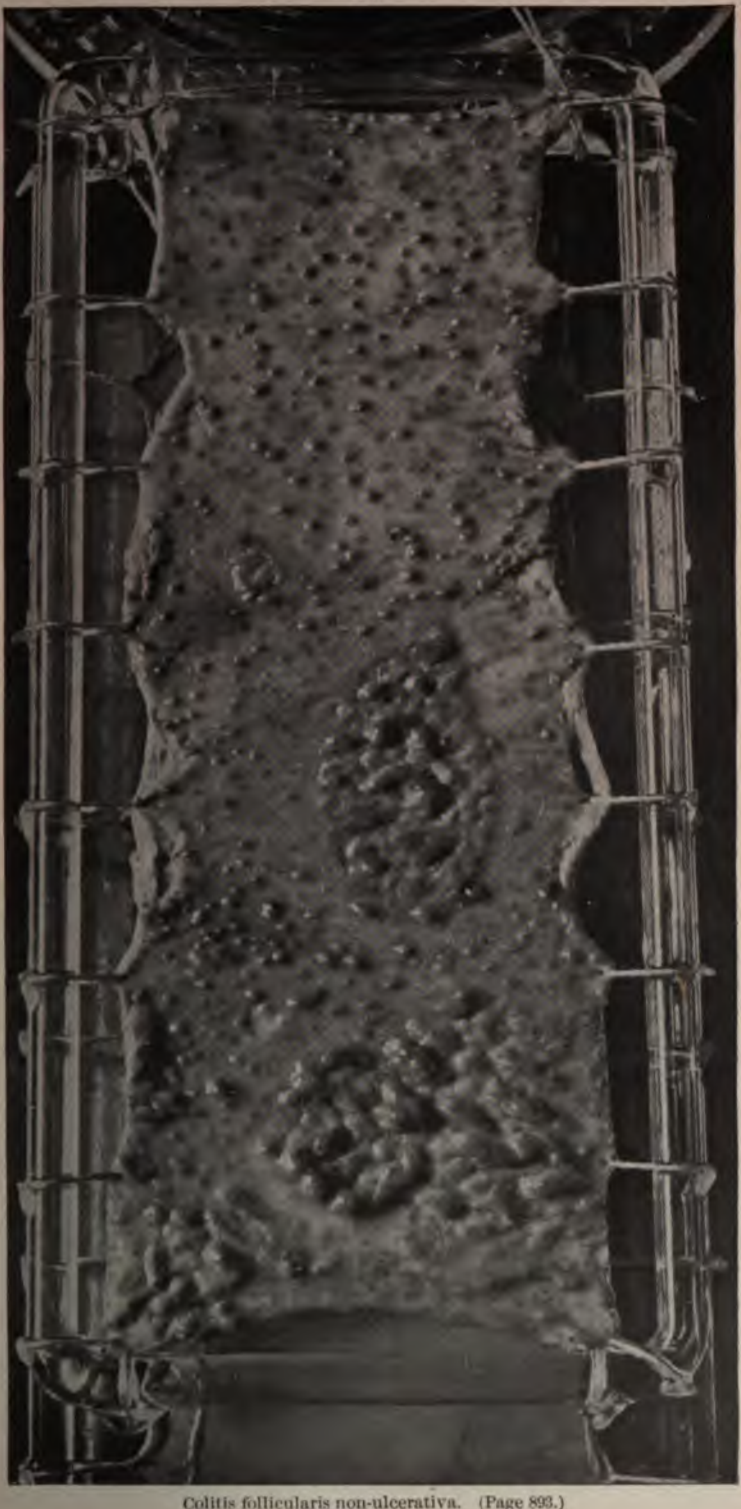
CASE 428. FIG. 114.



Non-ulcerative follicular inflammation. Simple hyperplasia of lymph-follicles. Female, 3 years old. Warren Museum, Harvard University. (Page 893.)

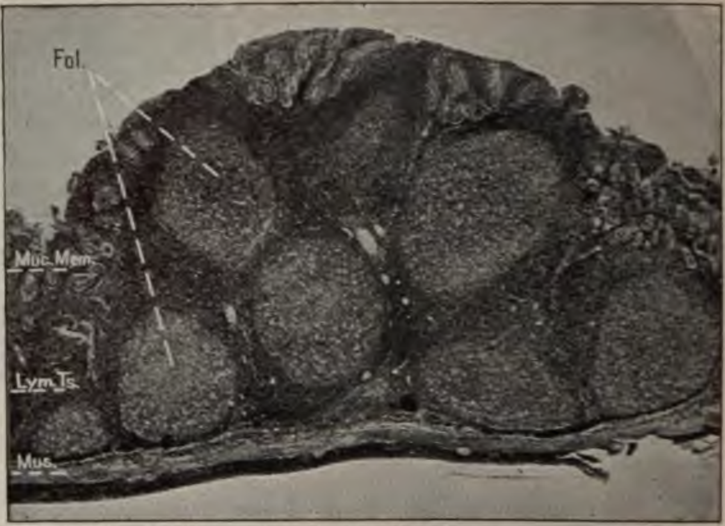
CASE 429. FIG. 115.

II.



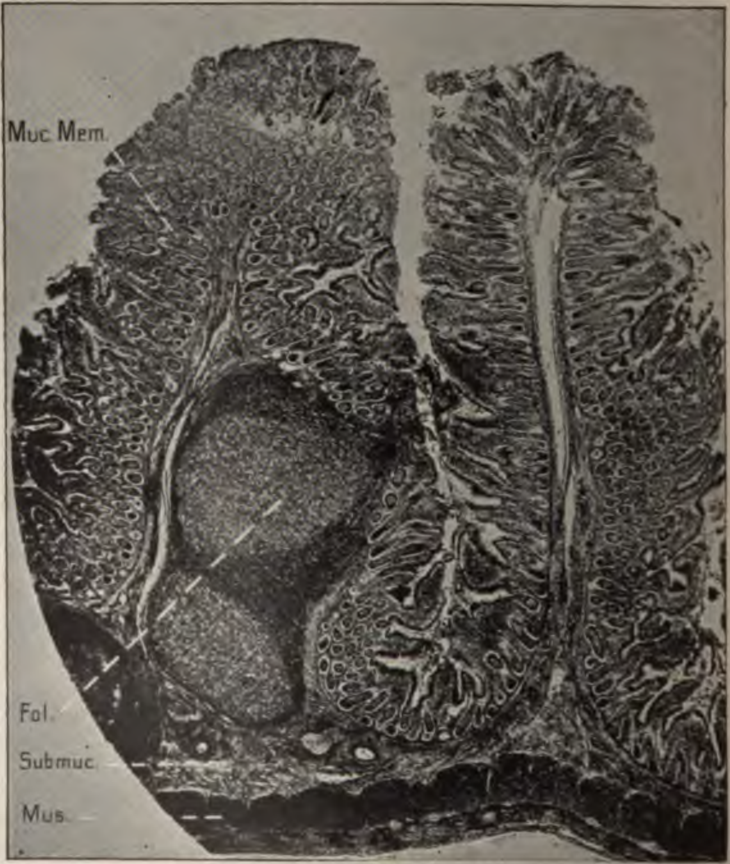
Colitis follicularis non-ulcerativa. (Page 893.)

Fig. 116



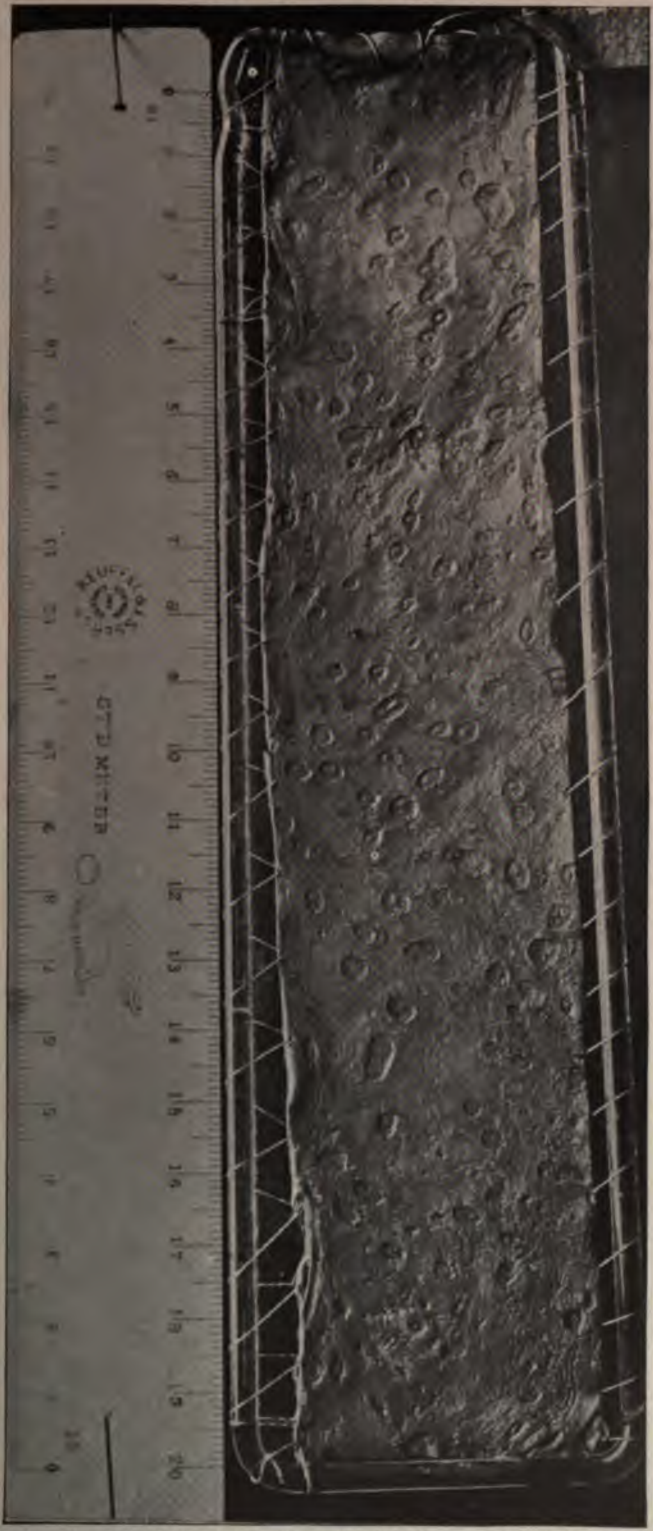
Hyperplasia of lymph-follicles (solitary glands). Muc. Mem., mucous membrane; Lym. Ts., lymph-tissues; Mus., muscle; Fol., follicles. (Page 893.)

Fig. 117.



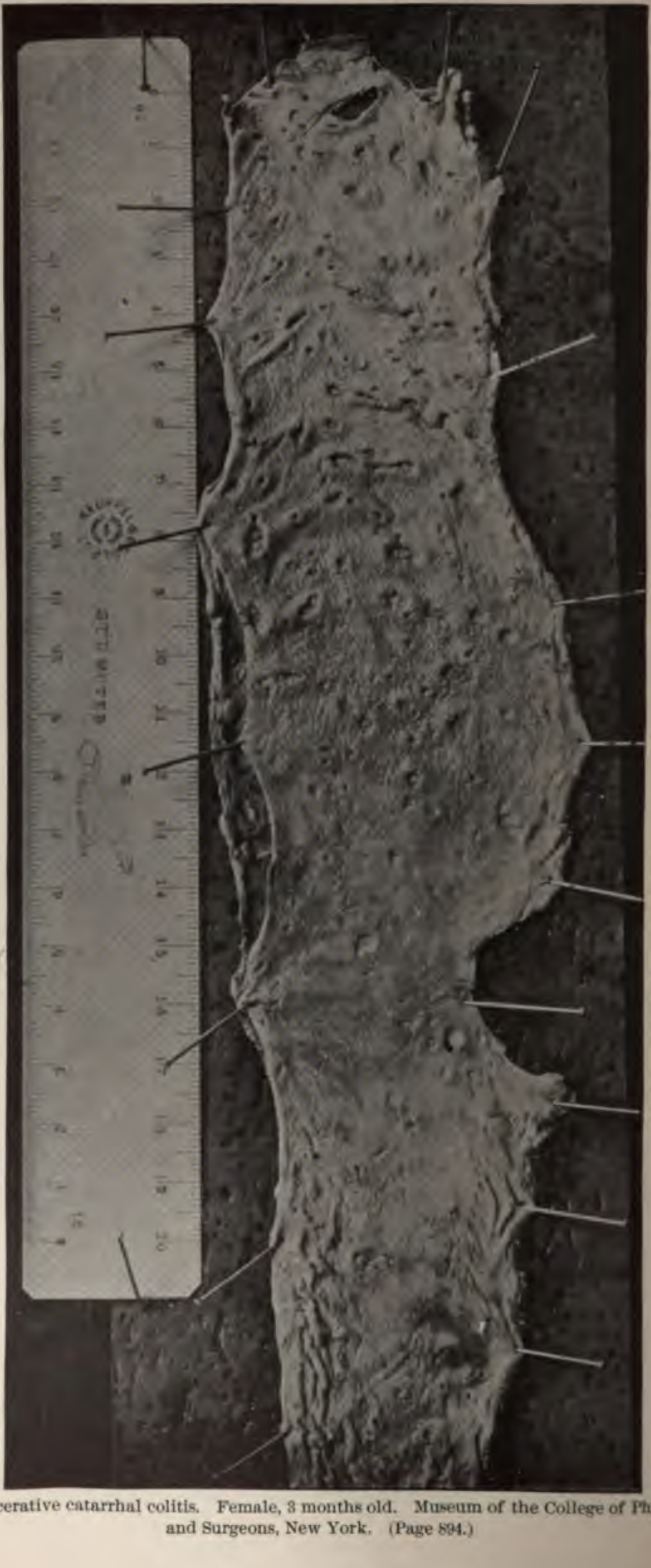
Muc. Mem., mucous membrane; Fol., follicles; Submuc., submucous tissue; Mus., muscle. (Page 893.)

CASE 430. FIG. 118.



Ileo-colitis ulcerativa follicularis. Infant, 16 months old. Museum of the College of Physicians and Surgeons, New York. (Page 893.)

CASE 431. FIG. 119.



Acute ulcerative catarrhal colitis. Female, 3 months old. Museum of the College of Physicians and Surgeons, New York. (Page 894.)

You see that the lesion is quite marked and simulates closely the hyper- lasia of Peyer's patches which is commonly seen in typhoid fever; but in is case it represents merely intestinal irritation.

This next specimen (Fig. 114) was found at the autopsy of a little girl, ree years old, who had been under the care of Dr. Webber.

The child (Case 428) was attacked with excessive vomiting after eating igs' feet, and the vomiting continued until her death, five days later. The sions are chiefly in the upper part of the colon, and consist of a general on-ulcerative follicular inflammation. The hyperplasia of Peyer's patches , as you see, extreme.

Through the kindness of Professor W. P. Northrup I am enabled to ow you some interesting specimens of lesions of colitis which occurred in is practice, and which are now preserved in the Museum of the College of hysicians and Surgeons, New York.

This specimen (Fig. 115) is one of acute catarrhal follicular inflamma- on without ulceration.

The infant, a male (Case 429), two years old, entered Professor Northrup's service ith a history of diarrhœa and general debility lasting two weeks. While the infant as in the hospital there was a continued high temperature, which at one time reached ° C. (104° F.). The symptoms were mostly of a cerebral type, and the abdominal symp- ms were not severe or prominent enough to indicate the marked lesions which were found the autopsy. The post-mortem examination, made by Professor Northrup, showed the llowing conditions:

Brain normal.

Stomach congested.

The small intestine contained a large amount of thick mucus. The solitary follicles ere enlarged, rather more in the upper third of the intestine. Peyer's patches were arkedly swollen, and a few solitary follicles appeared to be ulcerated. The mesenteric mph-glands were enlarged.

The mucous membrane of the colon was swollen: the follicles were enlarged and some- hat pigmented, but not ulcerated.

Here is another portion of the colon (Fig. 115, II.) taken from the same fant (Case 429).

As you see, the solitary follicles are very much enlarged, and in Peyer's atches, which are in the middle of the specimen, the hyperplasia is of a ery high degree.

I have also here some microscopic sections of this form of follicular flammation. In this first specimen (Fig. 116) you will see the great largement of the lymph-follicles.

In this next specimen (Fig. 117) you will notice the inflamed condition f the mucous membrane as well as the enlarged lymph-follicles.

This next specimen (Fig. 118) was taken from an infant (Case 430) six- en months old.

The infant before entering the hospital had had occasional attacks of diarrhœa for ree months, presumably caused by improper feeding. Soon after entering the hospital it pidly grew worse and died.

The autopsy, made by Dr. Northrup, gave the following results. No tubercular lesions. Bronchial lymph-follicles enlarged. Small intestine showed much swelling and congestion of Peyer's patches, but no ulceration. The colon showed extensive follicular ulcerations. In the small intestine and the colon were found masses and strings of greenish mucus; no blood.

This next specimen (Fig. 119) was taken from a female infant (Case 431), three months old.

The infant on entering the hospital was somewhat rhachitic, emaciated, and fretful. There were no vomiting and no fever. It took very little nourishment, and at this time was having one large, watery, fæcal discharge daily. The fæcal movements were greenish yellow. The infant apparently improved for about a week. The temperature was then found to have risen, and during the next week it varied from 36.6° to 37.7° C. (98° to 100° F.). During the next week the temperature was sometimes subnormal. At the end of three weeks the infant began to fail rapidly without any discoverable cause, and died suddenly.

The autopsy was made by Professor Northrup, and showed the following lesions: the mucous membrane of the ileum was swollen, and the lymph-follicles were enlarged, but not ulcerated.

The report of the examination of the colon, made by Professor Delafield, was as follows. Numerous ulcers, some round and some irregular in shape; an increased production of mucus: a profuse growth of connective tissue between the tubules, with dissappearance of the tubules; necrosis of the new tissue so as to form ulcers; the solitary follicles swollen, but not concerned in the formation of ulcers, which are simply necrotic. No amœbæ found. The process is one which would ordinarily come under the head of acute catarrhal colitis.

The next specimen (Fig. 120) is one which I am enabled to show you through the kindness of Professor Holt.

The infant (Case 432) was three months old, and was in the hospital under the care of Dr. Holt. It had no acute symptoms, but had never been well, and before entering the hospital had lost in weight and strength. It entered the hospital for vomiting and diarrhœa. Nothing was found on physical examination. While in the hospital it had from six to eight loose greenish discharges in the twenty-four hours, and vomited occasionally. Its temperature varied from 37.2° to 38.3° C. (99° to 101° F.). It gradually failed, and died twelve days after entering the hospital.

The post-mortem examination, as you see (Fig. 120), shows extensive follicular ulceration of the colon, especially in the lower part of the specimen, where there is a large ulcer. The tissues around the follicles are also involved, and the process has gone on to necrosis.

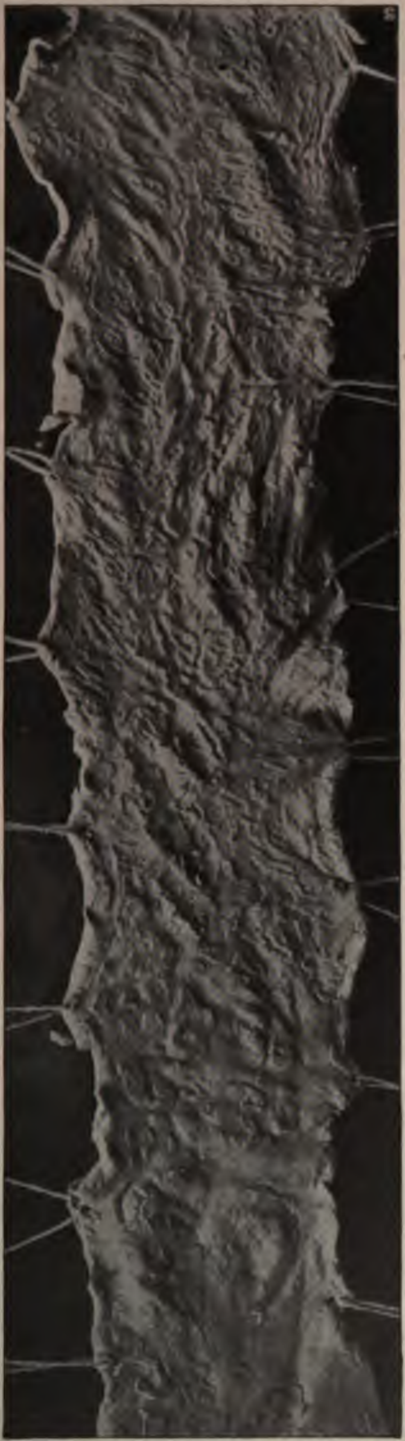
This next specimen (Fig. 121) was taken from a male infant (Case 433) six months old, also a patient of Dr. Northrup's.

The infant when it entered the hospital was in a very wasted condition, and died in a few days without any especial abdominal symptoms.

The autopsy, made by Dr. Northrup, showed numerous superficial abscesses on the body, a general bronchitis, and a beginning broncho-pneumonia. The lesions in the intestine were an inflammation of the solitary follicles of the ileum and of the colon, with small ulcerations at the apices of the follicles in the colon, no ulcers being present in the ileum. In the specimen these ulcers are, as you see, pigmented, which denotes a chronic condition.

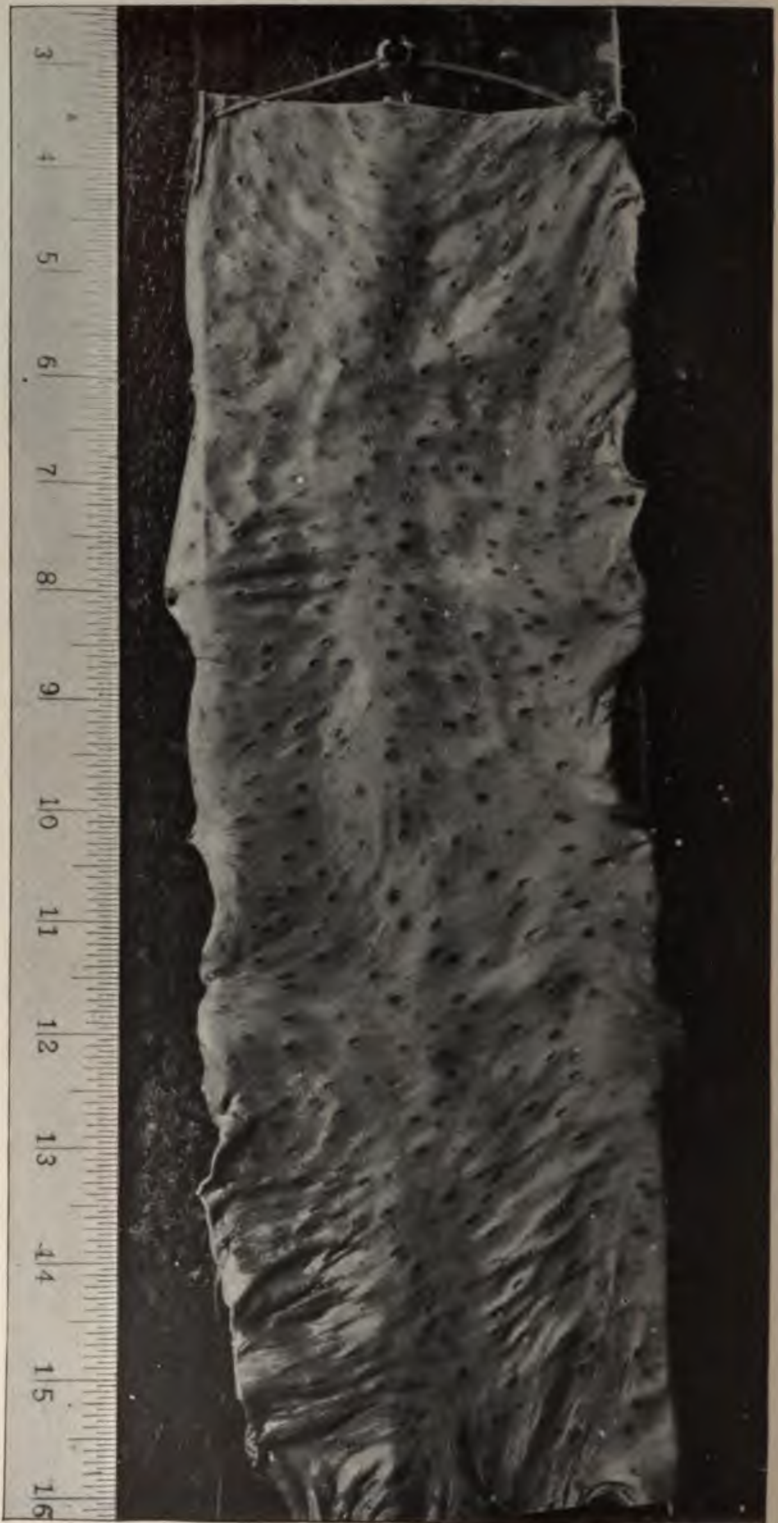
The apices of the follicles are sometimes found pigmented as the result of post-mortem changes, and may simulate these ulcerations.

CASE 432. FIG. 120.



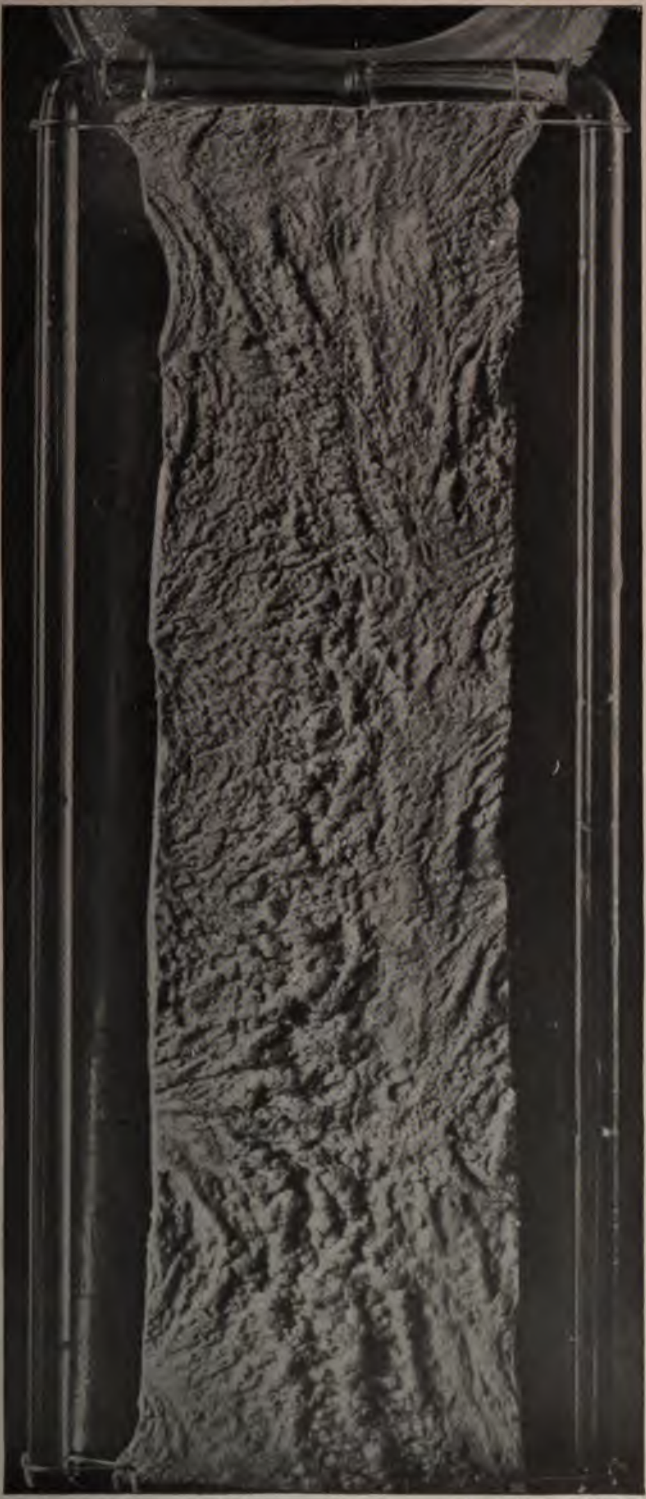
ation of follicles and surrounding parts of colon. The process has gone on to necrosis. Female, 3 months old. Warren Museum, Harvard University. (Page 894.)

CASE 433. FIG. 121.



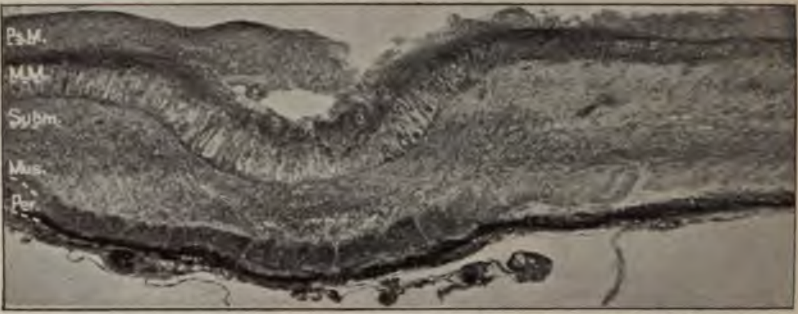
Pigmented follicular ulcers of colon. Chronic catarrhal ulcerative follicular colitis. Museum of the College of Physicians and Surgeons, New York. (Page 894.)

CASE 434. FIG. 122.



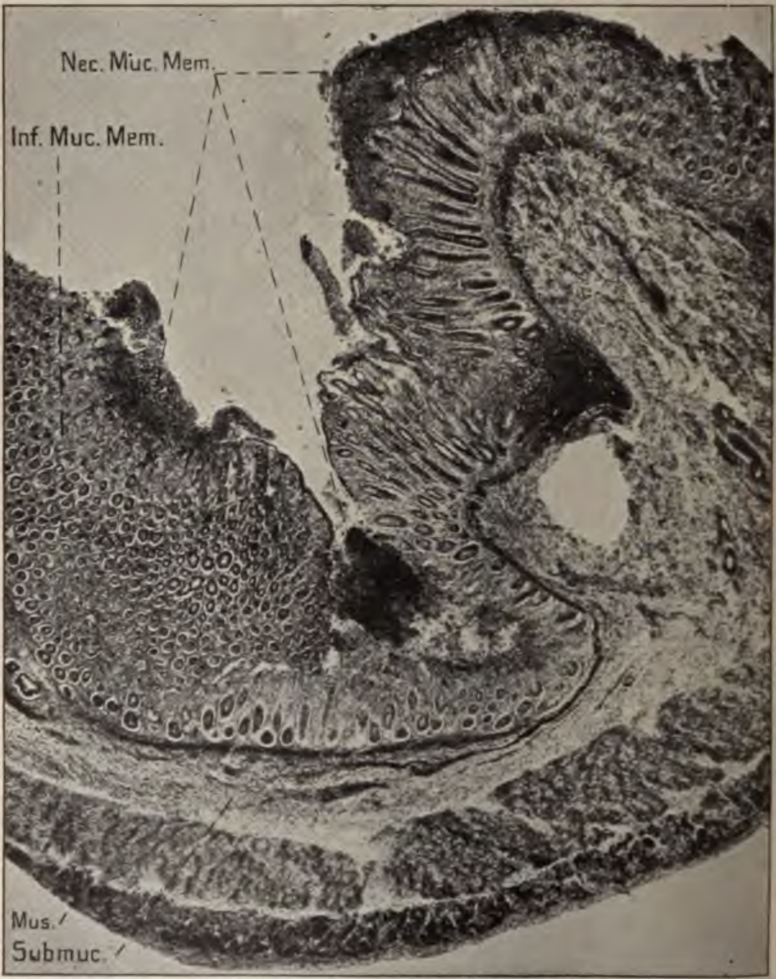
Pseudo-membranous colitis. Child, 3½ years old. Museum of the College of Physicians and Surgeons, New York. (Page 895.)

CASE 435. FIG. 123.



Pseudo-membranous colitis. Female, 4 years old. Ps. M., pseudo-membrane; M. M., mucous membrane; Subm., submucosa; Mus., muscle; Per., peritoneum. (Page 895.)

CASE 436. FIG. 124.



Nec. Muc. Mem., necrotic mucous membrane; Inf. Muc. Mem., inflamed mucous membrane; Mus., muscle; Submuc., submucosa. (Page 895.)

The next specimen (Fig. 122) is one of pseudo-membranous colitis.

This child (Case 434), three and a half years old, a patient of Dr. Northrup's, entered he hospital in a very reduced condition following an attack of whooping-cough. It was ttacked with diphtheria, and during the ten days that it was suffering from this disease here was a slight amount of diarrhœa, but no pain and no tenesmus.

The autopsy showed this pseudo-membranous inflammation through the whole length f the colon, most marked in the lower third. The other organs were normal. The microscopic examination of the colon confirmed the diagnosis of pseudo-membranous colitis.

I have here a microscopic section (Fig. 123) of another case (Case 435) of pseudo-membranous colitis.

This child, a female, four years old, was a patient of Professor Northrup's. It had always been delicate. It had pneumonia twice in its fourth year. Eight days before its death it was attacked with vomiting and diarrhœa. There was blood in the fæcal discharges. The pulse was rapid. The loss of strength and the pallor were marked. The eyes were sunken, and the tongue was dry. On the last day of its life it became very feeble, and died in convulsions. Early in the disease the discharges were frequent. Later, hey were from four to six daily, and were accompanied by tenesmus and tenderness of the abdomen.

The autopsy showed that the mesenteric lymph-follicles were not much enlarged; the follicles in the colon were slightly enlarged. The whole intestine was injected in patches, and contained fæcal masses of a yellowish color. The large intestine was filled with large quantities of fæces of foul odor and colored by bismuth. The whole surface was rough, and did not look like a mucous membrane, but rather as though a thin layer of gelatin had been poured over it. This film could be pulled away with the forceps. The solitary follicles were enlarged.

The microscopic section of this specimen shows a marked fibrino-purulent exudation, forming a membrane which characterizes the disease as pseudo-membranous colitis.

I have also here to show you, through the kindness of Professor Northrup, an interesting specimen (Fig. 124) of an intestinal lesion in connection with the pseudo-membranous condition which you have just seen.

This child (Case 436), three and a half years old, had whooping-cough. It was then attacked with diphtheria, and during the course of the disease the temperature was raised continuously, at times being as high as 40° C. (104° F.). During this attack it had diarrhœa with blood in the discharges, but no pain or tenesmus and no other symptoms of colitis.

The autopsy showed a broncho-pneumonia, and a normal condition of the stomach and small intestine. The colon showed an apparent exudation, which simulated that of a pseudo-membranous colitis so closely that before the microscopic examination was made it was supposed to be identical with the pathological lesions found in the case of pseudo-membranous colitis (Case 435) which I have just shown you. The surface appearance in the fresh specimen was identical. Under the microscope, however, the lesion proved to be only a superficial necrosis of the mucosa, with swelling of the lymph-follicles.

This specimen should impress upon you how important it is not to rely upon the macroscopic appearances of intestinal lesions without microscopic corroboration.

Now that you have seen these pathological lesions, you will understand why it is often impossible to differentiate them clinically from one another. I shall, therefore, speak of them together.

As illustrations of the difficulty and in many instances the impossibility of diagnosticating intestinal lesions I shall report to you some cases which have been under my care.

One of these cases was that of a little girl (Case 437), five years old, who during the hot weather in August had been having a slight attack of fermental diarrhœa, which began with vomiting, headache, and a slight rise of temperature lasting a few hours. This was soon followed by four or five greenish-yellow discharges in the twenty-four hours, and a normal temperature. The diarrhœa diminished in two or three days, and the child seemed much better, but after a few days she was suddenly attacked with a temperature of 39.4° to 40° C. (103° to 104° F.) and with frequent discharges of mucus and blood. She lost rapidly in weight, and looked very sick. After twenty-four hours, however, the movements became normal; and on the following day, although left weak and prostrated, she seemed perfectly well, and had no return of the attack. During the acute symptoms it seemed as if she were attacked by one of the more severe forms of colitis, but the rapid recovery left the diagnosis very doubtful.

The next case was that of a child (Case 438), seven years old, who entered my wards at the City Hospital with a history of having had a slight diarrhœa for a few days. The temperature was but slightly raised. The movements were infrequent, of a greenish-yellow color, and contained no blood or membrane, and scarcely any mucus. The child seemed fairly well on entering the hospital, but during the following few days became much exhausted. Although no other intestinal symptoms appeared, he sank rapidly, and died apparently from exhaustion.

The autopsy showed extensive lesions of the whole colon, the mucous membrane was greatly thickened, and there were numerous ulcerations.

The third case was that of a boy (Case 439), four years old, who was brought to the Children's Hospital for frequent vomiting following an attack of diphtheria. During the first three weeks that he was in the hospital the vomiting was the chief symptom. He was fed by nutritive enemata and improved in his general strength. Later, however, he became very much emaciated, the vomiting increased in frequency, and a few days before he died there was a slight diarrhœa. The temperature was normal or subnormal during the whole course of the disease.

During the last four or five days the symptoms had pointed almost entirely to the stomach, but the post-mortem examination showed nothing abnormal in the stomach, lungs, heart, kidneys, or spleen. The mesenteric glands were swollen in the region of the ileocæcal valve. The walls of the ileum and colon were thickened and reddened. There was a slight deposit of fibrin over part of the mucous membrane of the ileum. The lower 35 cm. (13¾ inches) of the colon were found to be much thickened, the inner surface was of a dark-greenish color, and beneath it the tissue was deeply injected. The thickening seemed largely due to an exudation on the mucous membrane, which could not be torn away. The thickening ended quite sharply, but on some of the valvulæ conniventes above a similar membranous deposit could be found. In the colon the thickening was most marked in the cæcum and the rectum, and least so in the transverse colon, and the process seemed older than in the ileum. Cultures from the various organs were negative. Various organisms were found in the ileum, but none that seemed to be of especial significance.

ETIOLOGY.—The etiology of these diseases I have already described under general etiology.

SYMPTOMS.—The symptoms of these forms of acute inflammatory ileocolitis vary greatly, as a rule, but in a general way they can be recognized by a group of symptoms which differ from those of the non-inflammatory diarrhœas spoken of as fermental diarrhœa and cholera infantum. The best work which has been done on the symptomatology of these diseases

at by Holt, but we still find that the symptoms of these different
s of ileo-colitis are very unsatisfactory and unreliable for differential
iosis.

'he onset of the disease may be preceded by a fermental diarrhœa, or it
be acute from the beginning and have prodromal symptoms of no
than a few hours. The temperature is elevated, the pulse is quickened,
the infant loses rapidly in weight and strength. The discharges are
ips ten or twenty, or even more, in the twenty-four hours. Where the
is are in the rectum there is tenesmus both before and after the dis-
ge, and in the beginning of the attack an almost continuous desire to
a movement. The discharges contain fæcal matter at first, but soon
ne small, and consist of mucus, with sometimes pus, blood, and shreds
embrane. The odor may be very offensive, but when the mucus pre-
nates there is very little odor. The color and consistency are extremely
ble, but generally the consistency is lessened and the color is a mix-
of green, brown, and yellow. The blood is usually from congestion
e blood-vessels and straining, rather than from ulceration. Therefore
annot determine from the presence of blood whether ulceration is pres-
r not. At first the abdomen may be soft and not tender, but later in
lisease it becomes distended, tympanitic, and somewhat tender, espe-
r along the course of the colon. Vomiting may occur at times. In
e cases the child is very restless, and there may be delirium and con-
ons. The appetite is usually much lessened. The urine is nearly
ys lessened in quantity, is high-colored, and sometimes contains a
l amount of albumin, especially when the temperature is high. Acute
ritis is, however, rare in these cases. Where there is much tenesmus
straining, and where the discharges are especially frequent, prolapse of
ectum may occur. The discharges often cause great irritation around
nus and on the buttocks.

IAGNOSIS.—These forms of ileo-colitis are diagnosticated from the
ental diarrhœas by the continued heightened temperature, the more
ent discharges, the small amount in each, the presence of blood or
brane, and the tenesmus. They may be differentiated from cholera
tum by the continuous and excessive vomiting and the serous dis-
ges of the latter disease.

ROGNOSIS.—The prognosis of ileo-colitis, where ulceration has not
red, is usually favorable, the duration of the disease being a few weeks.
cases, however, are more severe, and sometimes prove fatal in a few
Where there is ulceration, the prognosis is rather unfavorable.
re there is a diminution in the frequency of the discharges and fæcal
er begins to reappear, and where the nervous symptoms and exhaustion
n, the prognosis is good; but where the symptoms increase in severity
he face looks pinched, where intractable vomiting arises and the nervous
toms predominate, the prognosis is very unfavorable.

he prognosis is less favorable where the ileo-colitis is complicated by broncho-pneumonia or tuberculosis. It is much influenced by the time of the year at which the attack takes place, the prognosis being worse if the disease occurs at a time when the convalescence is during a long heated period. The prognosis is also worse where the infants have to be treated in crowded cities and in the midst of unsanitary surroundings.

Although there are no symptoms typical of the different forms of acute ileo-colitis, yet their clinical pictures differ somewhat.

It is usually found in the simple catarrhal ileo-colitis, where ulceration has not taken place, that the symptoms are milder and that there is apt to be vomiting. These cases generally begin to improve in one or two weeks, and recover entirely in another week. An intestinal disturbance of a mild character may result, however, and prolong the disease. The children are usually a long time in regaining their strength, and relapses are quite common in this form if the diet is not carefully regulated.

Sometimes, however, simple catarrhal ileo-colitis may be represented by symptoms of a very severe type, and it may run a rapid course, and end fatally.

Where follicular ulceration has taken place the stomach is not apt to be much involved, the temperature is not, as a rule, high, and the course of the disease is rather slow, irregular, and prolonged. The infant fails steadily, and commonly dies. A remission in the symptoms and an improvement in the character of the fæcal discharges should lead us to infer that ulceration has not taken place. Where the inflammation is simply follicular, without ulceration, the cases are very apt to recover.

Pseudo-membranous ileo-colitis is rare in infants, but when it occurs it is the most severe of all the forms. I have already stated that it is this form which is usually spoken of as epidemic or sporadic dysentery. The temperature is high,—39.4°, 40°, or 40.5° C. (103°, 104°, or 105° F.). There are apt to be blood and membranous detritus in the discharges. The progress of the disease is usually rapid and without remission, and death may take place in a week or ten days. The nervous symptoms, such as restlessness and delirium, are quite prominent. The diagnosis of this class of cases, as I have just told you, can be made positively only by finding shreds of membrane in the discharges.

TREATMENT.—The treatment of these forms of ileo-colitis should usually be in the beginning the same that I have already described for fermental diarrhœa. It may in this sense be spoken of as prophylactic, for in a large number of cases the organisms which produce ileo-colitis find a means of entrance through the irritated mucous membrane produced by a preceding fermental diarrhœa. Where the case is seen in its earlier stages, a mild laxative should be given, in order to clear away, as far as possible, the pathogenic organisms, which are present in large numbers. Small doses of castor oil act most efficiently, and can usually be given, especially to infants, without causing nausea or gastric irritation.

In addition to this treatment by the mouth, thorough irrigation of the colon should be employed. This should be done twice in the twenty-four hours with warm sterilized water containing 3.75 grammes (1 drachm) of borate of sodium to the pint of water. One or two gallons of water should be allowed to flow in and out of the intestine at each irrigation. After the irrigation, small enemata of thin mucilage, about 120 c.c. (4 ounces), containing 15 c.c. (½ ounce) of bismuth in suspension, may be given once in three or four hours.

According to the degree of pain, restlessness, and general discomfort, a slight amount of opium can be given in these injections, but in all cases this drug should be administered with great care; one drop of tincture of opium in the first year, and two drops in the second year, once in five or six hours, will usually be sufficient to make the infant comfortable. The effect of the opium should be carefully watched, and the dose increased or decreased as is necessary.

Where the tenesmus is extreme, it is well to use suppositories containing from 0.015 to 0.03 gramme (¼ to ½ grain) of cocaine. These suppositories will often give great relief if the painful lesions are mostly in the rectum, but where the lesions are higher in the colon they are not of much value.

The use of antiseptics by the mouth I do not recommend. Bismuth can be given by the mouth with some advantage in these cases, but the dose must be considerable to accomplish good results. One-half drachm in the twenty-four hours should be given to a child a year old, and for older children the dose should be proportionately increased. Alcoholic stimulants can be given with benefit at all stages of the disease if there is evidence of a weakened heart, or if much exhaustion is present.

A very limited amount of food of any kind should be given during the first twenty-four hours. Sterilized water containing an alcoholic stimulant and barley water had better be given at first, as it has been found that where a sterile liquid is taken by the mouth the number of bacteria in the intestine diminishes rapidly. When a perfectly fresh milk can be obtained it can be used, if sterilized and modified in its various elements so as to be adapted to the digestion of the especial case. A moderate percentage of fat and sugar, such as 3 and 5, and a proteid percentage of about 2, is a very good prescription to begin with. Weak broths can also be given.

In some cases of ileo-colitis, after the acute symptoms have ceased the diarrhœa continues for many months and the disease becomes chronic. In these cases the temperature may be normal, and there is no especial pain or tenderness. The appetite often returns, but the child does not gain in weight, or it loses. The discharges are not so frequent as during the acute stage of the disease, varying from six or eight to two or three in the twenty-four hours. The discharges have a lessened consistency, and contain mucus and undigested food. There may at times be exacerbations of the symptoms, and the children are very apt to die of some intercurrent disease.

The treatment is change of air if possible. and otherwise is essentially dietetic. The rules which I have already given you in speaking of the treatment of fermental cases are applicable also to this class of cases.

The pathological conditions most commonly found in these chronic forms of ileo-colitis are great thickening of the muscular tissue, pigmentation of the mucous membrane, and very extensive ulceration.

Amœbic Ileo-Colitis.—The next form of ileo-colitis which I shall speak of is the *amœbic.* It has its own definite anatomical lesions, which are usually in the colon.

The disease is caused by a well-recognized organism, called the *amœba coli.* It is very rare in northern climates, and is most frequently met with in tropical countries. A frequent source of infection by the amœba coli is drinking-water.

I have here a specimen (Plate III., Fig. c, facing page 112) from the intestine of a case of amœbic ileo-colitis. The large round bodies which you see lying in more or less clear spaces are the amœbæ coli. The organism can also be detected by directly examining the discharges under the microscope.

The characteristic pathological lesion of this form of ileo-colitis is the peculiar, undermined condition of the edge of the ulcers and of the mucous membrane. The amœbæ are found not only in the intestine, but also in the various organs, and with especial frequency in the liver.

The disease is usually acute in its onset, but sometimes it may be gradual. The duration may be two or three months.

There are no especial symptoms by which to distinguish this form of ileo-colitis from the others of which I have just spoken, and the only positive proof of the existence of the disease is the presence of the amœbæ in the discharges.

The disease is rare in children, and the prognosis is very unfavorable.

The treatment which has been followed by the most favorable results is, in addition to frequent and thorough irrigation of the intestine, injections of solutions of sulphate of quinine (1 to 5000). This treatment, however, affects only the amœbæ which are in the intestine, and not those which are embedded in the tissues.

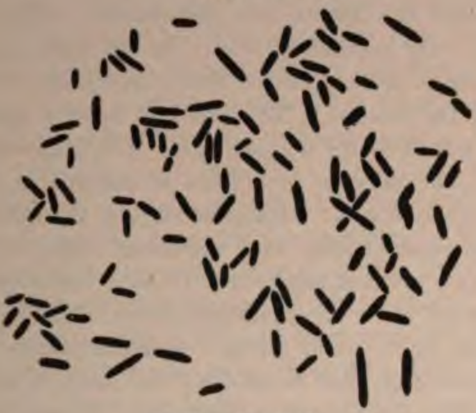
Typhoidal Ileo-Colitis (Typhoid Fever).—The typhoidal form of acute ileo-colitis is an infectious disease with a definite pathology, and is characterized by constant changes in the lymph-follicles, chiefly at the lower end of the ileum, in the mesenteric lymph-glands, and in the spleen. The disease is produced by the bacillus of Eberth, which is constantly present in the lesions. Infection takes place largely through the gastro-enteric tract. The usual mode of conveyance into the body for the typhoid poison is infected milk or water. Typhoid fever is exceedingly rare in the first two years of life, is uncommon under three years, and after the third year becomes more common as the child grows older.

I have here a specimen (Fig. 125) of the bacillus of typhoid, showing its morphology.

It is about three times as long as it is broad, and is about one-third as long as the diameter of a red blood-corpuscle. It is rounded at the ends.

The pronounced pathological lesions, severe symptoms, and great violence in type which are so characteristic of the typhoid fever of later years are so rare in infancy and early life that I shall confine myself in what I have to say concerning this disease to the conditions which it presents in the latter period.

FIG. 125.



Bacillus of typhoid.

PATHOLOGY.—Although the more advanced and severe lesions of typhoid fever may occur in the early as well as in the later years of life, yet its characteristic lesions in young subjects are the milder and less severe pathological changes of the disease. These consist essentially of a hyperplasia of the solitary lymph-follicles and Peyer's patches, and the process, instead of going on to ulceration, usually terminates in early resolution with fatty degeneration of the cells. Hemorrhage and perforation are therefore rare complications in the typhoid fever of early life. There is, however, nothing distinctive of typhoid fever in this hyperplasia of the lymph-follicles in children, for it is not uncommon to find this condition where death has occurred from other diseases of the intestine. It may also be present in such diseases as measles, diphtheria, and scarlet fever. Very marked hyperplasia of the lymph-follicles may be produced in children by irritating substances and by foreign bodies, not only food, but also drugs, such as turpeth mineral. I have, in fact, seen, at the post-mortem examination of a child, marked enlargement of the lymph-follicles caused by doses of turpeth mineral given during life as an emetic. The pathological conditions in typhoid fever in the early years of life may be said to correspond to those which are met with in the aborted forms of the disease in later life.

SYMPTOMS.—The stage of incubation of the disease lasts from one to two weeks. The symptoms are, generally, not severe. The prodromal stage is usually short, young subjects having less power of resistance to the poison than adults, in whom the prodromal stage is often prolonged. As a rule, the temperature is moderate, but it may be high, as in adults, without, however, producing as severe symptoms, since children, commonly, are less affected by a high temperature than adults.

The duration of the disease is generally much shorter than in adults. This short duration depends largely upon the mild form of the intestinal lesions, and usually shows that marked ulceration has not taken place. The temperature chart in this mild typical form of the disease is not apt to be so regular as where the lesions are pronounced. The temperature, although it returns to the normal by lysis, does not show so gradual a lysis as where marked lesions have occurred and where other sources of toxæmia have complicated the disease. The pulse is usually quickened in correspondence to the height of the temperature. The respirations are not especially increased. The nervous symptoms so marked in later life are not prominent in early childhood. Headache slighter than that in adults may occur. Delirium, convulsions, and vomiting may be present. These symptoms, however, are not common. In some cases cerebral symptoms simulating somewhat those of meningitis arise, and are probably due to cerebral congestion or to toxic action. Aphasia occurs rather more frequently in young children than in adults. Its cause is not known, and it appears usually when the disease is declining. It may last for one or two weeks.

The characteristic of typhoid fever in young children, as I have seen it, is apathy. The child takes the nourishment which is given to it, is not especially restless, and usually lies in a half-somnolent condition. As the disease progresses, it gradually returns to a more natural mental condition.

Although it is probable that in most cases of typhoid fever there is some enlargement of the spleen, it is often impossible to detect this change by palpation, and percussion of the spleen in young children is well known to be very misleading. In some cases the rose-colored spots appear on the abdomen, but in quite a number I have been unable to detect them. There is apt to be a slight bronchial catarrh. The bowels are often constipated, though sometimes diarrhœa is present. The tongue is not so likely to be dry as in older subjects, and, although coated, it soon becomes clean at the tip and edges. The abdomen may be somewhat distended and tympanitic, but this symptom is often not marked, and pain and tenderness are rather infrequent. Epistaxis is rare in the typhoid fever of children. There is at times a slight albuminuria during the height of the fever, but a complicating nephritis is rare.

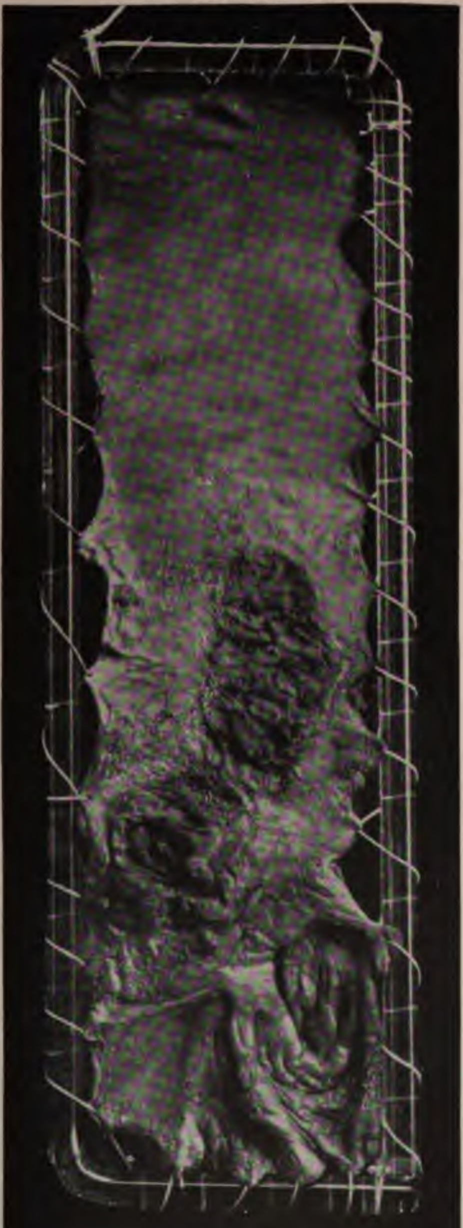
DIAGNOSIS.—During the first few days, typhoidal ileo-colitis may often be mistaken for various forms of febrile gastro-enteric disease. A number of acute diseases, such as the exanthemata and pneumonia, may simulate in their prodromal stages those cases of typhoid fever which begin with violent symptoms. The vomiting which occurs in the prodromal stage of typhoid fever may, in connection with the child's apathy, simulate the arly stages of tubercular meningitis. It is therefore often impossible, in he early days of the disease, to make a positive diagnosis, and in some ases we are left in doubt as to the diagnosis for a week or ten days. he characteristic symptoms of the acute diseases already referred to, and f tubercular meningitis, will later be so apparent as to leave the diagnosis o longer doubtful. Jacobi lays stress on the probability of typhoid when here is a continuous high fever which is well borne by the infant, and hen the intestinal symptoms are not violent. We should also remember hat the differential diagnosis between the typhoidal form of ileo-colitis and alaria is at times, especially in children under two years of age, very ifficult, and perhaps impossible, until the blood has been examined. Epi-emic influenza may in its onset simulate typhoid fever, but the period of oubt is very short. Acute miliary tuberculosis may in its typhoid type imulate typhoidal ileo-colitis very closely. Where, in the latter disease, he rose-colored spots do not appear, the delirium, distended abdomen, nlarged spleen, and even the irregular temperature, at times common to oth diseases, may make the resemblance so close that the differential diag-osis can not be made until the post-mortem examination.

PROGNOSIS.—The prognosis of typhoid fever in early childhood is good. he complications, whether arising from local disturbance of the intestine r from cardiac and pulmonary disease, are rare in comparison with those et with in later life. You must remember, however, that the disease aries very much in its severity in different epidemics and in different indi-iduals, and that a child may have a severe type of typhoid fever and die rom it.

TREATMENT.—The treatment of the typical mild form of typhoid fever n young children is to keep the child in bed and to feed it regularly every wo or three hours with fresh milk, modified according to the condition of ts digestion, and heated to 75° C. (167° F.). As a rule, antipyretic drugs hould not be used. The child should be bathed at least twice a day ith water at a temperature of 32.2° C. (90° F.), not necessarily for the urpose of reducing the temperature, but as a hygienic measure. In most f the cases which I have seen this is all the treatment that has been found ecessary from the beginning to the end of the disease. Where there are reat restlessness and delirium, with a high temperature, 40.5° to 41° C. 105° to 106° F.), baths should be given once every three or four hours, but he temperature of the water should not be below 26.6° to 29.4° C. (80° to 5° F.), as this is usually sufficient to allay the symptoms. If the tem-erature remains high and there are symptoms of serious import, such as ccur in the advanced stages of the adult type of typhoid fever, colder ater can be used; but, as a rule, it is not wise to employ water of a low emperature in children to the extent to which it has been found useful in dults. Alcoholic stimulants should be given where there are signs of xhaustion.

The various complications which arise should be treated symptomatically.

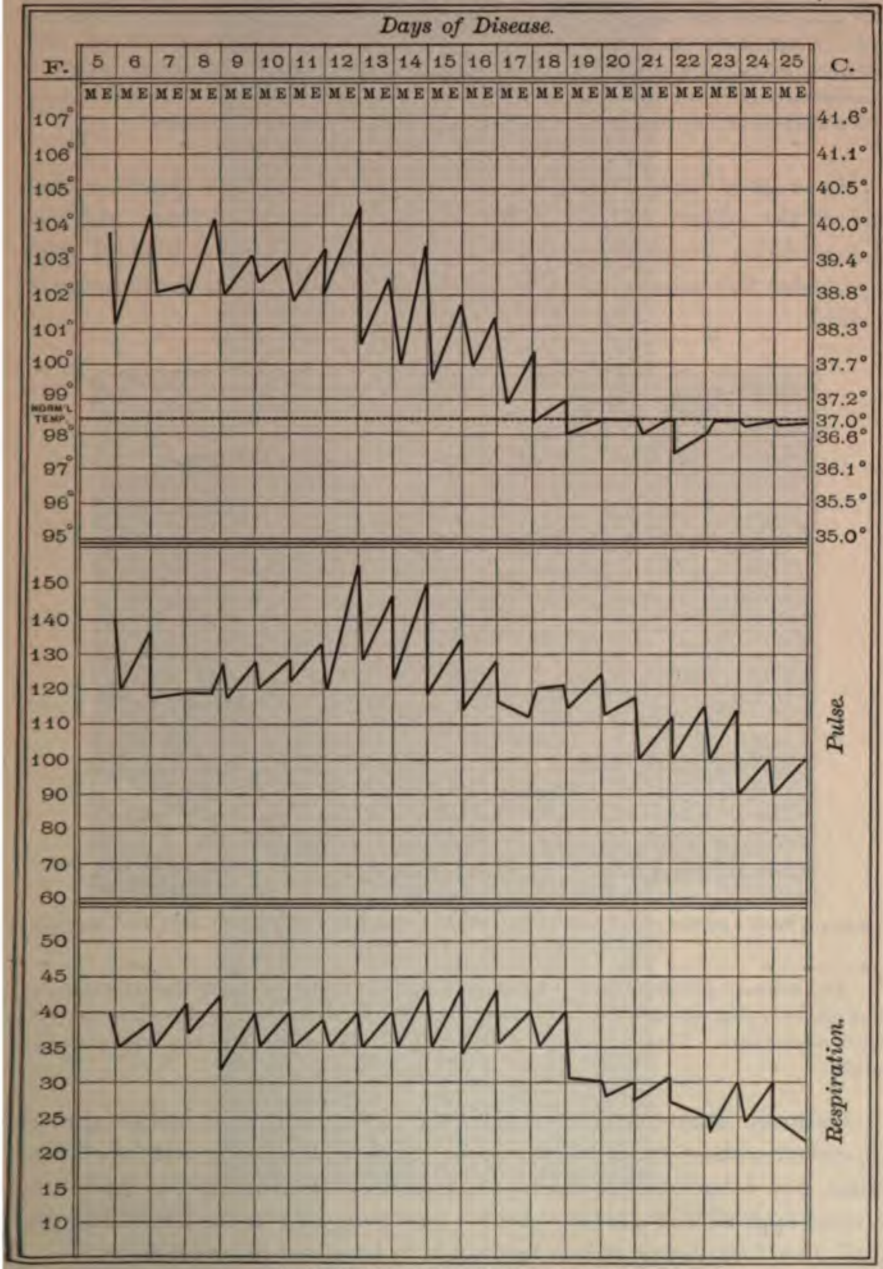
CASE 441. FIG. 126.



Typhoidal ileo-colitis, showing ulcers of colon. Female, 2 years old.
U. S. Army Medical Museum.

Through the kindness of Dr. Billings I am enabled to show you this intestine (Fig. 126), which was taken from an infant with typhoidal ileo-colitis.

CHART 31.

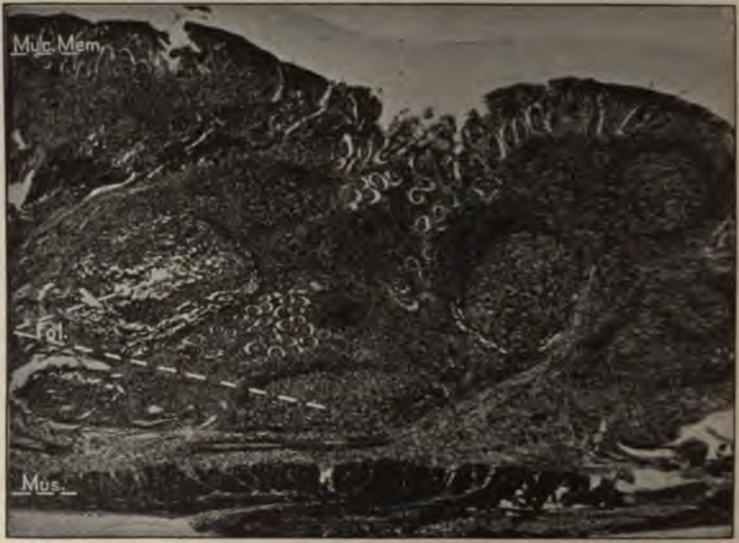


Typhoidal ileo-colitis. Male, 5 years old.

The infant (Case 441) was a patient of Dr. S. S. Adams, of Washington. In this case the irregularity of the temperature curve and the prominent symptoms of cerebro-spinal irritation rendered the diagnosis so obscure that typhoid fever was not suspected until a few days before death. The post-mortem examination showed marked congestion of the entire brain, chiefly on the right side. The left hemisphere was covered with a gluey substance which filled the sulci and was especially abundant around the Sylvian fissure. The heart was normal. The lungs showed marked hypostatic congestion. The liver was normal. The gall-bladder was empty and pale. The spleen was enlarged. The kidneys were normal. The stomach was congested. The mesenteric glands were enlarged and soft. The intestines contained a quantity of yellowish watery fæces. The lesions which you see in this specimen are in the ileo-colic portion of the intestine. You see that there is thickening and ulceration of Peyer's patches, and to a less extent of the solitary follicles.

In order to impress upon you that in infants swelling of Peyer's patches and of the solitary follicles is not distinctive of typhoid fever, and that this condition frequently occurs from irritations of various kinds, I show you under this microscope (Fig. 127) a section taken from the intestine of a child.

Fig. 127.



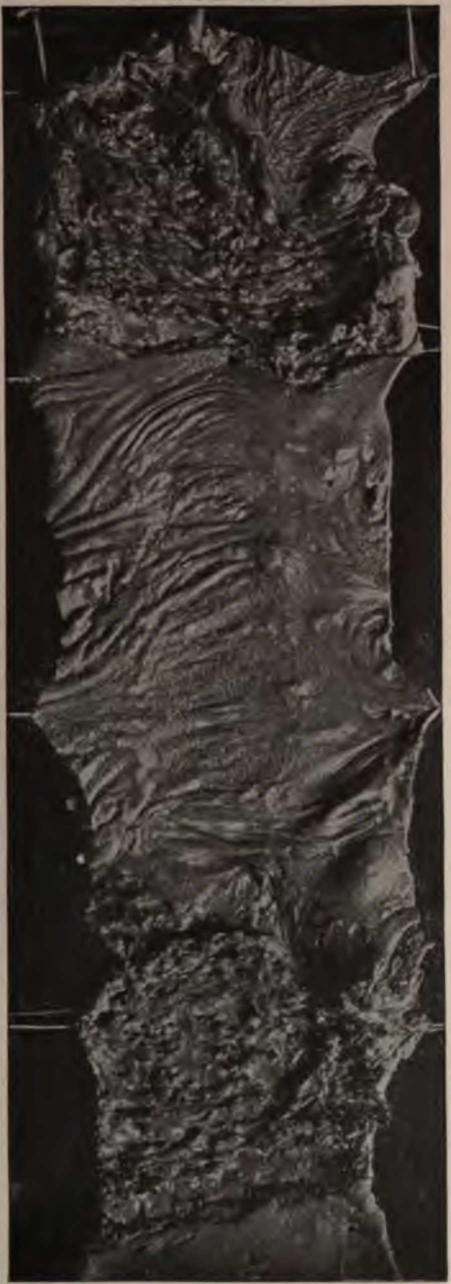
Enlarged Peyer's patches closely simulating the lesions of typhoidal ileo-colitis. Muc. Mem., mucous membrane; Fol., enlarged follicles; Mus., muscle.

The macroscopic appearances of this intestine so closely simulated the early stage of typhoid fever that cultures were made from it by Dr. Prudden to settle this question. No bacilli were found. This condition is often found in children in acute non-typhoidal ileo-colitis.

Chronic Ileo-Colitis.—Under chronic ileo-colitis we include chronic appendicitis, those forms of ileo-colitis which follow acute attacks of ileo-colitis, and tubercular disease of the intestine. I have already spoken of chronic appendicitis and the chronic form following acute ileo-colitis, and shall, therefore, devote only a few words to intestinal tuberculosis.

Tubercular Ileo-Colitis.—Tubercle of the intestine in infancy and childhood is quite common. The small intestine is most frequently involved. The disease may be primary in the intestine, but this is very

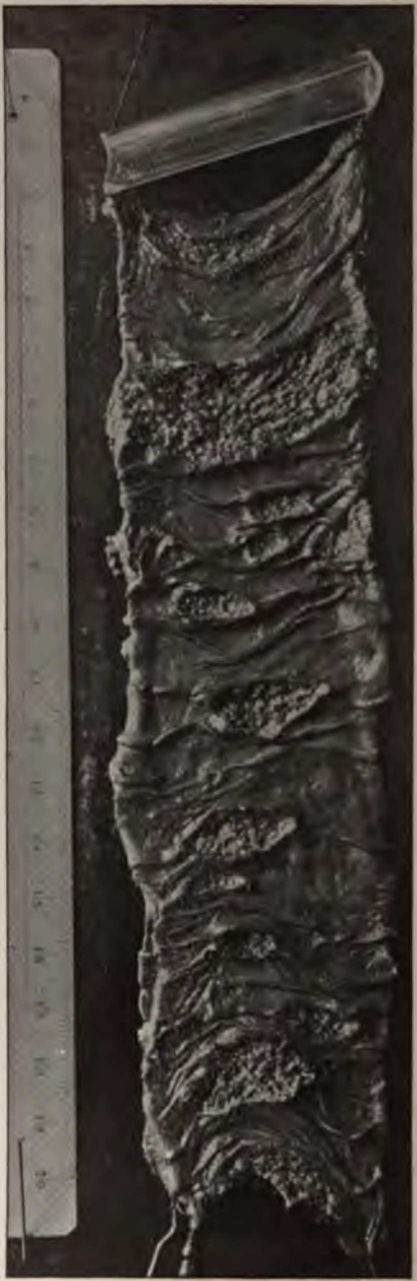
CASE 442. FIG. 128.



Tubercular ulcers of colon. Female, 8½ years old. Museum of the College of Physicians and Surgeons, New York.

CASE 443. FIG. 129.

I.



Tubercular ulcer of small intestine. Female, 2¾ years old. Museum of the College of Physicians and Surgeons, New York.

rare. At the Boston Children's Hospital I have had one case where the tubercular lesions were confined to the intestine and to the mesenteric glands. In this case Professor Councilman considered that the evidence was in favor of the intestinal tubercle antedating the tubercle of the glands. In the great majority of cases the tubercular ileo-colitis is secondary to tuberculosis elsewhere, and in such cases frequently follows tuberculosis of the mesenteric glands.

PATHOLOGY.—According to Osler, the ulcers are situated chiefly in the ileum, and involve the solitary follicles and Peyer's patches. The tubercular ulcer has the following characteristics. In contradistinction to the typhoidal ulcer, the long diameter of which coincides with the long axis of the intestine, the tubercular ulcer is transverse to the long axis, rarely ovoid, and often irregular in outline. The edges overhang, and the base is infiltrated.

Through the kindness of Professor Northrup, I have here some specimens of tubercular ulcers. This first specimen (Fig. 128, facing page 906) was taken from a female (Case 442) eight and one-half years old.

This child, a patient of Dr. Northrup's, was attacked, two months before her death, with chills, fever, and prostration. The temperature at first varied from 38.8° to 39.4° C. (102° to 103° F.), but as the disease progressed the temperature gradually fell. There was rapid emaciation. The abdomen was sunken at first, but later became tense. There were pain, tenderness, and resistance in the right inguinal region. The submaxillary, cervical, and inguinal lymph-glands were enlarged. The urine contained albumin and hyaline casts. There was diarrhœa. The autopsy showed the lungs to be normal. The bronchial and retroperitoneal lymph-glands were enlarged and cheesy. The colon shows two large, sloughing ulcers, one in the region of the cæcum and the other in the ascending portion. You see that they are transverse to the axis of the colon, and that their edges are overhanging. The entire membrane is thickened, and there is some follicular ulceration.

This next specimen (Fig. 129) was also taken from a patient of Dr. Northrup's.

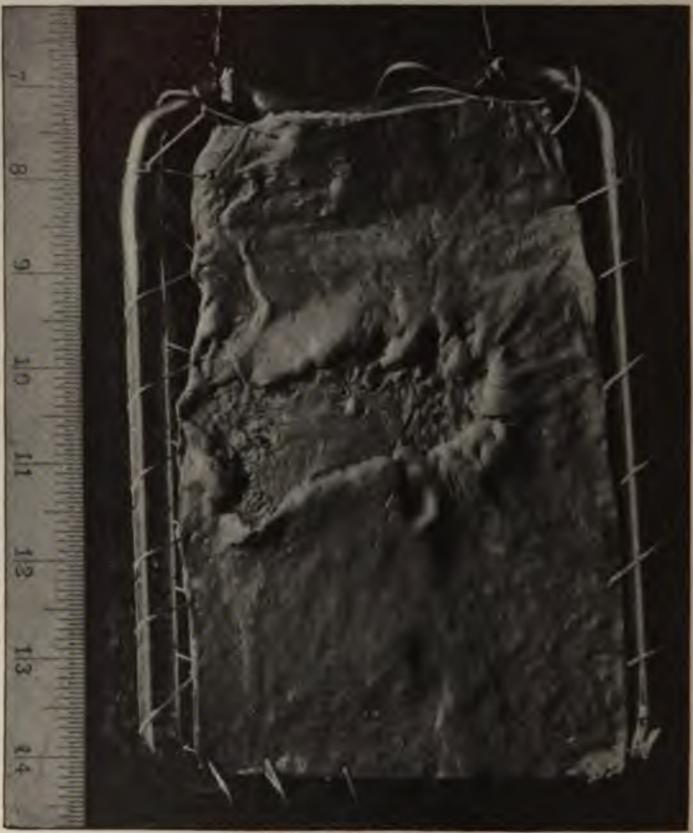
The child (Case 443), a female, two years and eight months old, had had diarrhœa occasionally for a year. It had also had convulsions. It died soon after entering the hospital. The autopsy showed these extensive ulcerations in the small intestine (I.) and this large ulcer in the cæcum (II., page 908). There were also tubercular ulcers in the middle third of the colon.

The peritoneal surface showed miliary tubercles. The mesenteric and bronchial lymph-glands were markedly enlarged and cheesy.

SYMPTOMS.—The symptoms of tubercular ileo-colitis are varied and rather indefinite. The most common symptom is a persistent diarrhœa. The diarrhœa does not, however, correspond to the extent of the lesions, as large ulcers may exist and constipation be present, especially if they are in the ileum. In cases of primary tubercle of the intestine the only sure means of determining the tubercular character of the disease is the finding of the bacillus tuberculosis in the discharges. Where the disease is secondary to tuberculosis elsewhere, the tubercular involvement of the intestine may be suspected when at any time during the course of the disease the infant is attacked with diarrhœa of an obstinate nature. In these cases the diagnosis can also be established by finding the tubercle-bacilli in the discharges.

CASE 443. FIG. 129.

II.



Large tubercular ulcer of cæcum.

The prognosis is very unfavorable, and death may occur either from the severity of the intestinal symptoms or, as pointed out by Osler, more rarely by perforation or hemorrhage.

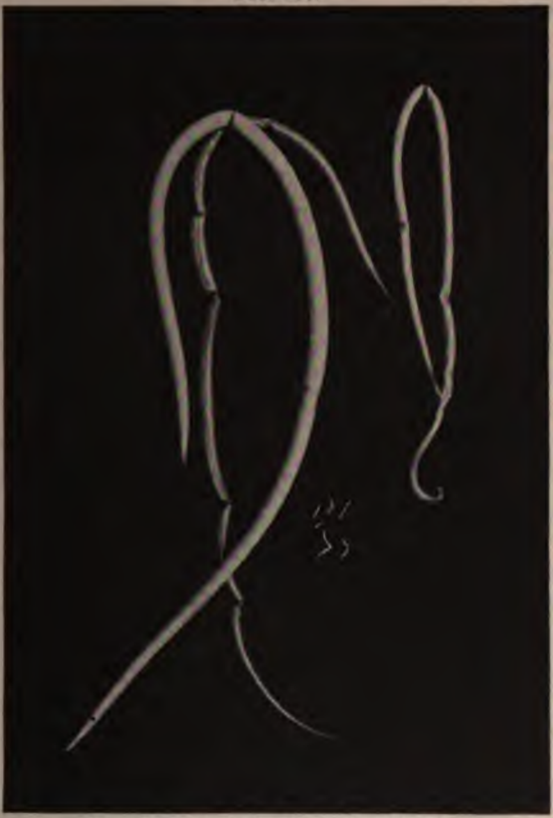
ANIMAL PARASITES.—The animal parasites which are found in the intestines of infants and children are the same that occur in older subjects. The only ones, however, which are common and important enough to speak of are the *oxyuris vermicularis* (pin-worm), the *ascaris lumbricoides* (roundworm), the *tænia solium*, and the *tænia mediocanellata*.

OXYURIS VERMICULARIS.—The oxyuris vermicularis is a minute worm which looks like a little piece of white thread. The female is from 0.6 to 1.2 cm. ($\frac{1}{4}$ to $\frac{1}{2}$ inch) in length. The male is about one-third as large, and has the tail rolled into a spiral.

I have here some of these worms to show you (Fig. 130). Their de-

›pment takes place in the large intestine, and the mature worms deposit r eggs in the rectum. They enter the intestine through the mouth, and dren are very apt to reinfect themselves by carrying the eggs on the ers or under the nails to their mouths.

FIG. 130.



Oxyuris vermicularis. Ascaris lumbricoides.

These worms sometimes exist in large numbers, and their development › rapid that it is often difficult to dislodge them completely. The most mon symptom of the oxyuris is an intense itching about the anus. The p of the child is disturbed by this irritation, and various nervous symp- s develop in children who are infested with this parasite. Thus incon- nce of urine sometimes results. In girls the parasite, by migrating a the anus to the vulva, may cause a vulvo-vaginitis.

DIAGNOSIS.—The diagnosis of the presence of these, as of other intes- l parasites, can be made only by finding the worm or its ova. Where are suspected, an enema of clear water should be given. If the para- are present, they will be dislodged, and careful inspection will disclose r presence. Wherever there are symptoms of reflex irritation in the hborhood of the anus or the genital organs, the oxyuris should be sus- ed and sought for. The parasites can often be found in the fæcal dis-however, pass through the rectum either with the fæces or alone, and may migrate into the stomach, œsophagus, or nose. Instantaneous death has resulted from the entrance of these worms into the air-passages. They may also at times enter the common and cystic bile-ducts, and they have even penetrated farther and caused abscess of the liver. There is no danger of their perforating a normal intestine, but where ulceration has been present perforation has occurred.

SYMPTOMS.—There are no especial symptoms produced by this worm, and we can diagnosticate its presence only by seeing it or by finding the eggs in the fæcal discharges. The worm may in some instances produce a feeling of discomfort or even colic in the region of the umbilicus. Neither of these symptoms, however, can be depended upon, and an anthelmintic is required to determine whether the parasite is present. As a rule, the presence of these parasites in the intestine, unless in very large numbers, is not especially important.

TREATMENT.—The most efficacious treatment of this form of parasite is with santonin, which should be given in the same doses and with the same caution as I have already described in speaking of the treatment of the oxyuris.

In addition to santonin, the freshly prepared fluid extract of spigelia and senna, in doses of half a teaspoonful for a child two years old, and a teaspoonful for older children, can be given two or three times a day, care being taken not to produce too much irritation. The oil of chenopodium, three or four drops on sugar for a child two or three years old, and eight or ten drops for older children, can also be given. A cathartic should be used in connection with these drugs, as well as with santonin.

TÆNIÆ (Tape-worms).—Two forms of tæniæ occur in children. One of these is the tænia solium, the pork tape-worm. It has a slight projection at the apex of its head, around which are a series of hooks, and below which are four sucking-disks. The other form is the tænia mediocanellata, the beef tape-worm. It has a blunter head than the tænia solium, and does not have the circle of hooks.

There is nothing especial to be said concerning these worms, and I refer to them merely because at times they occur in early life. They are never met with in nursing children when milk forms the exclusive diet. There are no especial symptoms produced by this worm, and the diagnosis is made entirely by finding the segments in the fæces. There is no especial danger to life from the presence of the tape-worm.

I have here two specimens (Fig. 131, I., II., page 912) of tæniá to show you.

The worm in the bottle was from a child from whom the entire worm was expelled, and you can see, by means of a magnifying-glass, the head. The absence of hooks shows it to be the variety called tænia mediocanellata. I show you the other worm in order to impress upon you the importance of obtaining the head. You see that the head is not present, and

There is no anthelmintic which I have found especially successful in expelling the tæniæ. One of the most harmless is the alkaloid pelletierine from pomegranate. One-half teaspoonful of the tannate of pelletierine can be given to a child from three to five years old. As dizziness and headache are sometimes complained of, it is well to have the child kept in bed and lying down until the effect of the anthelmintic has passed off. The oleoresin of male fern may also be used. The dose is 0.94 to 1.88 gramme (¼ to ½ drachm). The cathartic which is most useful in these cases is Epsom salt, 7.5 to 15 grammes (2 to 4 drachms).

It is hardly worth while to mention the other numerous anthelmintics which have been recommended, as they are usually inefficient.

and childhood do not differ from those which are met with in later life. A rapid increase and decrease in the size of the liver are not infrequently met with in disease, and careful measurements have shown that even a very slight disturbance of health may cause in young children a variation of from 2 to 4 cm. (¾ to 1½ inches) in the size of the liver.

ACUTE YELLOW ATROPHY OF THE LIVER.—It is uncommon for the liver to be decreased in size, but this occurs in the rare cases of acute yellow atrophy at times met with in children. The disease is insidious in its onset, and is characterized by general symptoms of malaise, with icterus and bile-stained urine. In the beginning of the disease the liver is enlarged, but in the later stages it is decidedly diminished. Cerebral symptoms and vomiting are quite prominent, and death invariably occurs.

In most diseases which are accompanied by hepatic disturbance it is much more common to find the liver enlarged than to find it diminished in size. This enlargement may occur from a number of causes, among which is *mechanical congestion*, arising in the course of cardiac disease. I shall presently show you, when speaking of diseases of the heart, a case (Case 503, page 1042) illustrating this form of enlargement.

The morbid conditions of the liver which are most commonly met with are *fatty infiltration*, *amyloid infiltration*, *tuberculosis*, and *interstitial hepatitis*. This latter form of hepatic disease may arise under various pathological conditions. Thus, it is a frequent lesion in syphilis, and may occur in a number of systemic diseases. It may also result from the use of alcohol, and at times it occurs apparently unassociated with disease of any other organ.

Other pathological conditions of the liver, such as hepatic abscess, parasites, such as hydatids, and new growths, such as carcinoma, adenoma, and, as occurred in a case at the Boston Infant Hospital, sarcoma, are too rare to be considered in a general clinical lecture on children.

FATTY INFILTRATION OF THE LIVER.—Fatty liver in early life does not differ pathologically from that which is met with at a later period. The liver may or may not be enlarged, and there are no especial hepatic symptoms which characterize this condition, the symptoms being those of the general disease from which the child is suffering. It may be found associated with a number of diseases, especially rhachitis and tuberculosis. When the liver is enlarged from this cause its surface is found to be smooth and palpation is painless.

The prognosis, unless the disease is dependent upon some incurable disease elsewhere, is fairly good.

The treatment is essentially dietetic and hygienic.

TUBERCULOSIS OF THE LIVER.—Tuberculosis of the liver occurs in connection with general tubercular disease of other organs, and does not in itself present any especially characteristic clinical manifestations. The disease is commonly found in the form of miliary tubercles and cheesy nodules. Except in rare instances where large caseous masses cause obstruction and later disintegration of the tissues, with hepatic enlargement and abscess, it is not usually recognized during life.

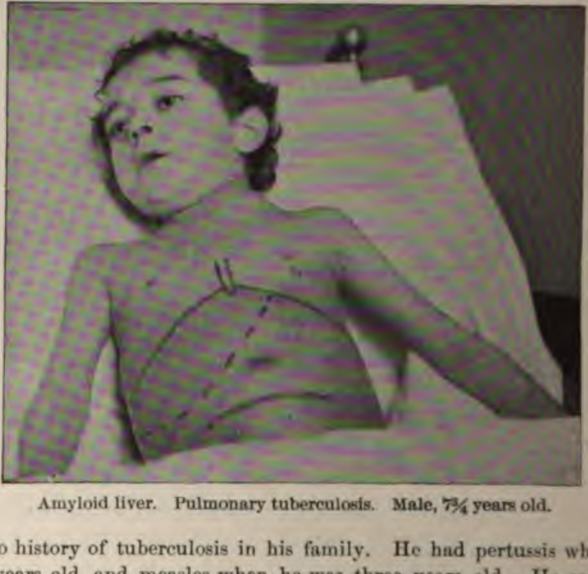
AMYLOID LIVER.—When amyloid changes are present in the liver, other organs, such as the spleen, kidneys, and intestine, are involved. Amyloid infiltration may occur in the course of tuberculosis, where there is chronic disease of the bones, with extensive suppuration, and in wasting diseases. A very prominent symptom in this condition is extreme anæmia. The liver is, as a rule, very much enlarged, and commonly more so than in any of the other hepatic disturbances. Its surface is smooth, and there is rarely hepatic tenderness or pain. Ascites is rare, and there is usually no icterus.

The diagnosis is not difficult if we find that the child has one of the diseases which I have just mentioned as being the causes of amyloid changes.

When these changes occur in the liver the prognosis is very grave, and there is no treatment which will be of more than temporary benefit. The treatment, therefore, is simply symptomatic.

This boy (Case 444) is seven and three-quarter years old.

CASE 444.



Amyloid liver. Pulmonary tuberculosis. Male, 7¾ years old.

There is no history of tuberculosis in his family. He had pertussis when he was one and a quarter years old, and measles when he was three years old. He seemed well and strong until seven months ago, when he became listless and began to have fever and to perspire profusely. Four weeks ago he began to vomit occasionally, to complain of headache, and to cough. You see that although he has evidently lost in weight he is not especially emaciated. His entire skin is extremely pale and has a waxy look, which is apparently not due to jaundice. His mucous membranes show much anæmia. His tongue is heavily coated, and his breath is offensive. He is dull and apathetic. The cervical glands are enlarged and slightly tender, but do not fluctuate. The glands are moderately enlarged in the axillæ and groins. The percussion of the right lung, especially at the apex, is dull, and there are numerous râles. The area of cardiac dulness is not enlarged, but there is a slight systolic murmur at the apex. The spleen is slightly enlarged. The edge of the liver can be felt below the line of the umbilicus. The area of hepatic dulness is increased, as is represented by this broken line. I have also indicated the lower part of the sternum and the lower borders of the ribs by black lines. There is no hepatic tenderness, and the child does not complain of pain. The lower part of the abdomen is dull on percussion as high as the line which I have drawn under the umbilicus. This is due to a slight amount of ascites. The legs are swollen. The urine has a specific gravity of 1010, and contains a slight trace of albumin, an occasional hyaline cast, and renal epithelium. The temperature has varied from 37.2° to 39.4° and 40.5° C. (99° to 103° and 105° F.). The increased size of the liver is probably due to amyloid infiltration.

(Subsequent history.) A few days later the child grew rapidly weaker, and died of exhaustion.

INTERSTITIAL HEPATITIS (Cirrhosis).—The syphilitic form of hepatitis as it occurs in infancy I have already described in my lecture on Hereditary Syphilis (page 489).

Interstitial hepatitis as it occurs in childhood may be *atrophic* or *hypertrophic*. The general symptomatology differs but little from that of the adult. In the beginning the symptoms are very apt to be confounded with those of simple congestion arising from digestive disturbances. There may be abdominal pain, slightly augmented by pressure. Diarrhœa and constipation alternate. There are usually ascites and slight jaundice, and at times dilatation of the subcutaneous abdominal veins. Stigmata composed of collections of dilated minute veins are sometimes observed on the face. The temperature is irregular. As a rule, it is not much heightened, and in fact is often subnormal.

The prognosis and treatment in early childhood are the same as in adults. A certain number of cases seem to have followed scarlet fever and measles.

Alcohol is sometimes an etiological factor in infancy and early childhood. Where the disease is caused by alcohol the pathological condition is, as a rule, atrophy. Enlargement is not common, and the symptoms are the same as in the adult, the ascites being especially prominent.

Where the hepatitis is apparently not dependent on disease elsewhere, and is not due to alcohol, there are no characteristic symptoms beyond the enlargement of the liver. In this form the ascites is usually small in amount, and the diagnosis can be made only by eliminating the other forms of enlargement.

I have here a case of hepatic enlargement which seems to represent clinically that form of hepatic disease which is commonly spoken of as hypertrophic cirrhosis.

This little girl (Case 445, page 918) is eighteen months old.

There is no history of syphilis or of tuberculosis. She had pertussis when she was ten months old, and the cough lasted for several months. She has never taken alcohol in any form. She was well until three months ago, when she began to complain of pain in the abdomen and to become pale. Two weeks before entering the hospital she had diarrhœa, and her abdomen was noticed to be swollen. On entering the hospital and being placed on a proper diet, the diarrhœa ceased, but the swelling of the abdomen increased. The child, as you see, is well developed, but pale. The abdomen is much enlarged. The edge of the liver can be felt nearly as low as the line of the umbilicus. I have marked out the

The *non-inflammatory* diseases are mostly represented by new growths. These may be of a malignant nature, such as carcinoma and sarcoma, or they may be lipomata or of a cystic character. In this connection it is well to say that tumors of the omentum are rare, but that cysts and hydatids may occur in this region.

The differential diagnosis of these various forms of peritoneal and omental growths can scarcely be made during life.

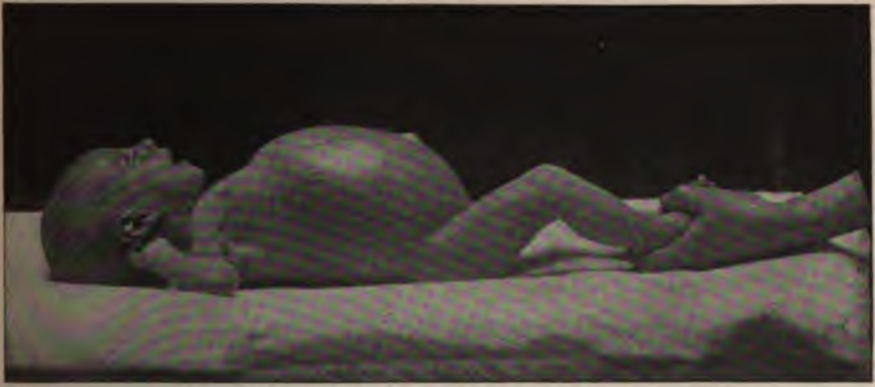
The treatment is essentially surgical.

The *inflammatory* diseases of the peritoneum are represented by peritonitis.

PERITONITIS.—Inflammation of the peritoneum may be acute or chronic, and is a condition of great importance in infancy and early life. Peritonitis may occur in the infant and child as it does in the adult. It is so rare in infancy and childhood as an idiopathic disease that the cases in which it has been studied post mortem have occurred almost exclusively during uterine life. Many of these, moreover, have presented a history of syphilitic infection. The septic form of peritonitis is not infrequently met with in the early weeks of life, and I have already referred to it when speaking of phlebitis umbilicalis (page 425).

I have here an infant (Case 446) who was brought to the hospital yesterday to be relieved of extreme distention of the abdomen.

CASE 446.



Probable intra-uterine peritonitis. Infant, 5 weeks old.

The infant weighed at birth 4500 grammes (10 pounds), seemed strong, and nursed for three weeks. There is no history of syphilis or of tuberculosis. After birth it began to be icteric. It passed meconium, but the fæcal movements since then have always been white. A few days after birth the abdomen began to swell, and it has since continued to increase in size. The skin is very tense, and the veins connected with the portal circulation stand out in marked relief all over the abdomen. The infant vomited once four days ago, and again this morning. It has become much emaciated. There is distinct fluctuation in every part of the abdomen, and dulness on percussion. An examination shows the heart and lungs to be normal.

(Subsequent history.) Laparotomy was performed by Dr. Lovett on the following day. On opening the abdomen a stream of pale fluid was thrown into the air with considerable force. A quart of this fluid was removed, and was examined by Dr. Whitney. It proved to be a jaundiced ascitic fluid, but the examination did not reveal its cause. It had a specific gravity of 1016, and contained 2¼ per cent. of albumin. It also showed bile pigment. The sediment contained much blood, many red corpuscles, an occasional white corpuscle, and fat-corpuscles. The flocculi were composed of finely granular material showing in places cells in a state of fatty degeneration.

After the fluid was evacuated a digital examination showed extensive adhesions in the intestine, especially on the under surface of the liver, where nothing but a matted mass could be found. On the supposition that there was some obstruction to the flow of bile into the intestine, an attempt was made with the finger to free the intestine from the lower surface of the liver.

The child rallied well from the operation, and thirty-six hours later a small spot of yellow bile appeared in one of the white movements. After this time bile was passed regularly and the movements became normal. The child's general condition and its nutrition improved.

At the end of ten days it was taken home, but it soon began to fail, and after three weeks died of malnutrition. No autopsy was allowed.

Acute Peritonitis.—Infants and children of any age may be attacked by acute peritonitis. It may occur in cases of tuberculosis, of the infectious diseases, of syphilis, and, most frequently of all, of appendicitis. The disease in any of the above forms is exceedingly rare between the ages of six weeks and two years. Where some definite cause, such as one of those just enumerated, cannot be found, the diagnosis is at times difficult from a want of prominence of some of the symptoms, such as the tympanites.

PATHOLOGY.—The pathological manifestations in acute peritonitis are reddening and loss of the normal glistening appearance of the peritoneum, soon followed by an exudation varying from a serous to a thick fibrinopurulent character. This exudation glues the coils of intestine together, forming adhesions, which, however, can be readily separated without the aid of a knife.

SYMPTOMS.—The symptoms of acute peritonitis vary according as the process is general or localized. The localized form of peritonitis corresponds in its symptoms to what I have already described in speaking of appendicitis, which is its most frequent cause. In general peritonitis the symptoms in infants, as I have already stated, are often obscure. In children the symptoms are usually pronounced and characteristic. The child is attacked with abdominal pain and with general abdominal tenderness. The abdomen becomes distended and tympanitic, and the child assumes the position which will most relax the abdominal walls,—that is, with the thighs flexed and the knees bent. Vomiting is very apt to be present, and is augmented when food is given. The bowels are often constipated, although at times there may be diarrhœa. The temperature is usually high, 38.3° to 40.5° C. (101° to 105° F.); in some cases, however, the temperature may be normal or subnormal. The pulse is small and rapid. The respirations are not only accelerated, but also superficial, as deep respiration causes pain. The face has an anxious expression, and shows great suffering. Where recovery takes place, these symptoms gradually subside after a few days, the tenderness, pain, and tympanites disappear, and the child's face assumes a tranquil look. When improvement does not take place, the pulse becomes weaker and quicker, the breathing more superficial and rapid, there is chilling of the extremities, and the child dies usually within a week.

PROGNOSIS.—The prognosis in these forms of acute general peritonitis is always serious. Constant vomiting makes it especially grave.

TREATMENT.—In treating cases of acute general peritonitis when seen in the early stages, a saline, such as sulphate of magnesium, can be given in doses of 1.87 c.c. to 3.75 c.c. (½ to 1 drachm), according to the age of the child. When, however, the disease is more advanced and there is great pain, opium will have to be resorted to. Where the peritonitis is of a high grade, where repeated doses of opium are demanded to relieve the pain, and where from the severity of the symptoms it is probable that a fluid beginning to be purulent is present, the case should at once be placed in the hands of a surgeon, as the question of laparotomy will then have to be decided.

I find in my notes the record of a case of general peritonitis:

An infant (Case 447), nineteen months old, previously apparently healthy, was attacked with vomiting and diarrhœa. On the following day the face was pale, the alæ nasi were working slightly, the respirations were 36, and the temperature was 39.4° C. (103° F.). The respirations gradually increased to 74, and the temperature rose to 40.3° C. (104 6° F.). The abdomen became very much distended and tender, and the face pinched and anxious. On the evening of the second day from the onset of the disease the temperature rose to 41.1° C. (106° F.), the infant became very restless, the pupils were contracted, and death took place a few hours later.

The autopsy was made by Dr. W. F. Whitney.

The heart and lungs were normal.

The spleen was enlarged, and was covered with a fibrinous exudation.

The kidneys were pale, and normal in size.

The liver was covered with flakes of recent lymph, and on section showed the acini to be red and their periphery yellowish and opaque. The mesenteric lymph-glands were slightly enlarged, and the smaller ones were translucent on section and presented evidence of hyperplasia. A small pocket of the larger glands was found to have become cheesy in the central portions, and in two of these the process had extended through the substance of the gland and had broken through its peritoneal covering. About these points of rupture there was a small zone of reactive inflammation.

PATHOLOGICAL DIAGNOSIS.—Acute general peritonitis, which, from an absence of any other source, must be considered to have been caused by the rupture of the cheesy, degenerated mesenteric glands.

In this case the high temperature and the distended abdomen rendered the diagnosis comparatively clear. The case is important on account of the cause, for there is seldom any noticeable enlargement of the mesenteric glands under the age of three years, and these glands seldom soften, but either retrograde or harden from calcification.

Chronic Peritonitis.—When acute peritonitis is localized in one portion of the intestine it may become chronic and form fibrous adhesions, but in the majority of cases chronic peritonitis, especially when general, is of *tubercular* origin.

Tubercular Peritonitis.—The original source of the tubercular process is often obscure. It may be primary in the peritoneum, but is more likely to be secondary to tubercular mesenteric glands.

PATHOLOGY.—The process consists in the formation of miliary tubercles on the peritoneal surface, which give rise to opaque cheesy thickening, often nodular, with firm adhesions of the adjacent surfaces. An exudation into the peritoneal cavity is usually present, the quantity generally being considerable and the quality fibrino-purulent.

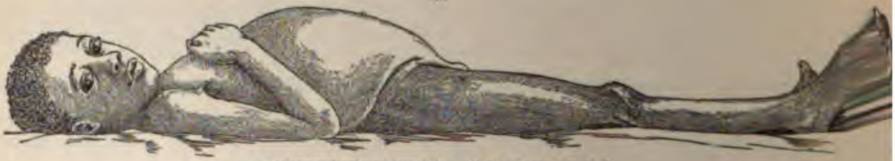
SYMPTOMS.—The initial symptoms of tubercular peritonitis are usually ill defined. There is a gradual loss of appetite and flesh, with occasional abdominal pain, which, as a rule, is not of a severe character. Attacks of diarrhœa are common, and are apt to be paroxysmal. The temperature is at times raised, especially in the latter part of the day. After these general symptoms have lasted for a number of weeks, the abdomen is noticed to be distended. A physical examination may show that there is nothing abnormal in the thorax, and that the morbid condition is confined entirely to the abdomen. At first the abdomen is resonant on percussion, but later may be dull, owing to masses of tubercle or to the presence of fluid. There is seldom any tenderness noticed on examining the abdomen.

DIAGNOSIS.—In a typical case, where the symptoms which I have just mentioned are present, the diagnosis is not difficult. Occasionally, however, there are no definite signs by which a diagnosis can be made, the only tangible sign being a seeming abdominal tumor, the resemblance of which to other abdominal tumors is so close that the diagnosis can be made only by laparotomy. You must nevertheless remember that most doubtful cases of abdominal tumors in children are tubercular.

PROGNOSIS.—When untreated, the prognosis of tubercular peritonitis is very variable. In some cases the disease after a number of months retrogrades, and the patient recovers. In most instances, however, the child becomes more and more wasted, the fever becomes more pronounced, the diarrhœa continues, the emaciation becomes extreme, and the child dies, usually of exhaustion. The surgical treatment of the disease has made the prognosis much more favorable.

CASE 448.

I.



Tubercular peritonitis. Male, 9 years old.

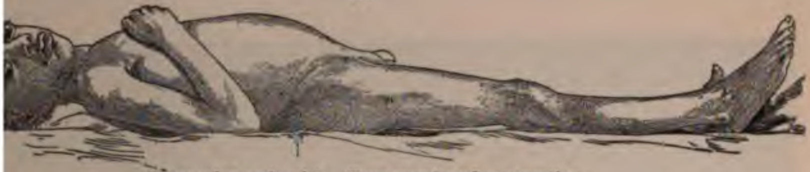
TREATMENT.—The treatment of tubercular peritonitis is essentia surgical, especially where there is ascites of any amount. In some cas opening the abdomen and evacuating the fluid will not only give relief will produce a permanent cure. In my experience at the Boston Childre Hospital, this procedure is often followed by complete arrest of the dise

I have some cases of tubercular peritonitis here in the wards to show you.

s colored boy (Case 448, I., page 922) is nine years old.
father died of phthisis. He has never been strong, but has had no acute illnesses.
eeks ago he began to have diarrhœa, and soon after enlargement of the abdomen.
as no pain, vomiting, nor cough. He has lost greatly in weight. You see that he
iated. He has a temperature of 38.3° C. (101° F.). His abdomen is much dis-
and there is a distinct wave of fluctuation. Physical examination shows nothing
ormal.

Case 448.

II.

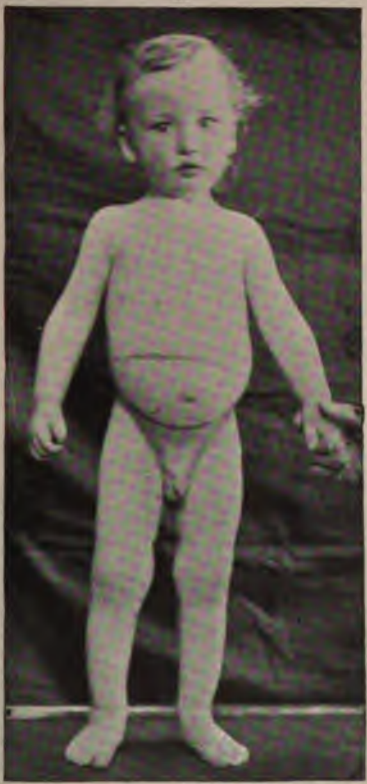


Tubercular peritonitis. Four months after operation.

bsequent history.) Laparotomy was performed by Dr. Bradford, and the fluid
ed. Tubercle-bacilli were found in the peritoneal tissue. When seen six months
wound had healed perfectly, and he was strong and well. This picture (II.) was
ur months after the operation.

Case 449.

I.



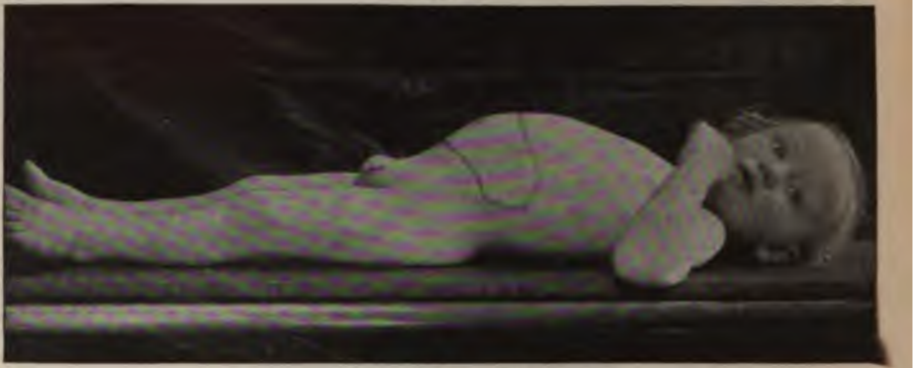
Tubercular peritonitis. Male, 2 years old.

s next case (Case 449, I.), a boy, two years old, is especially interesting in regard to
s.

He has not had general symptoms of serious import, but has lost slightly in weight, appetite, and strength. From time to time during the last six months he has complained of abdominal pain and tenderness. An examination of the abdomen shows a hardened, slightly irregular mass extending directly across the abdomen from one side to the other, 5 cm. (2 inches) above and the same distance below the umbilicus. It is not especially tender on pressure. Nothing else abnormal is detected about the child. As you see (II.), the line of percussion does not change when he is lying on his back, and there is no evidence of ascites.

Case 449.

II.

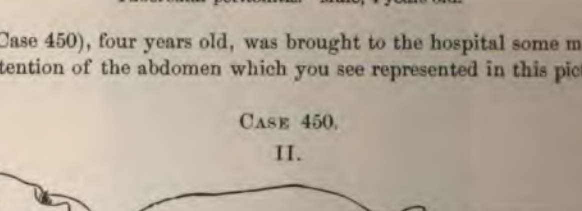


Tubercular peritonitis.

(Subsequent history.) Laparotomy was performed by Dr. Lovett, and a mass of cheesy nodules matting together the intestine was found. An examination of a portion of the mass showed the presence of the bacillus tuberculosis. No fluid was present. The child recovered, but sufficient time has not elapsed since the operation to allow us to decide whether the disease will return.

Case 450.

I.

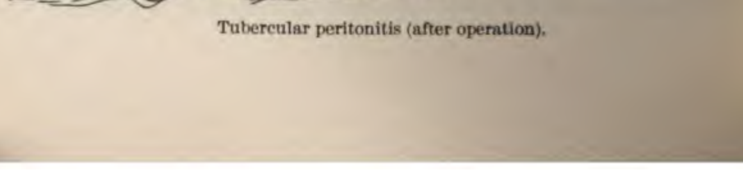


Tubercular peritonitis. Male, 4 years old.

This boy (Case 450), four years old, was brought to the hospital some months ago with the extreme distention of the abdomen which you see represented in this picture (I.).

Case 450.

II.



Tubercular peritonitis (after operation).

A physical examination showed nothing abnormal except in the abdomen, which was ll on percussion and showed fluctuation in every part. The child had gradually lost in ight, appetite, and strength.

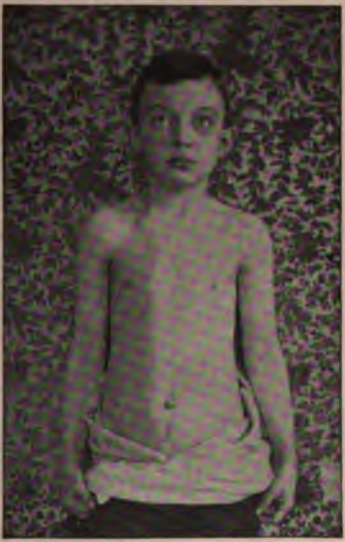
Laparotomy was performed by Dr. Lovett, and a large amount of ascitic fluid evac- ed. Tubercle-bacilli were present in the diseased peritoneum. The wound healed, but he course of a few weeks the fluid reaccumulated, and laparotomy was again performed Dr. Lovett. You see his condition now (Case 450, II., page 924), some weeks after the ond operation. No fluid can be detected.

(Subsequent history.) There was no recurrence of the ascites, and the child recovered npletely.

This boy (Case 451) is eleven years old

CASE 451.

I.

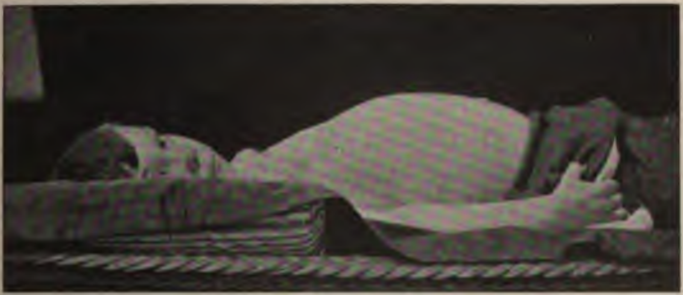


ubercular peritonitis. Male, 11 years old. Four years after operation, showing scar under umbilicus.

He is, as you see, well and strong, and shows no symptoms of tubercular disease. You ll notice the scar under the umbilicus, which marks the line of incision made when the arotomy was performed.

CASE 451.

II.



Tubercular peritonitis.

He entered the hospital four years ago, and here is a picture (II.) of him which was ken at that time.

DIVISION XV.

DISEASES OF THE KIDNEYS, BLADDER, AND GENITAL ORGANS.

LECTURE XLVII.

KIDNEYS.—Diseases of the kidneys may be congenital or acquired.

CONGENITAL DISEASES.—The congenital abnormalities, such as congenital cystic kidney, absence of one kidney, hypertrophy of the remaining kidney where one is absent, anomalous shapes of the kidney, and malpositions of the ureters, are important, but are so closely connected with purely surgical questions that they need merely be referred to in a medical lecture. The lobulated kidney, which I have already described (page 44, Fig. 9) as a normal condition in intra-uterine life, may to a greater or less degree continue into infancy and childhood, but has no pathological significance. Movable kidneys are rare in early life, but have been reported.

ACQUIRED DISEASES.—Renal disease as a primary affection in infancy and childhood has been considered rare, but this view has been modified by later bacteriological investigations, which have shown that nephritis is not uncommon in cases of general infection. Secondary renal lesions are comparatively common.

Renal diseases, with the exception of the nephritis following scarlet fever, have not been satisfactorily studied in children. A series of systematic examinations of the urine, in connection with later post-mortem examinations of the kidneys in the same cases, sufficiently extended to give us data for a precise diagnosis in an especial case, has not yet been made. Owing to the variation in the symptoms, the diagnosis of renal disease in the child must for the present depend upon the systematic and routine examination of the urine.

The diseases of the kidney in infancy and childhood are not so varied as in adults. They are chiefly represented by active hyperæmia (acute parenchymatous degeneration) and the nephritis following scarlet fever, which I have already fully described in my lecture on scarlet fever.

PHYSIOLOGICAL ALBUMINURIA.—Before speaking of the diseases of the kidney I shall describe a condition which is usually called physiological albuminuria.

This condition is not infrequent, and may occur at any period of infancy and childhood, but is most common between the fifth year and puberty. The amount of albumin present is, as a rule, less than one-twelfth per cent. It is not present in every micturition, and in many cases seems to depend upon over-exercise or a highly nitrogenous diet. The albumin is rarely present in the urine which is passed in the morning immediately after rising, and this is an important point in differentiating physiological albuminuria from periodic albuminuria due to pathological causes, such as uric acid. The children who have this physiological albuminuria often seem to be in good health, but sometimes they are rather delicate. The diagnosis can be made only by repeated examinations of the urine passed at different times in the day, and by observing the effect of exercise and diet upon it. The presence of blood-corpuscles or abnormal elements in any amount from the kidney shows that there is a pathological condition. An occasional hyaline cast and albumin as high as one-fourth per cent. for short intervals may be present. The albumin often disappears for a time and returns again. Children between the ages of three and seven years excrete nearly double the quantity of urine and of urea for each kilogramme of their weight that adults do. The amount of urea excreted in children between the ages of three and seven years is 0.973 gramme for each kilogramme of their weight. This fact is to be borne in mind in estimating the quantity of urea passed in cases of nephritis, because otherwise the kidneys might appear to be excreting a normal amount of urea and yet the amount be abnormally small for the age.

The prognosis in these cases of physiological albuminuria is good, and, so far as I know, no cases have been reported in which the condition terminated in nephritis.

The treatment of this condition is to regulate the diet, exercise, and general hygiene carefully. If the children are anæmic, iron is indicated.

GENERAL PATHOLOGY AND ETIOLOGY.—According to Councilman, to whom I am indebted for much information on this subject, the acquired diseases of the kidney in childhood show considerable differences from the renal diseases of the adult. In childhood there is a greater liability to those acute affections, such as scarlet fever, measles, and diphtheria, in the course of which nephritis is apt to appear. Children under the age of fifteen years are less subject to many pathological conditions, such as disorders of the circulation, which in the adult frequently lead to chronic lesions of the kidney. Children do not usually have those disorders of the circulation which result in granular kidney, for lesions of the arteries, especially the condition known as arterio-sclerosis, do not commonly occur in childhood. While it is true that typical examples of the small granular kidney are sometimes met with in children, these lesions of the kidney are primary, and the lesions of the circulatory system are secondary and dependent on the renal lesions. A part of the chronic diseases of the kidney in the adult is without doubt to be referred to the continuous action on the kidney of slight pathological conditions, an action from which the child's age protects it. One pathological lesion not perfectly recovered from, moreover, makes the kidney more prone to disease, and a greater effect will be produced a second time by the same cause, and chronic disease will result. In the kidney of the adult, with the advance of years there is a gradual decline in the power of regeneration, and slight troubles are not readily recovered from. The kidney of the child, on the other hand, is an organ which possesses great power of growth and regeneration. For this reason a condition which in the adult organ is either not recovered from at all, or lays the foundation for chronic disease, will in childhood result in complete recovery. Again, the child is not exposed to certain conditions which are productive of chronic lesions, or which may lay the foundation for them. Among these may be mentioned alcoholism and excesses of various sorts. Many cases of nephritis in the adult are to be referred to causes acting not through the blood, but through the urinary tract. The child, on the other hand, is not exposed to the dangers arising from hydronephrosis and pyelonephritis, except to a very limited degree. Although the causes of disease are less numerous and less common in children than in adults, yet when the same etiological factor is present the same morbid condition is produced in the kidney. The various cachectic conditions will lead to amyloid infiltration in the child as they do in the adult, and amyloid infiltration of the kidney makes up by far the larger part of the chronic cases of albuminuria in children. We may also meet with certain chronic lesions in the child's kidney, such as are seen in tuberculosis, and these may lead to albuminuria and nephritis.

The acute diseases of the kidney, as a rule, either tend to recovery or are in themselves fatal; so that only a small number of chronic diseases are met with which result from the acute diseases. These are not to be referred to the continuous action of the poison of the acute disease, but to the effect on the kidney of the lesions produced by the acute process. An example of this is the condition of chronic nephritis after scarlet fever, where the acute lesions gradually pass into the chronic. These chronic lesions are to be attributed to the disorders in the circulation of the organ brought about by the destruction of the glomeruli.

GENERAL SYMPTOMATOLOGY.—The general symptoms connected with the various forms of nephritis are so similar that it will be less confusing to mention first the common symptoms which may occur in any of the forms of nephritis, and then to describe the etiology, pathology, and urinary examination of the different forms.

One of the most common symptoms in nephritis is œdema, which occurs frequently in acute nephritis and in chronic parenchymatous nephritis. The œdema generally appears first in the eyelids, and then in the hands and feet. There may be general anasarca. Not infrequently, however, œdema is absent or not marked. Vomiting is not infrequent in the beginning of the disease, and in some cases is, perhaps, due to the heightened temperature. It may occur later in the disease as a symptom of uræmic poisoning. In such cases there is marked diminution in the amount of the urine, or even suppression. A peculiar dull white color of the skin is not uncommonly seen in chronic parenchymatous nephritis, and is quite striking. In acute nephritis fever is often present to a greater or less extent, but is a variable symptom. Lack of appetite, and weakness, are common in both acute and chronic nephritis. Headache is a variable symptom. It is a common symptom of uræmia, and sometimes the only one. Amaurosis may occur as the result of albuminuric retinitis, or it may be a functional symptom of the uræmic poisoning and disappear later if the patient recovers. Hypertrophy of the left ventricle is apt to occur in interstitial and chronic parenchymatous nephritis. Both diseases are, however, very uncommon in childhood. In acute nephritis following scarlet fever dilatation and moderate hypertrophy of the left ventricle are not uncommon. Transudation into the serous cavities has been reported in a number of cases, as has also œdema of the larynx.

Before I mention the details of the urinary analyses in the various diseases, you should understand that in all cases of nephritis the amount of urea should be carefully estimated from time to time, as a decrease in the urea always shows a pathological condition, and a return to the normal amount is usually indicative of recovery unless there is a complication with some other disease. Any interference with metabolism, whether in the liver or in the lung, may diminish the amount of urea in the urine. In children during convalescence from acute nephritis the urea returns to or exceeds the normal amount, while in chronic nephritis it is always diminished, as it is in adults. A sudden and excessive diminution of the urea in acute nephritis is suggestive of uræmia. In acute and chronic nephritis the chlorides are diminished when an effusion such as ascites is increasing, and gradually return to the normal amount as the effusion is absorbed.

Active Hyperæmia (Catarrhal Nephritis. Acute Parenchymatous Degeneration).—ETIOLOGY.—An active hyperæmia of the kidney may arise in the course of various acute infectious diseases. It may also be caused by an excess of uric acid, and by such irritating drugs as turpentine, cantharides, and arsenic. When the action of these causes is very intense, an acute nephritis may result.

PATHOLOGY.—The pathological conditions resulting from active hyperæmia of the kidney are a gradual degeneration and desquamation of the renal epithelium, and an injection of the blood-vessels. There is also to some extent an infiltration of round cells. The process seems to affect chiefly the epithelium of the tubules.

SYMPTOMS.—Unless the hyperæmia is very pronounced, there are, as a rule, no general symptoms, though œdema and other symptoms may rarely be present, as in acute parenchymatous nephritis.

DIAGNOSIS.—The diagnosis is made by the examination of the urine. The urine is clear, and its color is often normal. The amount is diminished. The specific gravity is higher than normal. There is a slight sediment, with a trace of albumin, perhaps one-eighth per cent., or at times a little more. Microscopic examination shows the presence of renal epithelium and blood-corpuscles; the latter, however, not in sufficient number to color the urine. There are also leucocytes, and hyaline and fine granular casts, with an occasional epithelial cast and blood cast; the last three varieties, however, are not very numerous.

Prognosis.—The prognosis in active hyperæmia of the kidney is good, and the pathological condition usually disappears when its cause has been removed.

Treatment.—The child should be placed on a diet exclusively of milk, so as to avoid any further irritation of the kidneys, and should be made to drink a great deal of water. It should be kept quiet, and its general hygiene should be carefully regulated.

I have here a case (Case 452) which is probably one of active hyperæmia. This boy, aged five years, had varicella when he was six months old, and measles when he was one year old. He had no other diseases until three weeks ago, when, without any apparent cause, he is said to have had a convulsion and to have vomited. He has never had any œdema, and a general physical examination shows nothing abnormal. An examination of the urine shows it to be high-colored and cloudy, to have a specific gravity of 1016, a large trace of albumin, and a yellowish-brown sediment consisting of amorphous urates. A microscopic examination shows the presence of uric acid crystals, hyaline, granular, and epithelial casts, and a few leucocytes. The total amount of urine passed in the twenty-four hours is from 360 to 450 c.c. (from 12 to 15 ounces). Heating the urine causes the high color, cloudiness, and brown sediment to disappear.

(Subsequent history.) Three weeks later there was only a slight trace of albumin in the urine, which was of a normal color, had a specific gravity of 1020, and contained a few hyaline and granular casts. A few weeks afterwards the urine was found to be normal. No other abnormal symptoms occurred during the whole course of the disease.

Passive Hyperæmia.—In addition to the active hyperæmia which I have just described, a *chronic passive hyperæmia* may occur, dependent upon diminished arterial or increased venous pressure. This condition occurs in chronic cardiac disease with disturbance of compensation, in chronic pulmonary disease, and where there is mechanical hinderance to the venous circulation, as from the presence of abdominal tumors.

Symptoms.—The symptoms which occur in the course of passive hyperæmia are not referable to the kidney, but depend upon the disease which causes the hyperæmia. The urine in this condition is high-colored and often considerably diminished in amount. It has a high specific gravity, and often a heavy sediment of amorphous urates. There is a slight trace of albumin, usually under one-eighth per cent. Microscopic examination shows a few hyaline casts with renal cells adherent, and an occasional blood-corpuscle. There are, however, very few of these elements in the sediment. The peculiarity of the urine in passive hyperæmia is that it varies. If the heart becomes stronger, the urine is passed in larger quantities, is not so highly colored, and contains a smaller amount of albumin.

Prognosis.—The prognosis in cases of passive hyperæmia of the kidney depends upon the cause of the condition.

TREATMENT.—The treatment is to be directed to the cause or causes of the congestion.

Acute Nephritis.—ETIOLOGY.—The most common cause of acute nephritis is scarlet fever. Other diseases in the course of which it may arise are diphtheria, measles, varicella, erysipelas, typhoid fever, malaria, pertussis, and pneumonia. With the exception of its occurrence in scarlet fever, diphtheria, and measles, the disease is not frequent. Cases have been reported where it has arisen in the course of extensive affections of the skin, such as eczema. It also occurs after the application of drugs to the skin, and from the internal administration of such irritating drugs as cantharides, turpentine, salicylic acid, and arsenic. Cases of primary nephritis have been reported where no cause could be found. Although it is difficult to estimate with certainty the importance of cold as a causative factor in the etiology of acute nephritis, and although it has been denied that cold can produce this condition, yet numerous cases have followed exposure to wet and cold. Many of these primary cases, however, were probably due to micro-organisms, as the disease has been not infrequently observed in connection with general septicæmia.

PATHOLOGY.—I have already fully described the pathology of the acute nephritis which follows scarlet fever. In the nephritis arising from the other causes which I have just mentioned, the pathological changes differ chiefly in the degree in which the different portions of the kidney are affected. The process appears to be a mixed one, but some portions of the kidney are more involved than others.

SYMPTOMS.—The symptoms of acute nephritis are such as I have already described in my lecture on scarlet fever. In general, the symptoms arising in cases due to other causes than scarlet fever are the same, but less severe than those which I have described in connection with that disease. The amount of albumin and the quantity of the urine depend chiefly upon the degree to which the glomeruli are affected. The number of casts and epithelial cells depends chiefly upon the degree of the involvement of the tubules. The interstitial changes can scarcely be determined by the urine. It is well to bear in mind that the urine may vary from day to day in any affection of the kidneys. In one type of the ordinary diffuse nephritis the urine presents the following changes. The color varies from red to brownish-red, according to the quantity and freshness of the blood which it contains. The specific gravity is high. The amount is markedly diminished, and there may even be anuria. There is a heavy dark-red sediment, with a large amount of albumin, usually more than one-quarter and often one-half per cent. Microscopic examination shows a large quantity of blood, numerous renal cells, leucocytes, a large number of casts, epithelial, blood, and fibrinous, also both fine and coarse granular casts and a fine detritus. All these elements are stained yellow or brown by the blood pigment. As the process advances towards recovery there are usually found, in a few days, more abnormal blood-corpuscles showing themselves in the form of pale rings. There are more granular casts and detritus, and fewer epithelial and blood casts. Fatty elements, such as fatty renal cells and free fat, begin to appear. There are also more hyaline casts, usually with a few cells and blood adherent to them. Still later, there is a preponderance of hyaline casts, with fewer epithelial cells and blood-globules. During this time the amount of urine increases, until during the convalescence it finally rises above the normal amount. The color changes to smoky, and finally becomes pale. The specific gravity diminishes. The albumin diminishes to a trace, but this trace may persist for a long time. The elements in the sediment become fewer. Acute exacerbations are not uncommon.

PROGNOSIS.—In general the prognosis is good. The disease rarely becomes chronic. Death may occur in the beginning from the severity of the disease, or later from uræmic poisoning. Some cases end fatally from some intercurrent disease, such as pneumonia, or from a nephritis occurring in the course of a general septicæmia. The majority of the cases, however, recover after from four to eight weeks, although a trace of albumin and a few hyaline casts may persist for several months, the child in other respects being quite well.

TREATMENT.—The treatment of acute nephritis is the same as that which I have already described in the nephritis following scarlet fever.

Chronic Parenchymatous Nephritis.—ETIOLOGY.—Chronic parenchymatous nephritis is not a common disease in childhood, and its etiology is still very obscure. Some cases have followed an attack of acute nephritis, and in these there has generally been an interval during which the urine has simply contained a trace of albumin and a few casts, the symptoms of a chronic affection of the kidney appearing later. Cases have also occurred in connection with long-continued suppurative processes in the bones, joints, or elsewhere, arising in the course of tuberculosis or syphilis. In these cases amyloid infiltration is also apt to occur. There are also instances where no cause whatever can be discovered.

PATHOLOGY.—The pathological condition is the same as in the adult.

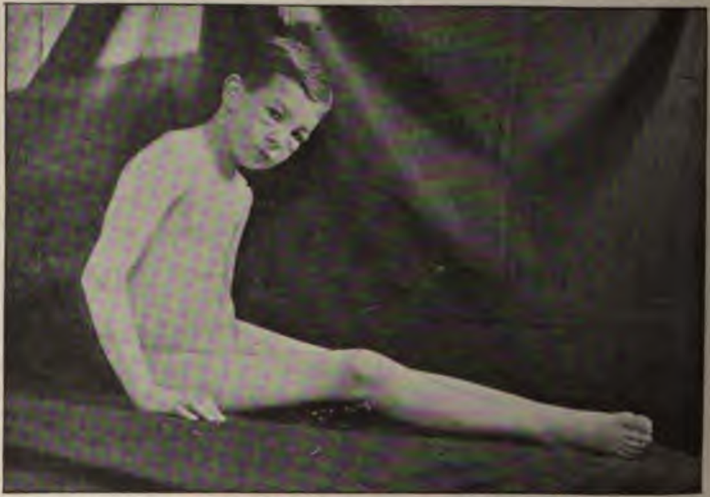
SYMPTOMS.—The symptoms of chronic parenchymatous nephritis are insidious in the beginning and are prolonged. There are marked pallor, a tendency to œdema, and a transudation into the serous cavities. Cardiac hypertrophy, weakness, loss of appetite, headache, and at times vomiting and diarrhœa, are among the common symptoms. Retinal changes sometimes occur, and there is a tendency to intercurrent diseases, such as pneumonia and pleurisy. Uræmic intoxication may be expected later. The urine may be high or pale in color. It is diminished in amount, but not markedly so, as in acute nephritis. The sediment is usually heavy. The specific gravity is diminished. There is a large amount of albumin, often one-half per cent. or more. There are frequently amorphous urates in the sediment, which must be removed by heat before the microscopic examination is made. Microscopic examination shows a characteristic preponderance of fatty elements. There are fatty renal cells, free fat, fat in the casts, and cells completely fatty. There are also compound granular cells, and granular, epithelial, and hyaline casts. There are often acute complications in the kidney, in which case the amount of urine becomes markedly diminished, and the sediment shows blood, blood casts, and epithelial casts in addition to the large number of fatty elements. When the disease is complicated with amyloid infiltration, the diagnosis of the latter can hardly be made from the urine.

PROGNOSIS.—The prognosis is not good. Some cases having the clinical symptoms of the disease have apparently recovered. Most cases, however, die from uræmic intoxication or from some intercurrent disease, such as pneumonia. There may be a remission in the symptoms for a time.

TREATMENT.—The treatment is to restrict the diet as far as possible to milk. Good hygienic surroundings, and as much rest as possible, are indicated. Diuretics may be used when the amount of urine is diminished. I have already described in my lecture on scarlet fever the best treatment with diuretics (page 563).

Here is a boy (Case 453), eleven years old, with nephritis which has lasted a year. The examination of the urine by Professor Wood shows the probability of a chronic parenchymatous nephritis with an acute exacerbation.

CASE 453.



Probable chronic parenchymatous nephritis with an acute exacerbation. Male, 11 years old. Relapse after being out of bed five days.

This child had pertussis when he was three years old, scarlet fever when he was four years old, and measles and pneumonia when he was five years old. He is reported to have remained well from that time until nine months ago, when, without any known cause, such as exposure to cold or sickness of any kind, his face and eyes began to be œdematous. This was followed by œdema of the legs and ankles, and was accompanied by dyspnœa. The urine was noticed to be nearly of the color of blood, and to be lessened in amount. He was kept in bed for six weeks, and is said not to have complained of any especial discomfort. During this attack his appetite remained fair. Since the beginning of the attack he has grown somewhat weak and become pale. Six weeks ago the paleness and œdema about the eyes increased, and the urine became smoky again. This was followed by œdema of the ankles, feet, and legs, accompanied by dyspnœa. The bowels have been regular, and there has been no vomiting. He sleeps well. On entering the hospital his face looked pale and waxy. There was considerable œdema of the face, especially of the eyes. His tongue was slightly coated, and there was œdema of the ankles, feet, and legs. Nothing abnormal was found in the heart or lungs, and there was no evidence of ascites.

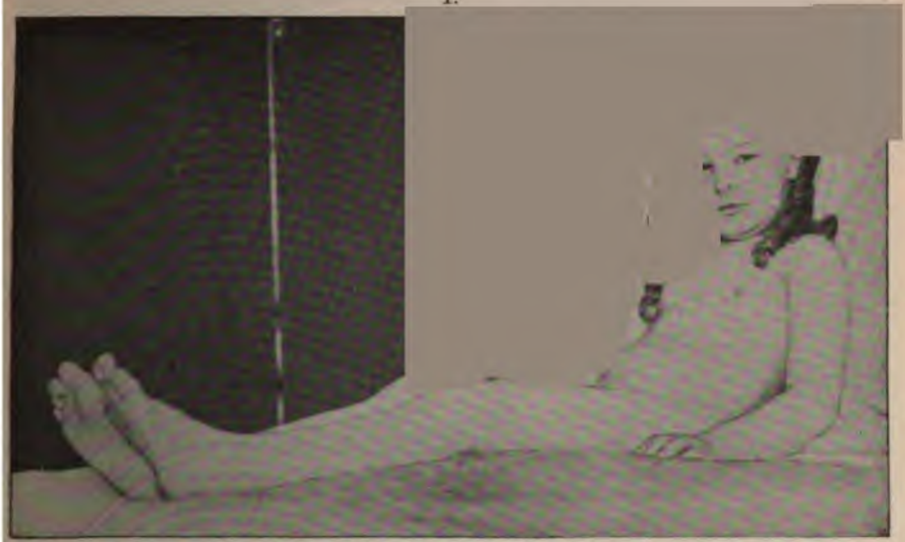
He was kept in bed and given a diet of milk. Under this treatment the œdema and anæmia disappeared rapidly, and in two weeks he was allowed to be dressed and about the ward. This was five days ago. Yesterday he again had œdema of the face, and was immediately put to bed. As you see him to-day, the œdema under the eyes is especially prominent. From 750 to 900 c.c. (25 to 30 ounces) of urine are passed in the twenty-four hours. An examination shows it to have a specific gravity of 1010, an acid reaction, to contain urea 4.75 grains to the ounce, to have the chlorides diminished, and to contain $\frac{6}{10}$ per cent. of albumin, but no sugar. The sediment shows numerous hyaline casts of medium diameter, some of large diameter from the straight tubules, many coarse and fine granular casts, numerous fibrinous casts, and many casts with renal cells adherent; also epithelial casts and blood casts; an excess of renal epithelium, most of it granular or fatty; compound granular cells, a large amount of abnormal blood, free fat, and fatty casts. His temperature has varied from 36.6° to 37.2° C. (98° to 99° F.).

(Subsequent history.) After remaining in the hospital for two months, with temporary periods of improvement, he was discharged in about the same condition as when he entered.

Here is a girl (Case 454), nine years old, with nephritis.

CASE 454.

I.



Probable chronic parenchymatous nephritis with an acute exacerbation, Female, 9 years old. Second week of the disease.

This child had measles when she was two years old, scarlet fever when she was three years old, varicella when she was six years old, and pertussis when she was eight years old. She apparently recovered entirely from all these diseases, and was well until one week ago, when, without any apparent cause, her face and feet began to swell. She complained of no pain, and had no other symptoms. As you see her in bed, you will notice the marked and extensive œdema of the entire face, body, and limbs. You see that the œdema is pronounced under both eyes, but especially so under the right one. There is great pallor of the skin, and the feet and hands are much swollen. Nothing abnormal has been detected in the heart or lungs. There is no ascites. She has no headache, and does not complain of any discomfort.

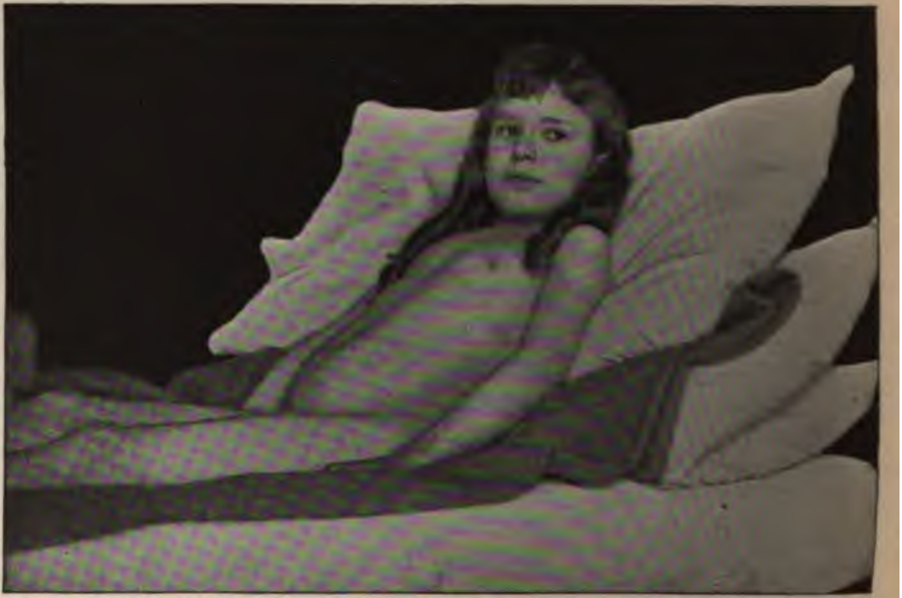
An examination of the urine shows the color to be pale, the reaction acid, the specific gravity 1012, the sediment moderate; it contains albumin ¼+ per cent., and no sugar; the sediment contains considerable abnormal blood, some free fat, and a number of hyaline and fine granular casts of medium and small diameter, many of them short and with oil-globules adherent. There are some fatty renal epithelium, leucocytes, casts with renal epithelium, and hyaline casts with a few renal cells adherent. There are also several fatty casts. The casts are not very numerous.

She is being treated by absolute rest in bed, bitartrate of potassium, digitalis, and a diet of milk, as I have recommended in my treatment of the nephritis following scarlet fever (page 545).

I show this child because she illustrates the appearance of a case of marked nephritis, with its excessive universal œdema and peculiar pallor of the skin. The diagnosis of the exact lesion of the kidney in this case is, however, very uncertain, as the pathological processes in the kidney are not confined to any one part of the organ, and the urinary analysis is often for this reason unsatisfactory.

Case 454.

II.



Probable chronic parenchymatous nephritis with an acute exacerbation. Female, 9 years old. (Ten days after treatment was begun.)

I have provisionally called it a case of probable chronic parenchymatous nephritis with an acute exacerbation. The presence of blood may be due to an acute exacerbation, but might also mark it as an acute nephritis involving chiefly the parenchyma of the organ, as shown by the predominance of cells. It is significant in this case as pointing towards a chronic process that the urine has always been pale, showing that blood in sufficient quantity to color the urine has not been present.

(Subsequent history.) In about a week the œdema rapidly diminished and the urine increased in amount. An analysis of the urine at this time showed that the color was pale, that it had a specific gravity of 1010, a trace of albumin, and a slight sediment, consisting of a small amount of blood, renal epithelium, and a few casts with blood. The total amount of urine passed in the twenty-four hours was 2010 c.c. (67 ounces).

This picture taken at this time (II., page 936) shows how the general œdema has passed away, and how the skin has lost the extreme pallor which it presented on the child's entrance into the hospital.

An examination of the urine three weeks later showed the color to be pale, the reaction acid, the specific gravity 1014, the albumin ¼+ per cent. It contained hyaline and fine granular casts of small diameter, many with oil-globules and renal cells adherent; also free oil-globules, fatty and granular renal epithelium, some normal and abnormal blood, leucocytes, and squamous cells. The casts were not very numerous, and there was not much change from what was found in the urine three weeks previously. At this time the urine again became scanty, and the œdema and pallor returned, but she did not complain of any discomfort. An examination of the urine eight weeks later showed it to be pale and cloudy, the reaction acid, the specific gravity 1018, and that it contained considerable sediment, and albumin ¼+ per cent. The sediment consisted chiefly of hyaline casts of medium and small diameter, many of them having renal cells and fat adherent. There were also a few finely granular casts, considerable abnormal blood, free fat, fatty renal cells, epithelium, leucocytes, and occasionally blood, epithelial, and fatty casts.

The diagnosis cannot be positively established until the case shall have been under observation for a much longer period.

Chronic Interstitial Nephritis.—Chronic interstitial nephritis is so exceedingly rare in childhood that very little need be said concerning it. A few congenital cases have been reported.

Etiology.—The etiology is obscure. In some cases it seems to have followed a chronic parenchymatous nephritis. In others no cause could be found.

Pathology.—The pathology is the same as in adults.

Symptoms and Diagnosis.—The diagnosis can scarcely be made from the symptoms. The disease is progressive and slow, with no characteristic symptoms. Cases have been reported in which there were general symptoms of headache, weakness, dyspnœa, palpitation, and disturbance of vision. Hypertrophy of the left ventricle occurs as a constant lesion. There is little tendency to anasarca; retinitis may be present. Baginsky refers to the lack of development of the children in these cases, and this condition was noticed in a case of this disease which occurred at the Boston Children's Hospital.

This child (Case 455), a girl, twelve years old, showed the development of a child of about seven years. The only symptom until she died of uræmic poisoning was persistent headache. The post-mortem examination showed marked interstitial nephritis, but it was not possible to determine whether it was primary or not, and no previous history could be obtained.

In chronic interstitial nephritis the amount of urine passed in the twenty-four hours is increased. It has a low specific gravity, a very slight sediment, and a trace of albumin. The microscopic examination shows a few hyaline and finely granular casts and occasional renal cells. Sometimes towards the end of the disease highly refractive homogeneous casts resembling wax appear in the urine. At this time the amount of urine may be somewhat diminished, but the specific gravity does not rise, as the excretion of urea is interfered with.

Prognosis.—The prognosis is very unfavorable. The children usually die of cerebral hemorrhage or of some intercurrent disease, the fatal result occurring in from three to four years.

TREATMENT.—The treatment is symptomatic.

Amyloid Infiltration.—In connection with amyloid changes in other organs, especially the liver, spleen, and intestine, amyloid infiltration may occur in the kidney.

ETIOLOGY AND PATHOLOGY.—It occurs at times in connection with chronic suppurative processes in the bones or elsewhere, and also in tuberculosis, syphilis, and chronic wasting diseases. It is not, however, especially common in early life.

SYMPTOMS.—The symptoms are not referable to the kidney. The presence of amyloid changes in the liver and spleen, shown clinically by enlargement and by the examination of the urine, are the signs by which the diagnosis is made. The urine is usually passed in large quantity when the amyloid changes are advanced. The specific gravity is low, and albumin is present. When the amount of urine is not much increased, as may happen temporarily, the albumin occurs in large amount. Microscopic examination shows no characteristic sediment; but when, as may often happen, the disease is combined with chronic nephritis, the sediment will show evidence of this latter disease.

PROGNOSIS.—On account of the usual causes of this condition, the prognosis is unfavorable.

TREATMENT.—The treatment is symptomatic.

Pyelitis and Pyelo-Nephritis.—ETIOLOGY.—Pyelitis and pyelonephritis may be due to an extension upward along the genito-urinary tract of an infection caused by catheters, gonorrhœa, or cystitis. Cases due to these causes, however, are uncommon in comparison with those which result from the excretion of uric acid by the kidney or from pelvic calculi. The disease may also be caused by tuberculosis of the kidney and by malignant growths.

PATHOLOGY.—The pathology of this disease varies with the cause. After the pyelitis has lasted for a time the kidney is affected in almost every case, and pyelo-nephritis results.

SYMPTOMS.—In an acute attack of the disease, as when it is caused by uric acid or a calculus, there are often pain and fever. Typical attacks of renal colic, with vomiting, pain, and fever, may occur. If the condition be due to tubercles, malignant growths, or abscess of the kidney, there will be more or less cachexia and emaciation, and there may be local pain and tenderness.

The diagnosis is to be made from the examination of the urine. The urine contains pus, which gives it a cloudy appearance, and the sediment is heavy. The color varies: it may be red if there is considerable hemorrhage. The urine contains albumin, which varies from a trace to a considerable amount, according to the amount of blood or pus and the presence or absence of a concurrent affection of the kidney. The microscopic examination shows sometimes the whole field to be filled with pus-corpuscles, at other times the pus to be in clumps; there are also present small round cells with single nuclei, from the pelvis or from the kidney, and more or less blood. The diagnostic cell of pyelitis is the "caudate cell," which is a small cell about the size of a renal cell, having a single nucleus and a tail. If the kidney is affected there are casts of various kinds, hyaline, granular, epithelial, and blood. The casts may not be easily seen if the field is filled with pus. The presence of tubercle-bacilli in the sediment, shown by appropriate methods of staining, establishes the diagnosis of tuberculosis. In the freshly passed urine, uric acid is often present in the sediment in the form of irregular spiculated crystals. These may suggest the probable cause of the pyelitis.

PROGNOSIS.—The prognosis depends upon the cause. In malignant growths it is fatal. This is true to a greater or less degree where tubercle is the cause of the disease, as in almost every case it is present somewhere else in the body. When uric acid or a calculus is the cause, the prognosis is more favorable, and, as a rule, the outcome depends upon the fact whether the treatment is appropriate or not.

TREATMENT.—The uric acid should be treated by neutralizing the acidity of the urine, by placing the child upon a mild and unirritating diet, such as milk, and by making it drink freely of distilled water. Operative treatment is at times called for where a calculus is present.

Malignant Growths and Enlargement.—Tumors of the kidney are more common and more serious in the child than in the adult. The simple adenomata are probably equally common in both, but the child is much more liable to carcinomata and sarcomata than is the adult. Sarcomata are the most common in the first five years of life, and usually occur in one kidney.

SYMPTOMS AND DIAGNOSIS.—The diagnosis depends upon the recognition of a tumor of the kidney and the progressive emaciation and cachexia which arise. At times there is pain, but, as a rule, pain is absent. The urine sometimes gives evidence of a pyelo-nephritis; at other times hæmaturia and albuminuria occur at intervals, but generally late in the disease, at a time when the tumor can be felt through the abdominal wall. Some of the characteristics of a tumor of the kidney are that it is located in the hypogastric and lumbar regions, that it is deep-seated, and that it is not so commonly to be felt in the umbilical region as are tumors of the retro-peritoneal glands. The tumor is irregularly rounded, and usually does not have a well-marked border, such as is found in enlargement of the spleen and liver. In these cases of sarcomata of the kidney the health at first is often not much affected, but there are progressive emaciation and enlargement of the abdomen, commonly without pain.

PROGNOSIS.—The prognosis is very unfavorable, although temporary relief is often obtained by means of surgical interference.

TREATMENT.—The treatment is essentially operative.

AFFECTIONS OF THE SUPRA-RENAL CAPSULES.—The affection of the supra-renal capsules called *Addison's disease* has been met with in young children, but is exceedingly rare.

HÆMATURIA AND HÆMOGLOBINURIA.—Hæmaturia and hæmoglobinuria are, as a rule, easily recognized by the color of the urine if sufficient blood is present to color it. The color is red if it is due to fresh blood, or brownish red if due to blood-pigment which has been washed out of the corpuscles.

To determine the source and cause of the hemorrhage is often quite difficult. Hemorrhage from the bladder may be caused by a calculus, or by papillomatous growths, or may occur in cases of hæmophilia. When the blood comes from the bladder it is generally not uniformly diffused through the urine, and small clots are common. In addition to this there are symptoms of disturbance of the bladder, such as tenesmus and frequent and perhaps interrupted micturition. In hemorrhage from the kidney the blood is diffused through the urine. The color may be red or brownish red. The microscopic examinations show epithelium and casts from the kidney, and the elements are stained yellow and brown from longer contact with the blood. There are also normal blood-corpuscles, and others from which the hæmoglobin has been washed out, appearing as pale rings.

Hæmaturia may occur in hæmophilia and in purpura. Hæmaturia may also be a symptom of malignant growth in the kidney. It may be an early symptom occurring at intervals, but usually it appears at a later period, when the presence of a tumor can be detected by palpation. It may also be caused by uric acid.

In cases of hæmoglobinuria, notwithstanding the red or at times almost black color of the urine and the presence of albumin, there are no corpuscles to be found. Heller's test, which consists in adding hydrate of potassium to the urine and heating it, causes a precipitation of the phosphates, which carry down the blood-pigment mechanically as dark-red flocculi. A similar appearance may be given to the urine after the administration of senna and rhubarb. In such cases Heller's test would give the same results as if blood-pigment were present. The nitric acid test for albumin would, however, decolorize the urine, and the test for albumin would be negative. It is important to recognize the very dark urine resulting from carbolic acid poisoning, as it occasionally occurs after the external application of this drug. Under these circumstances the urine has a greenish tinge.

ETIOLOGY.—The etiology of paroxysmal hæmoglobinuria is as yet obscure. The child often appears to be in good health. The most frequent apparent cause is cold. Certain individuals have hæmoglobinuria whenever they are chilled, or wet their feet, or plunge into cold water. Some cases of hæmoglobinuria appear to be due to infection, as in scarlet fever, Winckel's disease, and malaria. Certain inorganic substances when taken by the mouth, especially chlorate of potassium, phosphorus, and arsenic, have produced hæmoglobinuria.

Some cases of hæmoglobinuria have severe symptoms at the time of the attack, such as chills, cold extremities, and a rapid, small pulse. Neither these symptoms nor the hæmoglobinuria last very long, as a rule. At times it is impossible to determine the cause of the hæmoglobinuria. A case which has lately come under my notice shows how difficult it is to determine the cause of this disease even when a post-mortem examination can be obtained.

A girl (Case 456), four years and eight months old, had for several weeks grown pale, lost in weight, and shown symptoms of indigestion. Later the urine was reduced in amount and was dark-colored. There was also slight œdema of the eyelids and feet. No other especial symptoms arose, and the child went out of the house as usual and seemed otherwise well. For three or four days before her death the pallor and œdema increased markedly, and the urine was lessened in amount and became still darker in color. About twenty-four hours later she became very dull, and on the following day was much blanched and almost unconscious, except that when she was aroused to be examined she would resist and scream. The urine showed the condition of hæmoglobinuria. The child died a few hours later.

The post-mortem examination, made by Professor Councilman, showed evidences of profound anæmia. The bone-marrow was red. There were hæmoglobinuria, fatty degeneration of the heart, liver, and kidneys, and hæmoglobin casts in the tubules of the kidney.

CHYLURIA.—Chyluria is a rare disease. Two forms are usually spoken of, the tropical and the non-tropical.

ETIOLOGY.—The tropical form is caused by a parasite, the filaria sanguinis hominis, a species of round-worm. This parasite is found in the blood, and at times in the urine, especially that passed towards night. The exact connection between the parasite and the chyluria has not yet been determined. In the non-tropical form the parasite has not been found. Cases have been reported where the parasite appeared in an individual residing in the tropics, and disappeared on his returning to a cold climate, although the chyluria continued. The chyle is supposed to get into the urine after it has left the kidney.

SYMPTOMS.—The symptoms of this disease are shown chiefly in the urine. The urine has a milky appearance, sometimes a sour odor, and tends to decompose rapidly. The reaction is slightly acid, or neutral. Microscopic examination shows the fluid to be filled with fine fat drops in suspension. The urine at times contains blood-corpuscles, and albumin is always present. The attacks are apt to be paroxysmal, lasting for days or weeks, then ceasing and again recurring. A fatty diet may or may not cause an increase in the chyluria. The individuals affected by the disease may have a healthy appearance. Coagula may at times be formed in the bladder and give rise to pain and difficult micturition.

PROGNOSIS.—The prognosis of chyluria is doubtful. It is a disease which lasts for a long time and may cause anæmia and emaciation from the loss of fat and albumin.

TREATMENT.—There is no treatment which is known to be beneficial.

Hydronephrosis.—ETIOLOGY.—Hydronephrosis may be congenital, in which case it may be due to constriction of the ureter. Both kidneys may be affected, but usually only one is involved. When acquired it generally affects but one kidney, and may be caused by obstruction to the escape of urine either from above, as by an impacted calculus in the hilus of the kidney or in the urethra, or from below, by the pressure from a tumor or enlarged mesenteric glands. The effects are mechanical, and are due to the pressure of the retained fluid on the kidney, which leads to the gradual absorption of the kidney-substance. These tumors sometimes acquire a large size.

Symptoms.—The main symptom of hydronephrosis is the presence of an abdominal tumor connected with the kidney. When the tumor has grown sufficiently large, fluctuation can be usually detected, and aspiration gives a fluid which ordinarily contains urea. Subjective symptoms may be absent. If only one kidney is affected, the other performs the function of both, and the general condition of the child may remain good.

Prognosis.—The prognosis is doubtful. Cases have been operated upon with success both by aspiration and by removal of the tumor.

Treatment.—The treatment of this disease is essentially surgical.

Acute Cystitis.—Acute cystitis is not a common affection in infancy and childhood.

Etiology.—It may be caused by a vesical calculus, by irritants, such as turpentine, and also occasionally by the extension of infection through the genital tract.

Symptoms.—The symptoms of acute cystitis in children do not differ from those which are met with in the adult. The chief symptom is frequent and painful micturition. This local symptom is usually accompanied by fever, which may be high, and by general symptoms of malaise, fretfulness, and crying from vesical pain. The urine is passed in small quantities, and, as a rule, is of a reddish color. The specific gravity is high. When freshly passed it is acid, but it quickly becomes alkaline; there is a heavy sediment, and it contains a trace of albumin. Microscopic examination shows chiefly pus in large quantities, squamous epithelium, and some blood. To establish the diagnosis it is necessary to obtain the urine by the catheter, or, in females, first to wash out the vagina thoroughly, as the epithelium of the vagina and that of the bladder are very similar.

Prognosis.—The prognosis of acute cystitis is good after the removal of the cause.

Treatment.—The especial cause of the attack must be looked for, and removed if possible. The child should be kept perfectly quiet in bed, and should be made to drink a great deal of water. The diet should be of milk. Sedatives should be used freely.

Chronic Cystitis.—Chronic cystitis may be caused in children, as in adults, by a vesical calculus, by foreign bodies in the bladder, by tumors, by papillomata, and by tuberculosis. The nuclei of the calculi are generally composed of uric acid, upon which phosphates are precipitated in alkaline urine, and this deposition is favored by the accompanying catarrhal inflammation.

SYMPTOMS.—Micturition is frequent and at times painful. Later there nay be a constant dribbling of urine, giving rise to an offensive ammoniacal dor and causing irritation about the genitals. Where there is a calculus n the bladder the stream is often suddenly interrupted during micturition nd the pain is more severe. Prolapse of the rectum is not uncommon with tone. In addition to these local symptoms there are general symptoms of næmia and loss of weight. The urine is ammoniacally alkaline, offensive n odor, and turbid, has a heavy ropy sediment, and contains a trace of lbumin. The sediment should be examined as soon as possible after the rine is passed, because the ammonia which is produced from the urea disntegrates the cells. The examination will show a large quantity of pus, ome blood, bladder-epithelium, and crystals of triple phosphate and urate f ammonium.

PROGNOSIS.—The prognosis of chronic cystitis depends upon the cause, pon the length of time during which the disease has persisted, and the resence or absence of a secondary affection of the kidney.

TREATMENT.—The urine should be diluted by giving distilled water in arge amount. It may be rendered less irritating by such drugs as salol nd buchu, and less alkaline by benzoate of sodium. Washing out the ladder is of use in many cases, and local applications may be made in uberculosis of the organ. Operative treatment is indicated when a calculus s causing the disturbance.

At times it is exceedingly difficult to determine by the general symptoms hether a calculus is present in the bladder. I shall report to you a case hich illustrates this difficulty.

A boy (Case 457), seven years old, began to have pain of a spasmodic character in the egion of the bladder during micturition. In connection with the pain there would be a udden stoppage of the flow of the urine and a bearing-down feeling in the rectum. These ymptoms simulated those of a vesical calculus so closely as to render a differential diagnosis ery difficult. The boy was of a nervous temperament, and was rather anæmic, but otherise was well and strong. Nothing abnormal was detected about the prepuce or the rectum. he pain was so annoying and caused so much trouble that it was deemed advisable to ave the bladder examined for stone. An examination was made by Dr. Bradford, and othing abnormal was detected. After the bladder had been examined, a decided improvenent took place, apparently connected with the passing of the sound, and the boy recovered ntirely after remaining at home from school for a few weeks and having daily exercise in he open air.

VULVO-VAGINITIS.—Vulvo-vaginitis is a very common affection in ittle girls. It arises from a variety of irritations, one of which is the xyuris vermicularis. In a very large number of cases the gonococcus of Neisser has been found in the purulent secretion. The gonococcus was found in all of six cases lately treated at the Boston Children's Hospital. The isease may also arise in children who are very much debilitated, and is met with at times in scarlet fever and in measles. Again, it is not infrequent in anæmic girls, in whom it occurs without any apparent cause.

PATHOLOGY.—The labia are reddened and are more or less swollen.

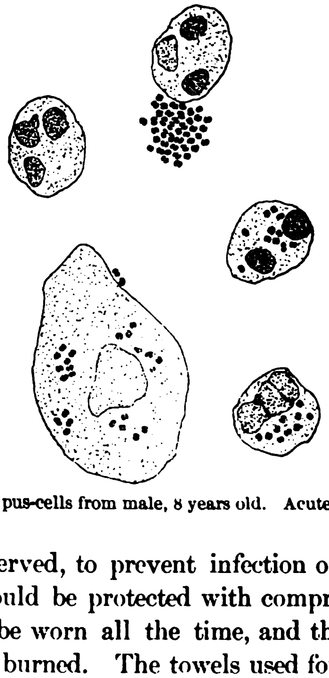
There is a thick, purulent discharge of a greenish-yellow color, usually offensive. At times there is more or less excoriation of the inner surfaces of the labia. The inguinal glands may be slightly enlarged and tender. The urethra is, as a rule, involved in the irritation, and is swollen and red.

SYMPTOMS.—There may be some fever in the early stages of vulvovaginitis. Smarting and burning are usually complained of, but at times the staining of the clothing first calls attention to the disease. The children commonly become pale if the disease persists for some time. Micturition is painful in some cases, and the disease is one of the many causes of dysuria. In many cases the children appear to be quite well, with the exception of the local condition.

PROGNOSIS.—The prognosis is good, but the disease is apt to be prolonged for several weeks or months. Complications may arise from the extension of the process into the urethra and the bladder, and cause additional symptoms referable to these parts.

TREATMENT.—Local applications to the vagina constitute the only satisfactory form of treatment. This is difficult in young children, but may be accomplished with a soft rubber catheter. Such solutions as boracic acid 4–100, corrosive sublimate 1–5000, or creolin 1–500, may be used. In some severe cases local applications of nitrate of silver 1 or 2 per cent. may be necessary. The labia should be kept separated by absorbent cotton, and the parts kept dry and covered with some mild dusting-powder. Absolute cleanliness must be observed, to prevent infection of the eyes and of other persons. The parts should be protected with compresses held in place by a bandage, which should be worn all the time, and the compresses should be frequently changed and burned. The towels used for the patient should not be left lying about, and should be carefully disinfected. Tonic treatment is

FIG. 182.



Gonococci contained in pus-cells from male, 8 years old. Acute stage of inflammation.

sometimes indicated. The urine should be kept dilute, in order to avoid irritating the inflamed surfaces, and any complicating cystitis should be treated. During the active stage of the disease the child should be kept as quiet as possible, and on a diet of milk.

Where the vulvo-vaginitis is caused by the oxyuris vermicularis, especial care should be given to eradicating the parasite from the rectum. After this has been done, the vagina is readily freed from the parasite by using an injection of warm sweet oil, which is to be allowed to remain for three or four minutes, the vagina then being syringed out with warm water.

Gonorrhœa also may occur in boys.

This boy (Case 458), eight years old, came to the hospital yesterday complaining of pain on micturition and on walking. The prepuce was found to be very much swollen, and there was a discharge of pus from the urethra. An examination of the discharge by Dr. Mallory showed the presence of gonococci in the pus-cells. This specimen (Fig. 130, page 944), taken from this case, shows the morphology of the parasite as seen by means of a Leitz homogeneous oil immersion $\frac{1}{12}$, Leitz stand Oc. No. 3, tube closed.

The gonococci are ovoidal or biscuit-shaped, and usually occur in pairs, the flat sides being opposed to each other. It is characteristic of them that they are found within the pus-cells as well as on their surfaces and free in the fluid.

ORCHITIS.—Orchitis, or inflammation of the testis proper, may occur in childhood from direct injury, but it is a rare disease. When present it is commonly accompanied by hydrocele. The orchitis which so commonly follows mumps in the adult is less common in children.

EPIDIDYMITIS.—Besides being due to trauma, acute epididymitis may be caused by any irritation of the mucous membrane of the urethra. In this disease the whole scrotum is apt to be hot and tender, and the child is in great pain. The epididymis is much enlarged and exquisitely tender, and pushes the testis forward. The cord is often implicated, becoming enlarged and painful on pressure.

The treatment should be energetic, as, owing to the swelling of the tissues about the testicle, there may be so much pressure that the gland will be seriously damaged, although the subsequent atrophy may not declare itself for a considerable time. The child should be kept upon his back in bed, the bowels freed with a cathartic, and a series of hot poultices kept upon the scrotum. In all inflammations of the testis or epididymis the scrotum should be placed in such a position that the lower end of the testicle points upward.

TUBERCULAR DISEASE OF THE TESTICLE.—As compared with the frequency of its occurrence in adults, tubercular disease of the testicle is rare in infancy and childhood. When the disease is present the gland is considerably swollen and often nodular, but rarely very tender. As the disease progresses, adhesions may form with the tissues of the scrotum, and the degenerated material may be discharged through a fistulous tract.

General treatment is indicated if the disease is just starting, but if it has already destroyed the usefulness of the gland it is safer to operate immediately and remove the focus of infection; here, of course, we should be guided by the conditions elsewhere.

Tumors.—In addition to tubercular disease of the testis, tumors may be found in infancy and early childhood. These may be congenital or acquired. The congenital tumors are very rare, and are usually of the dermoid variety. The most common of the acquired tumors are sarcomata, which are very malignant. The rapid growth and the large size of this variety usually render the diagnosis easy.

Phimosis.—In early life there appears to be a physiological adhesion of the prepuce to the glans penis. As the child grows older these adhesions normally disappear. When the adhesion between the prepuce and the glans remains permanent and the prepuce is very tight, the condition gives rise to various symptoms. Thus the escape of the urine may be mechanically hindered, and the urine collecting behind the glans may give rise to irritation. Smegma is also apt to collect around the corona. In this way an inflammatory condition of the prepuce (posthitis) or of the glans (balanitis) may arise. As a result of this there is swelling, and micturition is painful and difficult. In addition to these local symptoms many secondary disturbances arise from the local reflex irritation. Among these are nervous phenomena of greater or less degree, such as convulsions. Phimosis may lead to enuresis and masturbation.

In all cases of phimosis local treatment is indicated, and may be by dilatation, incision, or circumcision.—the latter being the most radical and producing the best results for complete relief from the morbid condition. In all cases, even if the phimosis is very slight, mechanical interference should be persisted in until absolute cleanliness can be secured, for in this way only will entire relief from the local and reflex symptoms be obtained.

Anuria.—I have already spoken of the forms of anuria which result from suppression of the urine in nephritis. Anuria may also occur in infants and in young children irrespective of any disease. The infant will not pass its water for perhaps twenty-four hours, apparently from no especial cause.

Hot applications over the bladder and making the child drink an increased amount of water will usually relieve this condition. It seldom calls for the use of the catheter, and serious results need not be apprehended.

Enuresis (Incontinence of Urine).—Enuresis is a condition in which there is an involuntary discharge of the urine. It may be continuous or periodic. It may also be diurnal, nocturnal, or both. It is of very frequent occurrence in infancy and early childhood. It is a symptom rather than a disease, and in most cases is a true neurosis. During the first year of life the infant has not learned to assume control of the mechanism of micturition, but during the second year this control is usually attained at an earlier or a later period according to the individual.

Etiology.—The causes of enuresis may be organic or functional, the latter in all probability being very commonly of a reflex nature.

The organic causes comprise such malformations as small ureters, a small bladder, exstrophy of the bladder, and hypospadias. Enuresis may also be caused by central lesions of the brain and cord.

The prognosis and treatment of these organic cases of enuresis vary according to the conditions which cause them, and need not be considered here. In a large number of cases the children are of a highly nervous temperament, but enuresis is also often present in children who otherwise do not show any nervous symptoms. As has been stated by Rachford in an admirable paper on this subject, this condition may depend upon (1) irritable and unstable nerve-centres, (2) anæmia with malnutrition, and (3) reflex stimulation of certain nerve-centres in the lumbar cord. The longitudinal and circular muscular fibres of the bladder, which by their contraction empty the bladder, are innervated by sensory and motor nerves from the lumbar region of the cord, and the external sphincter in the prostatic portion of the urethra, which by its contraction prevents the escape of urine from the bladder, is also innervated by sensory and motor nerves from the lumbar cord. The researches of Von Zeissl show the manner in which reflex causes may act in starting or checking the flow of the urine. Thus, a reflex carried to the proper centre in the lumbar cord would, through the motor fibres of the erector nerve, contract the muscular coat of the bladder, and through the inhibitory fibres of the same nerve relax the sphincter vesicæ. In this manner the urine which is being expelled by the contracting bladder is allowed to pass without hinderance through the relaxed sphincter vesicæ. It is also to be remembered that the act of urination is in part under the control of the will. Admitting these anatomical and psychical facts, it is easily understood how the causes which produce enuresis may act in two ways: either directly on the centres in the lumbar cord, making them more irritable or unstable, and in that way increasing their reflex excitability, or indirectly through exaggerated reflex causes that affect both accelerator and inhibitor influences sent to the bladder. These influences may be psychic, originating in the brain, or may be the result of external irritation originating in or near the bladder itself.

There is also during childhood a lack of development of the centres of inhibitory reflex acts, and in this way the muscular fibres of the bladder, having no inhibitory restraint, are excited to action by even so slight a reflex cause as a small quantity of urine in the bladder. For this reason enuresis is a normal condition during infancy, and ceases when the child's inhibitory mechanism is more developed (Soltmann). The inhibitory influence of the will is in abeyance during deep slumber, and nocturnal incontinence is therefore more frequent than diurnal. In any diseases which are accompanied by anæmia and malnutrition the reflex irritability of the lumbar nerve-centres is much increased, and enuresis may result. Reflex enuresis may be caused by irritation in any portion of the genito-urinary tract, as by a vesical calculus, cystitis, vulvitis, phimosis, very acid urine, and over-filling of the bladder, as in diabetes, or by an irritation of some

DIVISION XVI.

DISEASES OF THE LARYNX, TRACHEA, LUNGS, AND PLEURA.

LECTURE XLVIII.

DISEASES OF THE LARYNX AND TRACHEA.

LARYNGOSPASMUS.—NEW GROWTHS.—FOREIGN BODIES.—ŒDEMA.—LARYNGITIS.

LARYNX.—The affections of the larynx which occur most commonly in infants and young children are neuroses, new growths, lesions produced by foreign bodies, œdema, and laryngitis.

LARYNGOSPASMUS (Laryngismus Stridulus).—The neurosis which especially affects the larynx in infancy and childhood is what I have already described under the name of laryngospasmus when speaking of reflex irritation of the larynx in my lecture on Nervous Diseases (page 747). I shall therefore merely refer you to what I said at that time concerning it.

NEW GROWTHS.—New growths in the larynx in infants and children are rare. They may be congenital, but these are very uncommon. They may be malignant, such as epitheliomata and sarcomata, or benign, such as fibromata, myxomata, and papillomata. Those of the former class are so rare that they need here only be referred to. Of the latter class the fibromata and myxomata are too rare to be more than mentioned. The papillomata, on the other hand, although rare, are the most common laryngeal growths in early life. They may produce such serious results that it is important to recognize them at once. They may be congenital. Their cause is not known. Papilloma of the larynx in young children is usually multiple.

The symptoms of this growth appear at about the first, second, or third year. The first symptom that is noticed is hoarseness. This hoarseness, instead of passing off in a few days, as is common where it arises from other affections of the larynx, continues and grows more marked. The next symptom is dyspnœa. This appears at intervals of a few months, or may not arise for some years after the first alteration of the voice. The dyspnœa first appears at night, when the child is asleep. In the daytime, when the child is awake and running about, it may breathe freely. As the papillomata increase in size, the dyspnœa appears in the daytime also, especially when the child makes any exertion. When the child is awake and is quiet the breathing may not be noticeably affected, even after the growth has attained a large size. Cough may be present. Usually there is no pain or difficulty in swallowing. When a child presents these symptoms a careful laryngoscopic examination should be made at once, as in this way only can the diagnosis be verified.

The prognosis in these cases is bad unless the growths are removed.

The best treatment of multiple papillomata is to etherize the child and remove the growths through the mouth.

The difficulty of removal is in some cases so great that some of the most competent operators have preferred to postpone the operation until the child is older, or until the symptoms are so urgent that there is danger of suffocation. The child during this time must be kept under strict supervision, but local applications are not indicated. These growths, even when completely removed, have a tendency to recur.

FOREIGN BODIES.—Foreign bodies rarely lodge in the larynx, but this accident occurs more commonly in children than in adults, as children are apt to put articles of every description into their mouths.

The symptoms which indicate the presence of a foreign body in the larynx are an attack of sudden suffocation and a change in the sound of the voice in a child who has previously shown no signs of obstruction and no symptoms of laryngeal disease.

The accident is one which is so serious that the child should be placed at once in the hands of a laryngologist. The larynx should be examined with the laryngoscope, and the foreign body removed, if possible, with the forceps. Great care should be taken not to push the foreign body into the trachea, as tracheotomy would then be necessary. For the same reason it is inadvisable to introduce the finger blindly into the larynx, or to do anything which may cause a sudden inspiration.

ŒDEMA.—Œdema of the larynx is not a common condition in early life. It may arise from a number of causes, and is secondary to some disease elsewhere or to some local irritation. It occurs as a rare complication in nephritis and in the acute exanthemata. It may arise from irritation produced by local lesions, such as ulcerations, from foreign bodies, from inhalations of hot vapors, from the swallowing of corrosive liquids, and also as the result of any acute inflammation, such as erysipelas.

The diagnosis, as a rule, must be verified by a laryngoscopic examination.

The treatment is that of the disease or local irritation which is causing the œdema. The local application of cold, and, if necessary, scarification of the œdematous tissue, are indicated. If the attack is pronounced and suffocation is imminent, you should be in readiness to perform tracheotomy or intubation.

LARYNGITIS.—The most common inflammatory lesions of the larynx which occur in early life are (1) catarrhal and (2) pseudo-membranous.

Catarrhal laryngitis may be acute or chronic.

Acute Laryngitis.—The pathological condition which is present in the acute form of laryngitis is a redness or hyperæmia of the laryngeal mucous membrane, accompanied by more or less swelling and serous exudation. The cause of acute catarrhal laryngitis is often a simple extension of a catarrhal condition of the nose and pharynx to the larynx. More rarely a catarrhal condition of the bronchi and trachea may extend upward and involve the larynx. At times the condition appears to be the result of atmospheric changes and undue exposure to dampness and cold. The lumen of the larynx in infancy and in early childhood is so small that even a moderate swelling of the laryngeal mucous membrane may produce sufficient stenosis to give rise to marked obstructive symptoms.

SYMPTOMS.—The symptoms of acute laryngitis are a heightened temperature, 38.3°, 38.8°, 39.4° C. (101°, 102°, 103° F.), and even higher, hoarseness, and cough. These symptoms, occurring in connection with a preceding rhinitis or pharyngitis, or arising from a primary inflammation of the larynx, may continue for a number of days without any more serious manifestations, and if the child is kept in an equable temperature the attack may pass off within a week. In some cases, however, another set of symptoms may appear after the primary manifestations have lasted for a variable period. The child may have been as well as usual during the day, and may have been playing about. Towards the latter part of the day its voice may have become hoarser, but otherwise no especial symptoms may have arisen. The child, after being restless for a time, suddenly awakes, and springs up in bed frightened, often clutching at its throat as if it had a sensation of suffocation. The cough, which during the day was hoarse and somewhat metallic, is now loud and rasping. The child has difficulty in breathing, amounting to orthopnœa, and its face is congested. These symptoms continue for a variable period; usually they last for only one or two hours, but rarely they may continue for many hours. In one very uncommon case which was under my care the attack lasted for three or four weeks, during which time it often seemed as though suffocation was imminent. There was in this case no evidence of any lesion beyond a catarrhal laryngitis, and recovery finally took place. These attacks are partly due to obstruction in the larynx from the swollen mucous membrane, but are largely the result of a neurosis due to a highly sensitive condition of the mucous membrane. On the following day the hoarseness may continue, but the child may seem bright and may play about as usual. It is very common for the attack to recur on the second night with greater severity, but in certain cases one attack terminates the disease, and after a variable period of days, the voice becoming clearer each day and the temperature returning to normal, the child recovers. Children who have once had attacks of this kind are liable to have a recurrence until they grow older.

Diagnosis.—The diagnosis of acute catarrhal laryngitis is to be made from foreign bodies in the larynx, traumata, and membranous laryngitis. The symptoms in the first two are not preceded by catarrhal symptoms elsewhere, which are almost always met with in catarrhal laryngitis. In a typical case of acute catarrhal laryngitis with suffocative symptoms the diagnosis is not difficult. The acute, sudden onset of the attack in the night, the loud, metallic cough, and the heightened temperature, are distinctive from the moderate temperature and the slow, progressive stenosis caused by the formation of a membrane in the larynx.

Treatment.—The treatment of acute catarrhal laryngitis is to keep the child in a room of an equable temperature of about 20° to 21° C. (68° to 70° F.) until its temperature has become normal and the hoarseness has disappeared. I have also found that a few drops of wine of ipecac, given in the latter part of the afternoon and just as the child is going to sleep, are of benefit in preventing the spasmodic, obstructive symptoms which I have just described as occurring in the night. When the attack occurs in the night the symptoms of suffocation can be best relieved by a dose of from ten to fifteen drops of wine of ipecac, or an amount sufficient to nauseate slightly. An emetic will sometimes cut short an attack of this nature, but in many cases is not necessary. An amount of ipecac sufficient to nauseate slightly, but not to cause the child to vomit, will often so relax the spasm of the larynx that the attack will soon be relieved. In many cases, however, even if vomiting has been produced, the attack continues, and other measures for relief are required. In addition to the ipecac, moderate doses of tinctura opii camphorata may be given. An atmosphere of steam usually gives great relief to the spasm.

Acute laryngitis is a self-limited disease, and one in which the prognosis is almost invariably good. In children who are very weak and debilitated the interference with their respiration may prove to be serious, but these cases are rare and should be treated with stimulants until the disease has run its course. The symptoms of acute catarrhal laryngitis are so terrifying to the parents that the physician is often led to look upon the disease more seriously than is necessary. Many accidents have occurred from the improper management of the steam, from giving such emetics as turpeth mineral, and from the exhibition of strong drugs, the use of which is uncalled for. The necessity for operative measures rarely arises.

Chronic Laryngitis.—A chronic form of laryngitis occurs in both infants and children. Syphilitic infants, as I have already told you, are at times affected by chronic laryngitis. It may also occur in tubercular disease, but is not common. Where an acute laryngitis has occurred a number of times, or where an attack has been much prolonged by improper treatment, chronic laryngitis may result. In many of these cases the voice, on the slightest exposure to dampness, becomes hoarse, and this hoarseness, after a time, may be continuous.

The treatment is to apply astringents to the pharynx, which is almost universally involved, and to regulate the climatic surroundings of the child. Local applications to the larynx in these cases are seldom necessary.

Pseudo-Membranous Laryngitis.—A pseudo-membrane in the larynx may be caused by the inhalation of irritating vapors, or by the inspiration of corrosive liquids. These accidents are so readily recognized that there is no difficulty in determining the cause of the pseudo-membrane in these cases. Treatment for the relief of the stenosis should be instituted at once. This consists in the application of cold and such soothing inhalations as 3.75 c.c. (1 drachm) of compound tincture of benzoin in a quart of boiling water. The complicating œdema which is often present in these cases may require operative interference.

The most common cause of pseudo-membranous laryngitis, and the one which probably in all cases produces it, is some form of micro-organism. These micro-organisms, as I have already stated in my lecture on diphtheria, may be of several varieties. Until it is proved not to be so, however, pseudo-membranous laryngitis must be clinically looked upon as infectious and due to the Klebs-Loeffler bacillus. I must again impress upon you the fact that a simple catarrhal inflammation localized in the larynx may be produced by the Klebs-Loeffler bacillus. Pseudo-membranous laryngitis may then, until further investigations prove the contrary, be defined as an infectious inflammation of the mucous membrane of the larynx accompanied by a pseudo-membranous exudation, which may be caused by a number of micro-organisms, of which, according to our present knowledge, the Klebs-Loeffler bacillus is the most common.

I have described the symptoms, diagnosis, and treatment of pseudo-membranous laryngitis in a previous lecture (page 824), and shall, therefore, refer you to what I then said.

Some aid in the differential diagnosis of pseudo-membranous from acute catarrhal laryngitis can be obtained from the temperature, which in the latter is considerably raised, while in the former it is moderate and sometimes normal or subnormal. The slow course of a constitutional disease gradually causing obstruction is significant of this infectious form of laryngitis.

TRACHEA.—Pathological conditions of the trachea not connected with those of the air-passages above or below it are uncommon. The lesions of the trachea may be primary or secondary. In the latter they are merely an extension of the disease from the larynx or the bronchi, and do not play an especially significant part in the attack. The only primary disease of the trachea which is common in infancy and childhood is an acute inflammation occurring in its mucous lining. When this inflammatory condition is present, it produces an irritating cough which can usually be excited by gentle pressure over the trachea,—about the only method by which we can locate the disturbance.

The treatment is to protect the child from an atmosphere which is either too hot or too cold, from high winds, and from dust. Douching the front of the neck with cold water several times during the day is also desirable.

LECTURE XLIX.

DISEASES OF THE LUNGS.

BRONCHITIS.—BRONCHO-PNEUMONIA.—ATELECTASIS.—LOBAR PNEUMONIA.—GANGRENE.—TUBERCULOSIS.—PERTUSSIS.—ASTHMA.—PERIODIC CATARRH.

LUNGS.—The diseases which affect the lungs in infancy and childhood differ somewhat from the same diseases occurring in later life, on account of the differences which exist in the anatomical conditions at birth and during the early years of life, especially the first five. These differences I have described to you in previous lectures (pages 43 and 76). I then told you that the principal differences were that the bronchi occupied a relatively larger portion of the lung in the child than in the adult, that in the former the interstitial tissue was present in a larger amount, that the cavities of the air-vesicles were smaller, and that their walls were relatively thicker; also that the epithelial cells lining the air-vesicles were very numerous. These cells in inflammation tend to rapid cell-division, which is one of the characteristics that mark the pneumonia of childhood. These anatomical differences are of great significance when any part of the lung is diseased, and tend to make a congested lung of much more serious import in the young child than in the adult. I shall not attempt to describe to you all the various pathological conditions which may occur in the child's lungs, but shall restrict myself to those clinical groups of symptoms which represent the especial diseases. In order to do this I shall designate the disease according as the bronchi, the alveoli, or other parts of the lungs are most affected. You must remember that post-mortem examinations often show various lesions which during life were not represented by any definite symptoms, so that we cannot expect the clinical diagnosis to include entirely the pathological lesions. Beginning with the part of the lungs which is a direct continuation of the larynx and the trachea, I shall first speak of bronchitis.

BRONCHITIS.—Bronchitis is often secondary to some other disease, or to a direct extension from an inflammatory condition of the upper air-passages. In a number of cases, however, the group of symptoms by which we determine that bronchitis is present is so prominent from the very beginning of the attack that clinically we can describe a primary bronchitis.

By bronchitis we mean an inflammation of both the large and the small bronchi, with the exception of the ultimate divisions which lead directly into the alveoli, and which probably are never affected without involving the alveoli also. The disease may be acute or chronic.

The anatomical peculiarities of the mucous membrane lining the bronchial tubes—namely, the prominence of its capillaries and its comparatively loose connection to the muscular walls—render the bronchial mucous membrane peculiarly susceptible to congestion. Exposure to sudden atmospheric changes, especially humidity, appears to be of great etiological importance in the production of bronchitis. Any impurity of an irritating nature in the inspired air may in certain individuals result in an attack of bronchitis. A catarrhal inflammation of the upper air-passages is often followed by a similar inflammation of the bronchial mucous membrane. Bronchitis is of frequent occurrence in pertussis and measles. It is in children often a prominent symptom of typhoid fever, and is a frequent complication of pulmonary tuberculosis and epidemic influenza. There are also certain diseases of nutrition in which bronchitis frequently occurs. The most prominent of these is rhachitis, in which the complication of bronchitis is often of serious import.

Acute Bronchitis.—PATHOLOGY.—The pathological conditions which are present in acute catarrhal bronchitis are, according to Delafield and Prudden, a congestion and swelling of the mucous membrane, and an arrest of the functions of the mucous glands. Later, the mucous glands resume their functions with increased activity, the congestion diminishes, there is an increased desquamation of epithelium, an increased formation of the deeper epithelial cells, a moderate emigration of white blood-cells, and sometimes the red blood-cells also escape through the vessels. The whole process is a superficial one, and does not produce any change in the walls of the bronchi beneath the mucous membrane, unless it has persisted for some time, when there may be a slight thickening of the walls. When the inflammation involves the smaller bronchi they may be occluded. The occlusion of the smaller bronchi may result in the collapse of the group of air-vesicles to which they lead, and thus will be produced areas of atelectasis, which may be further changed by inflammatory processes. The bronchial glands are frequently enlarged, even in mild attacks of bronchitis.

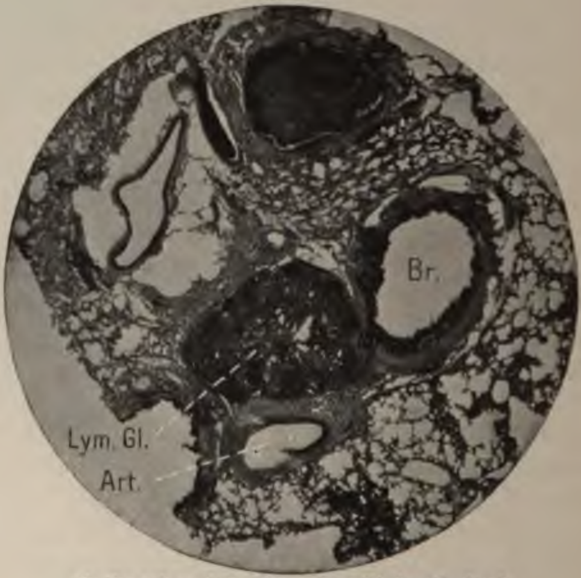
I have here the section of a lung (Fig. 133, page 956), made by Northrup, taken from a child, which shows the exudative inflammation of the bronchi which occurs in acute bronchitis.

The specimen shows hyperplasia of the lymph-glands due to bronchitis. This condition is very commonly found in bronchitis, especially when it occurs in debilitated children. There is desquamation of the epithelium lining the bronchi, as well as a slight thickening of their walls.

SYMPTOMS.—The onset of acute bronchitis is usually mild, but I have seen in a debilitated infant a simple, uncomplicated bronchitis begin with a convulsion. The symptoms are very variable in their intensity, and are usually more acute and definite in a previously healthy child than in debilitated children, in whom they are often subacute and of an insidious nature. In infants and young children the bronchitis is almost always preceded by a catarrhal condition of the upper air-passages. In the mild cases there is a heightening of the temperature, 37.7° to 38.3° C. (100° to

101° F.), cough of greater or less severity, and a slight lessening of the appetite. On physical examination the pulmonary resonance is found to be normal. A few sibilant and sonorous râles are heard with especial frequency in the area between the scapula and the vertebral column. Moist râles may also be heard. In severe cases the children suffer from more or less discomfort, produced probably by the thoracic pain, although in young children the locality of the pain cannot, as a rule, be determined. The cough is hard and dry, the respirations may be slightly raised, and the pulse quickened. The children may appear quite sick for two or three days, and the temperature may rise as high as 38.8° or 39.1° C. (102° or 102.5° F.); but when this latter point is reached the onset of a broncho-pneumonia should be carefully watched for, especially if after from twenty-four to forty-eight hours the temperature does not fall to 37.7° or 38.3° C. (100° or 101° F.).

FIG. 133.



Br., bronchus; Art., artery; Lym. Gl., lymph gland.

After a few days the severity of the symptoms lessens, the cough becomes looser, the râles gradually disappear, and under favorable conditions the symptoms subside entirely in a week or ten days. There is seldom any expectoration in children under six or seven years. In the more severe cases the râles are more numerous than in the mild form of the disease, but are of the same character. In the course of some cases of bronchitis a temporary localized diminution or even absence of the respiratory sound may result from the occlusion of a bronchus. This is especially common in infants, and ordinarily is not accompanied by a change in the percussion-sound. This form of bronchitis is the one which affects the larger and the medium-sized bronchi.

There is no characteristic temperature in bronchitis. As a rule, it is moderate, 37.2° to 38.3° C. (99° to 101° F.), but it varies greatly according to the individual and to the degree of nervous excitement.

DIAGNOSIS.—The diagnosis of the ordinary cases of acute bronchitis, where only the large- and medium-sized bronchi are affected, is not difficult, the only disease for which it is likely to be mistaken being broncho-pneumonia. In this latter disease the greater severity of the symptoms and the higher temperature will usually show its presence, even though the physical signs may be only those which I have described as occurring in bronchitis. In the more severe forms of bronchitis it is sometimes exceedingly difficult to make the differential diagnosis from broncho-pneumonia. If, however, the temperature, after three or four days, remains high, and rises to 39.1° or 39.4° C. (102.5° or 103° F.), with marked remissions and exacerbations, the diagnosis becomes doubtful, and in these cases we should strongly suspect that a broncho-pneumonia has arisen as a complication. We must, however, remember that in certain cases of broncho-pneumonia the temperature may be as moderate as in acute bronchitis, and we must therefore rely on a combination of symptoms rather than on any one symptom or sign. An important point in the differential diagnosis between bronchitis and broncho-pneumonia is that the physical signs in the former are much more frequently found in all parts of the thorax, while in the latter circumscribed groups of râles are often detected in different parts of the lungs. The râles in themselves, however, are not distinctive, as the râles in broncho-pneumonia are mostly those of the accompanying bronchitis. Although the physical signs of dulness and bronchial respiration are conclusive evidences that the case is not one of bronchitis alone, yet an absence of these signs does not justify us in excluding broncho-pneumonia. Where the dyspnœa, general prostration, and restlessness are slight and the temperature moderate, the case is likely to be one of bronchitis, while if these symptoms are marked, and are combined with cyanosis, dilatation of the alæ nasi, and a higher temperature, at least a provisional diagnosis of broncho-pneumonia should be made. In some cases the differential diagnosis will also have to be made from the onset of a pleuritis or of a lobar pneumonia, but the moderate temperature and respirations, the normal percussion-sounds, and the diffuse bilateral râles in bronchitis usually make the diagnosis from these diseases quite evident.

PROGNOSIS.—The prognosis, where no complication arises and the child is previously healthy, is good. In debilitated children, and especially where rhachitis is present, even a mild form of bronchitis may prove to be serious, on account of the danger of a complicating broncho-pneumonia, and in these cases the prognosis is much more unfavorable.

TREATMENT.—The treatment of acute bronchitis is essentially hygienic. The child should be confined to a warm, well-ventilated room which has a sunny exposure, and which is heated by an open fire to a temperature of about 20° to 21.1° C. (68° to 70° F.). A few drops of wine of ipecac should be given if the cough is unusually dry, and to this a few drops of tinctura opii camphorata may be added if the patient is excessively nervous. These remedies are all that will usually be needed in an attack of acute bronchitis. Where a rhachitic child or one who is much debilitated is attacked by the disease, especial care must be taken to support its strength by stimulants and food.

Besides the acute bronchitis which I have just described, I have met with a class of cases which are extremely rare, but which, apparently, are instances of an exacerbation of an ordinary bronchitis through the involvement of the smaller bronchi, *not the terminal ones*. I have seen only six of these cases. These, from their clinical history, seem to have been cases of bronchitis rather than of broncho-pneumonia. I speak of them separately, as the symptoms differ somewhat from those of an ordinary bronchitis. This form of bronchitis has no connection with what was formerly erroneously called capillary bronchitis, but which is now well known to be only an early stage of broncho-pneumonia. This form of bronchitis in my cases has commonly occurred in infants in the first two years of life, though I have met with it as late as the third year. The cause, so far as could be ascertained, was the same as in an ordinary bronchitis, a catarrhal condition of the upper air-passages usually preceding the attack. The onset of the disease was rapid, and the symptoms soon became very severe. The temperature was, as a rule, moderately raised, 37.7° to 38.3° C. (100° to 101° F.). The cough was continuous, and dyspnœa, with more or less cyanosis, rapidly developed. An examination showed normal resonance through the whole thorax, and fine moist râles. The respirations were rapid, the pulse was quick, and all the symptoms were of a violent and suffocative nature. The infants were much distressed, and were unwilling to be laid down. After from twenty-four to forty-eight hours the symptoms grew less severe, the temperature became normal or was only slightly raised, and the fine moist râles were replaced by coarse moist râles and the sibilant and sonorous râles of an ordinary bronchitis of the larger and the medium-sized bronchi.

In the early hours and days of the disease, when the symptoms are at their height, and if the infant is weak and debilitated, the prognosis is bad. If, however, the first few days are passed in safety, recovery almost invariably takes place.

This form of bronchitis is to be differentiated from broncho-pneumonia. The temperature, instead of remaining high and having the remissions of a broncho-pneumonia, soon falls so as to correspond to that of an ordinary bronchitis. The physical signs are those of bronchitis rather than of pneumonia, and the rapid recovery of the infant with the common symptoms of an ordinary bronchitis, rather than with the prolonged and characteristic symptoms of a broncho-pneumonia, verifies the diagnosis of an inflammation of the smaller bronchi.

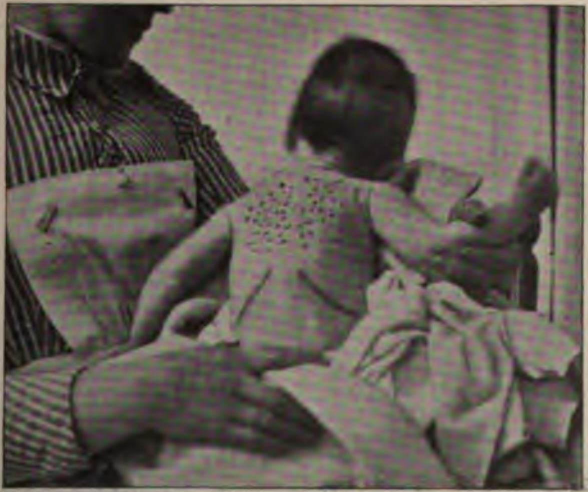
These cases may be complicated with broncho-pneumonia, as are the ordinary cases of bronchitis.

The treatment of this class of cases is very important, as death from ustion is liable to occur at any moment. The extreme congestion of blood-vessels of the smaller bronchi may in some cases occlude the air-

CASE 459.

I.

II.



Acute bronchitis. Female, 3 months old.

es, and areas of atelectasis may result. The indications for treatment to oxygenate the blood, to support the strength until the disease has run its course, and to prevent the infant from falling into a comatose condition. The treatment, therefore, is the administration of oxygen, the use of stimulants, consisting of aromatic spirit of ammonia alternating with brandy, and change of the position of the infant from time to time.

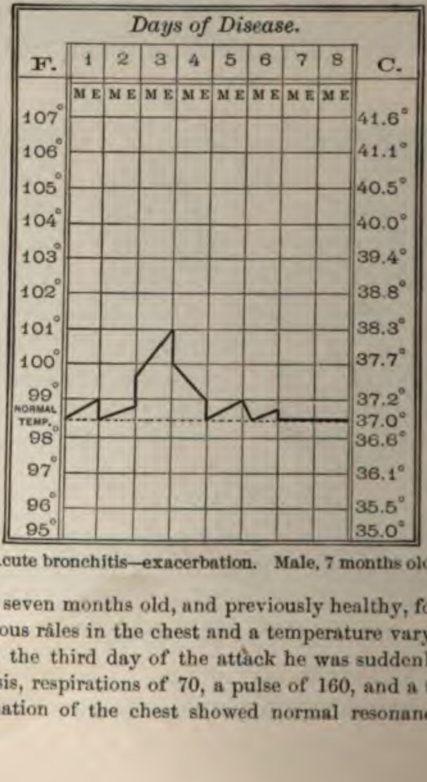
Here is an infant (Case 459, page 959), three months old, who has for the past few days had an attack of acute bronchitis, characterized by a paroxysmal, dry cough, slightly accelerated respirations and pulse, and a moderate temperature varying from 37.7° to 38.8° C. (100° to 101° F.).

The percussion of the chest has been normal, and there have been some sonorous and sibilant râles, with a few coarse moist râles heard on both sides of the chest. Early this morning the infant was attacked with excessive dyspnœa and cyanosis. Its pulse rose from 120 to 180, its respirations from 30 to 70, and its temperature from 37.7° C. to 39.1° C. (100° F. to 102.5° F.). An examination of the chest showed normal resonance and fine moist râles throughout both lungs. It has been very restless, refuses to take its food, and evidently wishes not to be laid down in its bed, but to be carried about. It is being treated with alternate doses of aromatic spirit of ammonia and brandy every half-hour. The physical signs are those of a diffuse bronchitis of the smaller bronchi, which you see I have indicated by small black spots painted on the front and back of the chest.

(Subsequent history.) After twenty-four hours the temperature fell to 38° C. (100.5° F.), the pulse to 150, and the respirations to 44. The fine râles were replaced by the ordinary coarse râles of a bronchitis, and the infant rapidly recovered.

The symptoms and course of all these cases are very similar, so that I shall speak only of one other child, whom I saw in consultation with Dr. Horace Marion, of Brighton.

CHART 32.



Acute bronchitis—exacerbation. Male, 7 months old.

A male (Case 460), seven months old, and previously healthy, for two days had a slight cough, with a few sonorous râles in the chest and a temperature varying from 36.6° to 37.2° C. (98° to 99° F.). On the third day of the attack he was suddenly seized with increased cough, dyspnœa, cyanosis, respirations of 70, a pulse of 160, and a temperature of 38.3° C. (101° F.). An examination of the chest showed normal resonance and fine moist râles throughout both lungs. The infant was treated with aromatic spirit of ammonia and brandy in alternate doses. On the following day the temperature fell to 37° C. (98.6° F.), and the fine râles were replaced by coarse râles and sonorous râles. The bronchitis lasted for a few days, and the infant then recovered entirely.

Here is the chart (Chart 32, page 960) which shows the sudden rise of temperature.

Chronic Bronchitis.—Chronic bronchitis may result from a series of attacks of acute bronchitis, or from a number of other causes. Among these may be mentioned various affections of the lungs, such diseases connected with malnutrition as rhachitis, and prolonged attacks of pertussis.

The pathological conditions occurring in chronic bronchitis vary greatly in degree, and the lesions found at the post-mortem examination are often slight in comparison with the severity of the symptoms during life. In most cases there is a considerable production of mucus, pus, and serum. In cases which have lasted for a long time, in addition to the inflammatory products affecting the walls of the bronchi there may be dilatation of one or more bronchi, and the muscular coat may be thickened or thinned. Emphysema may also result.

The symptoms of chronic bronchitis are very much the same as those of acute bronchitis, except that the temperature is not so apt to be heightened, while the general symptoms of malaise, anorexia, and loss of weight are more prominent. In severe and prolonged cases where emphysema is present, the thorax may assume the position of full inspiration, the ribs being permanently raised and the antero-posterior diameter of the chest increased. The physical signs are the same as in acute bronchitis, so far as the râles are concerned. The resonance is usually normal except where the chronic process has produced emphysema, in which case there will be areas of hyper-resonance often associated with a tympanitic tone. Occasionally atelectasis of considerable areas of the lungs may take place, with a resulting lessening of the respiratory sound. This occurrence may in some cases prove to be serious, but in others the accompanying symptoms are mild, and the alveoli may again return to their normal degree of inflation.

The differential diagnosis is to be made from chronic affections of the lungs in which the thickening of the interstitial tissue has taken place with a resulting lessening of resonance, and from the condition in which the bronchi are dilated. In the latter case there are accompanying symptoms of a profuse exudation of purulent matter.

There is one form of bronchitis which from its duration may be called chronic, and yet which from the very slight degree of constitutional symptoms that accompany it corresponds rather to a subacute affection. In these cases, which usually occur in infancy and in early childhood, the child often appears quite well, but for long periods of weeks, or whenever it is exposed to a damp atmosphere, a loud wheezing will be heard in the chest. Auscultation will reveal the presence of sonorous râles everywhere, and in this variety, as well as in other forms of chronic bronchitis, a roughened sensation may sometimes be felt on palpation during respiration.

The prognosis of chronic bronchitis varies according to the cause. Where it is secondary to disease of some other organ, it depends entirely upon the prognosis of that disease. In rhachitic children the prognosis is unfavorable, and in them a broncho-pneumonia is especially liable to develop, with a fatal issue. Cases of chronic bronchitis are also liable to be invaded by the bacillus tuberculosis. In cases which are the result of acute bronchitis in individuals otherwise healthy, the prognosis is favorable, provided the proper treatment can be carried out. As emphysema in chronic bronchitis is rare in children in comparison with adults, the chances for recovery in the former are correspondingly good.

The treatment of chronic bronchitis is essentially climatic. The children should be kept in a warm dry climate for some months after the bronchitis has entirely disappeared. Especial care should be taken that the child is suitably protected by flannel undergarments. Where other treatment is required, as a rule, tonics will prove of more benefit than the drugs which are usually administered for their direct effect upon the bronchial mucous membrane.

Fibrinous Bronchitis.—During the course of what may appear to be an ordinary bronchitis, in rare instances a fibrinous form of bronchitis has been met with. In this variety masses of fibrin in the bronchi form casts of various extent according to the number of the bronchi which are affected.

The disease may run a short course of days or weeks, but is usually chronic and may last for years. The paroxysms may also be periodic.

The diagnosis can be made only when portions of the casts have been expectorated.

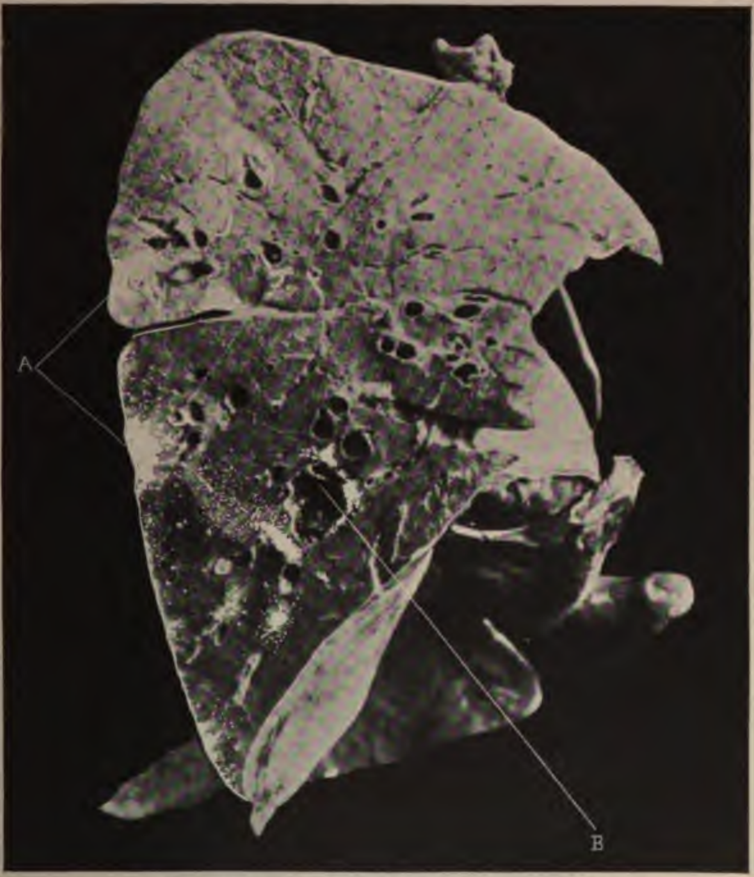
The treatment is chiefly by the inhalation of steam from lime water, and by supporting the strength with proper nourishment and stimulants until the disease has run its course.

Broncho-Pneumonia.—Broncho-pneumonia is an affection of the lung characterized by an inflammation of the walls of the terminal bronchi and of the alveoli. The disease may be acute or chronic. It may occur at any age, but is the most common and fatal form of inflammation of the lung during the first five years of life, and is much more fatal than lobar pneumonia at this period. During this early period, and especially during the first two or three years of life, the lung, from its embryonic type, is more frequently subject to the form of inflammation occurring in broncho-pneumonia than at a later and more developed period. The disease is usually secondary to bronchitis, and commonly occurs in connection with measles, scarlet fever, pertussis, and diphtheria. Broncho-pneumonia is also a very important disease, not only as grave in itself, but also because it is so frequently followed by tuberculosis.

Etiology.—A prominent predisposing cause of broncho-pneumonia is age, and where pneumonia occurs in a child under five years of age it is usually in the form of broncho-pneumonia. This is due principally, as I have already stated, to the anatomical conditions met with in early life.

Children who are weak or debilitated by previous diseases show a predisposition to broncho-pneumonia, and it therefore frequently arises in the course of tuberculosis, chronic gastro-enteric diseases, and rhachitis. Those seasons of the year which are marked by cold, moisture, and variations of temperature especially predispose to the development of broncho-pneumonia. All these conditions, however, in all probability merely prepare the way for the entrance of certain germs which produce the disease. What these micro-organisms are is still uncertain, as it is known that a number of different organisms can produce the disease. The origin of broncho-pneumonia from intestinal infection must also be considered. (Sevestre.)

FIG. 134.



Acute broncho-pneumonia involving different areas of the lung. A, consolidated lung-tissue; B, dilated bronchus. (Warren Museum, Harvard University.)

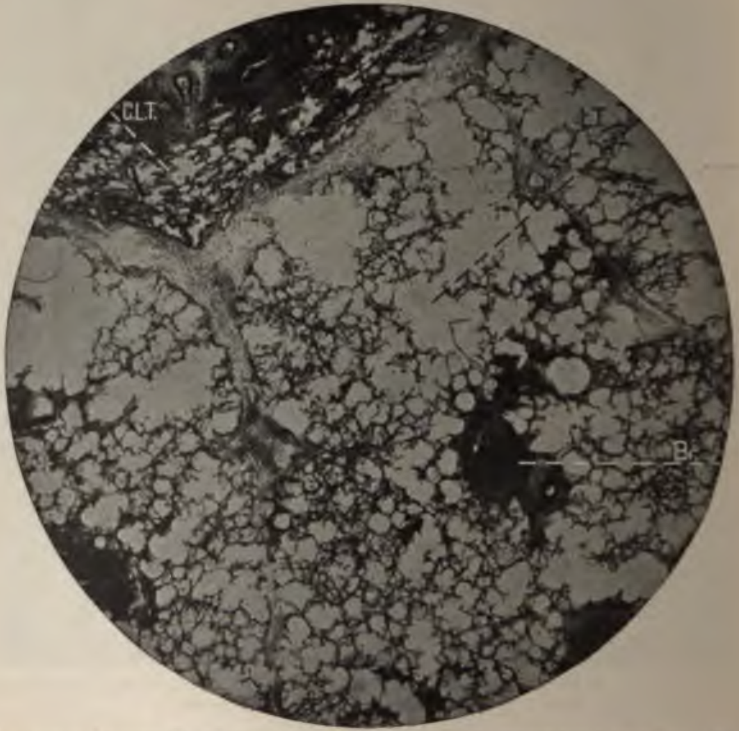
PATHOLOGY.—In broncho-pneumonia the inflammatory process affects the walls of the smaller and terminal bronchi, which become thickened and markedly infiltrated with cells. The inflammatory process then extends through the walls of the bronchi to the surrounding air-vesicles as well as to the terminal ones. In this way centres of consolidation are formed in different parts of the lung. The course of this inflammation varies in its rapidity, at times attacking only a small portion of the lung, and again being more diffuse in its onset and gradually invading large areas. The lesions are irregular in their distribution, and usually occur in both lungs. They are at times so extensive as to involve a whole lobe, but, as has been stated by Northrup, whatever the extent of hepatization, whatever the time occupied in its course, and whatever the post-mortem appearances, the essential lesion is an inflammation of the walls of the terminal bronchi and of the adjacent alveoli.

This lung (Fig. 134, page 963), taken from a young child, presents the macroscopic lesions of broncho-pneumonia.

You will notice that the areas of consolidation surround the bronchi, and that this bronchus (B) is markedly dilated.

This section of a lung (Fig. 135), made by Northrup, was taken from an infant sixteen months old, in whom the broncho-pneumonia was a complication of measles. It shows the early pathological lesions of broncho-pneumonia.

FIG. 135.

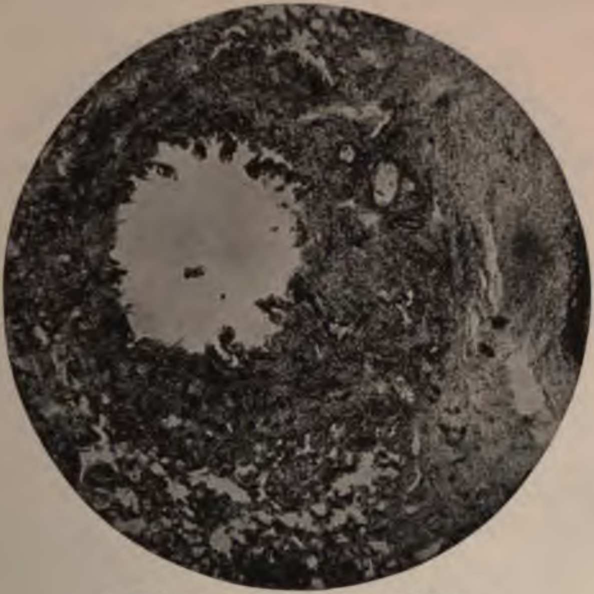


Broncho-pneumonia complicating measles. Early stage. C. L. T., consolidated lung tissue; Br., bronchiole; L. T., emphysematous lung tissue.

You will notice that in one of the lobules there are two bronchioles (Br.) with infiltrated walls and pus within them. They are also filled with exudation, and the lumen of each is almost entirely occluded. A portion of a neighboring lobule is consolidated (C. L. T.). A considerable portion of the lung tissue (L. T.) in the section is, as you see, normal or emphysematous.

Here is another section (Fig. 136), made by Northrup, illustrating the broncho-pneumonia which follows a diphtheria descending from the upper air-passages, and which may occur in any acute infectious disease. It shows the typical lesion of broncho-pneumonia.

FIG. 136.



Broncho-pneumonia secondary to diphtheria.

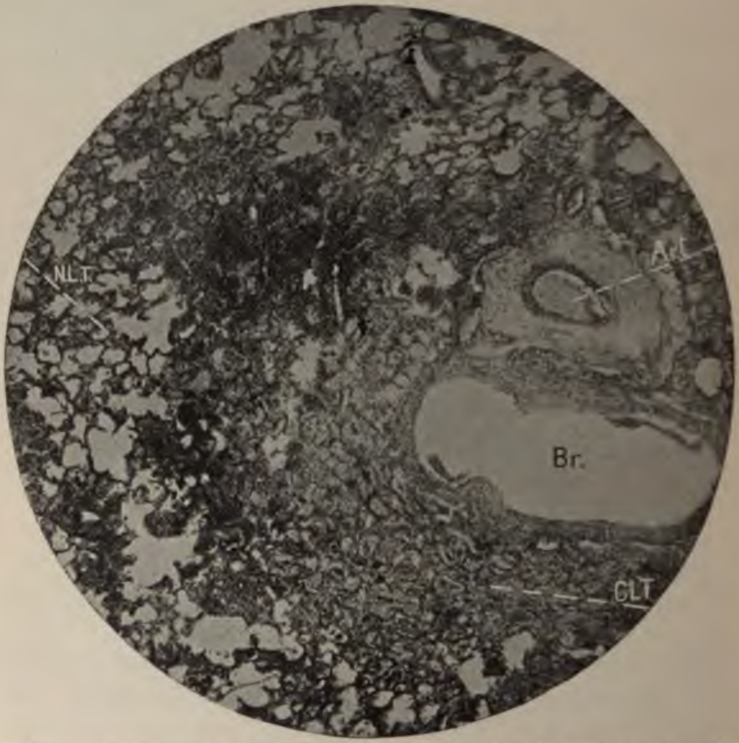
In the bronchus, which you see enlarges in the middle of the section, the lining mucous membrance is hanging in shreds into its lumen. The walls of the bronchus are densely infiltrated, and the contiguous alveoli are filled with exudation to a greater or less extent and are consolidated by it. This section was taken from the lung of a child three years old who during an attack of scarlet fever developed diphtheria of the larynx. The diphtheritic process descended later into the bronchi.

Here is another section (Fig. 137, page 966) taken from the lung of the same child, but showing the tissue relatively less affected.

Many of the consolidated alveoli contain free blood-cells. The bronchial wall (Br.) is infiltrated and almost entirely denuded of its lining membrane.

These smaller bronchi are surrounded by zones of intense congestion and infiltration. When the inflammation is intense and is accompanied by abundant secretion these bronchi frequently become dilated. This dilatation is associated with a weakened condition of the bronchial walls and with an abundant secretion. These dilatations probably, according to the observations of Northrup, wholly disappear on the recovery of the patient. As has already been described in the pathology of bronchitis, the bronchial lymph-glands are always enlarged in broncho-pneumonia, and there may be fibrin on the pulmonary pleura. According to Delafield, in the zones of peribronchitic pneumonia the walls of the air-vesicles are thickened or swollen, either with or without some cellular infiltration, and the cavities of the air-vesicles are filled with epithelial cells and pus-cells, with fibrin and red blood-corpuscles in varying proportion and amount. Fibrin when present is only in small quantities, and often is absent altogether. The capillaries in the walls of the vesicles are congested and prominent. The portions of lung which are not hepatized are congested and œdematous. The cavities of the air-vesicles are diminished by the enlarged capillaries and the swollen vesicular epithelium.

FIG. 137.



Broncho-pneumonia secondary to diphtheria. Br., bronchus; C. L. T., consolidated lung-tissue; N. L. T., lung-tissue nearly normal; Art., artery.

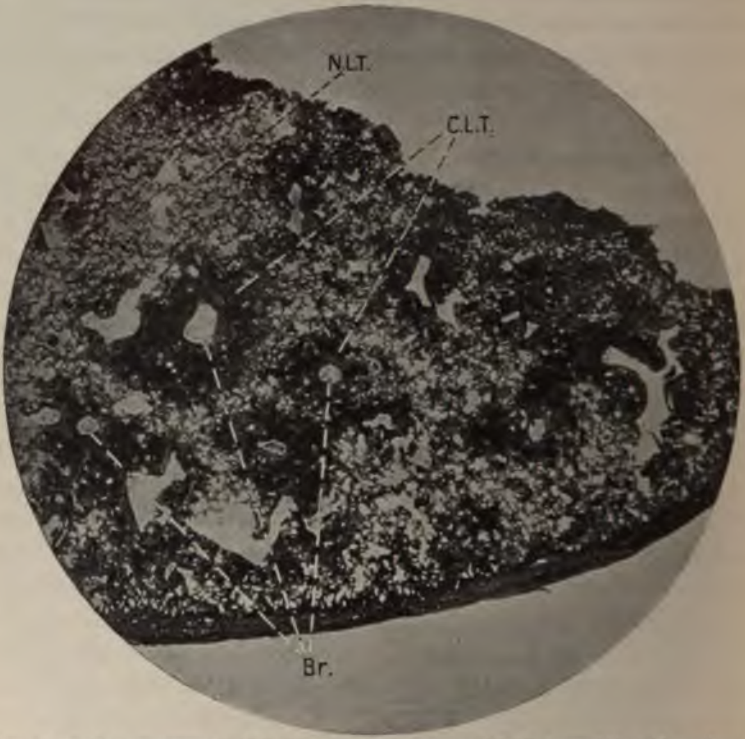
In addition to the other lesions which I have just described, areas of atelectasis are frequently found in broncho-pneumonia. This atelectasis is usually produced by mechanical causes, such as obstruction by pus or tenacious mucus. It may also arise as a result of enfeebled respiratory power. The blood-vessels become dilated, the walls of the alveoli partially collapse, the residual air is absorbed, and an exudation of serum with proliferative cells and leucocytes takes its place. The atelectasis is commonly symmetrical, affecting the posterior margin of both lower lobes of the lung, but it may also appear in irregular scattered areas in the posterior portions of the upper lobes (Northrup). It may occur either during the acute stage of the inflammation or later when the pneumonia has become chronic. There are no distinct stages in the pathology of broncho-pneumonia which correspond to those of lobar pneumonia. According to Northrup, broncho-pneumonia develops by the irregular invasion of successive portions of the lungs, and the process resolves in like manner. The different consolidated areas in the same lung may often show all the stages. The mottled appearance which is so often noticed macroscopically in these lungs may be caused by the presence of lobules of gray and red hepatization lying side by side. Of these inflammatory products the fibrin disintegrates quickly, and is therefore absorbed more rapidly than the cellular elements, which do not disintegrate so readily. In lobar pneumonia, therefore, absorption takes place sooner than it does in broncho-pneumonia, where the products of inflammation are mostly cellular and resolution and absorption are naturally slow.

Instead of the gradual disappearance of the various pathological lesions the pneumonia may persist. This persistent form of the disease may, according to Delafield and Prudden, follow a single attack of acute broncho-pneumonia, or there may be several acute attacks before the chronic condition becomes evident, and the course of the disease may thus vary in different cases. When this persistent broncho-pneumonia occurs, the proliferative cells take part in the formation of new connective tissue, and in this way persistent thickening is caused. The alveolar walls of certain portions may become similarly thickened. The walls of the bronchi and their surrounding tissue are especially subject to a persistent thickening and a formation of new connective tissue constituting chronic broncho-pneumonia and peribronchitis. The bronchi already dilated become still more enlarged by the contraction of the cicatricial tissue surrounding them. The uneven contraction of this new tissue, together with the pressure within the tubes facilitated by a weakened condition of the walls, allows of saccular as well as of fusiform dilatation of the bronchi. The epithelial cells of the dilated bronchi proliferate, and, falling from the bronchial walls, mix with the bronchial secretion. The remaining epithelium is swollen and loose. The lesions of chronic broncho-pneumonia are frequently associated with tuberculosis of the bronchial glands and with other tubercular lesions.

In connection with the pathological lesions occurring in chronic broncho-pneumonia a condition called *fibroid phthisis* has in very rare cases been noticed in children. The lesions which represent fibroid phthisis are manifested in the presence of connective tissue in the lung, with a corresponding destruction of the true parenchyma. These changes are usually unilateral, and should not be considered as representing a disease, since they merely occur in the course of various chronic pulmonary affections, among which are tuberculosis and chronic broncho-pneumonia.

Under this microscope (Fig. 138) you will see a section of the lung, made by Dr. Northrup, taken from a young child with chronic broncho-pneumonia.

FIG. 138.



Chronic broncho-pneumonia. N. L. T., normal lung-tissue; C. L. T., consolidated lung-tissue; Br., bronchi, some of them dilated.

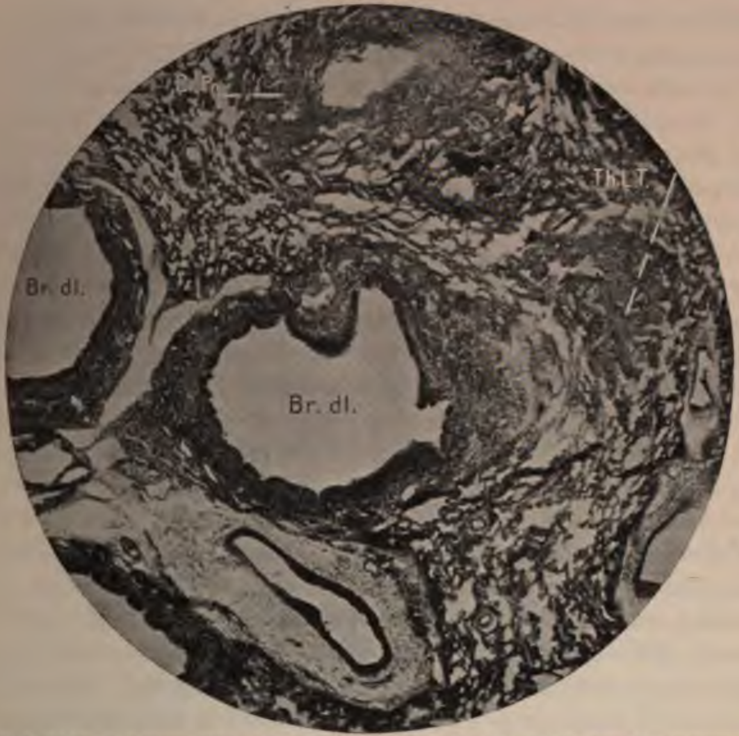
You will notice the areas of consolidated lung (C. L. T., peribronchitis) around the bronchi, which are dilated (bronchiectasis). You will also observe that there are areas of normal lung-tissue (N. L. T.).

Under this second microscope (Fig. 139) is a section, also made by Dr. Northrup, taken from a lung with chronic broncho-pneumonia in which the process has advanced still further than in the other.

In the middle of the specimen you will see a dilated bronchus with a section of a blood-vessel just below it. There is considerable connective-tissue formation about both. Here you see that the process of a peribronchitic pneumonia has gone further than in the other specimen (Fig. 138), and that there is, in addition to the dilated bronchi with the surrounding cellular infiltration, a tendency to the formation of connective tissue in the interlobular septa. This is the form of chronic broncho-pneumonia which is sometimes called interstitial pneumonia, and is usually characterized by a long course and delayed recovery.

A frequent lesion which occurs in the course of broncho-pneumonia is *emphysema*. According to Northrup, it is usually vesicular and situated in the anterior portion of the upper lobes. It is due to the diminished amount of air-capacity, together with the violent introduction of air into the chest caused by dyspnœa and coughing. This distention of the air-vesicles is supposed usually to disappear with the subsidence of the lesion which is causing the emphysema. Emphysema, both of the vesicular and of the interstitial variety, most commonly occurs in the pneumonia which follows pertussis.

FIG. 139.



Chronic broncho-pneumonia. Br. dl., dilated bronchus; Th. L. T., thickened lung-tissue; Br. Pm., broncho-pneumonia.

The interstitial variety may exist in the form of superficial sacs formed by the rupture of air-vesicles beneath lifting the pleura, or it may extend between the lobules in V-shaped tracts from the anterior edge of the upper lobe even to the root of the lung.

SYMPTOMS.—The symptoms of broncho-pneumonia vary greatly, owing to the many different lesions which commonly occur in the disease and which by their greater or less severity make its course exceedingly irregular. In so many instances is the broncho-pneumonia secondary to some other disease that the symptoms are necessarily modified by those of the initial affection. Thus, where broncho-pneumonia arises in the course of diphtheria, the symptoms are often obscured by the severity of the general symptoms of the diphtheria. Where broncho-pneumonia is secondary to measles and to pertussis, although at times its onset is difficult to detect, yet, as a rule, the quick respirations, the marked and continuous rise of temperature, and the evident exacerbation in the severity of the pulmonary symptoms, usually permit a diagnosis to be made even before the physical signs have become prominent. Its onset, however, in measles is, as a rule, rapid, while in pertussis it is slow and insidious.

The group of symptoms which characterizes a broncho-pneumonia arising during the course of bronchitis is somewhat more definite. In place of the moderate temperature and the absence of signs of serious disease which are usually met with in the course of an ordinary bronchitis, when broncho-pneumonia supervenes the temperature rises, the pulse and respirations are quickened, the alæ nasi dilate, there is more or less cyanosis, the cough becomes more frequent and painful, and the general aspect of the patient is that of one suffering from an affection of a severe type.

The temperature in broncho-pneumonia varies greatly, according to the extent and severity of the lesions. Corresponding to the intensity of the pneumonic onset, or to the especial disease which it complicates, the temperature rises rapidly or slowly and insidiously. The most common course in mild cases with gradual onset and terminating in recovery is for the temperature to rise gradually to 39.4° or 40° C. (103° or 104° F.), then to have a morning remission of three or four degrees for a number of days, and then to fall irregularly by lysis. A crisis is very rare in broncho-pneumonia, but sometimes occurs. Although the remissions in the temperature during the active stage of the disease are often quite marked, yet, as a rule, the temperature does not at this time fall to the normal. This is of service in differentiating certain cases of broncho-pneumonia, as well as lobar pneumonia, from malaria. Occasionally the temperature is reversed, the highest point being reached in the morning. This is rare, and is of no especial significance. Where the temperature instead of remitting remains high and steadily rises, the disease, as a rule, soon terminates fatally. Instead of the continued high temperature which occurs so often in fatal cases, a low temperature of only a few degrees above normal is sometimes met with, usually where the vitality is low and the power of reaction slight. The duration of the heightened temperature is very variable in broncho-pneumonia, and may last for a number of days or for weeks without the necessary result of the grave lesions of a more chronic process.

The pulse and respiration, though quickened, vary according to the severity of the disease and also according to the degree of nervous excitement. This latter is a very important element to be considered in determining the gravity of their rate. The pulse is at times very rapid, 160–180, and even higher; it usually varies from 130 to 150 or 160; though regular and full at first, it becomes weak and sometimes irregular as the disease progresses, and is very apt to remain rapid even after the temperature has declined and convalescence has been established. The respirations may be quickened by an unusually high temperature, but depend mostly on the extent of the involvement of the alveoli. They vary from 50 to 80, but they may be even higher, and are accompanied by dilatation of the alæ nasi. The respiration often shows a pause after inspiration instead of after expiration, as occurs in normal respiration, and is usually accompanied by an expiratory moan.

This sign, however, is not characteristic of broncho-pneumonia, as it may occur in lobar pneumonia and in various affections where the circulation is interfered with and where respiration is painful. In like manner the dilatation of the alæ nasi may occur in any disease accompanied by a heightened temperature and nervous excitement. Temporary exacerbations and changes in the rhythm of respiration are quite common in broncho-pneumonia, and in some cases a Cheyne-Stokes type of respiration has been noticed. This sign is usually one of grave import. Recession of the epigastrium and of the intercostal spaces commonly occurs in broncho-pneumonia, and varies according to the severity of the pulmonary lesions. In infants painful respiration is shown by a frown rather than by crying, while in young children it is shown by their whimpering and suppressed cries.

The physical signs of broncho-pneumonia are almost entirely those of the accompanying bronchitis, but in typical cases they correspond to the various pathological lesions which I have just described. According as larger or smaller areas of the lung are involved, corresponding areas of dulness on percussion may be found, provided these areas are sufficiently extensive not to be masked by other resonant portions of the lungs. They can, as a rule, be detected best by very light percussion. These areas of dulness are usually bilateral and of somewhat varied extent, though, as I have already stated, an entire lobe may in rare instances be sufficiently involved by the broncho-pneumonic process to produce very extensive areas of dulness. Over the area of dulness bronchial respiration, and in some cases increased vocal resonance and fremitus, may be found. On auscultation moist râles of all sizes may be heard all over the lungs, or, as is more usual, in circumscribed areas.

A symptom which occurs quite commonly in broncho-pneumonia is cyanosis. This may not only arise from the interference with the oxygenation of the blood from the lesions involving the air-vesicles, but may also be produced by a temporary atelectasis of certain portions of the lungs. The cyanosis is often accompanied by attacks of dyspnœa. When these symptoms result from atelectasis, the temperature, as a rule, does not rise, but may even be somewhat reduced, and areas of dulness may be detected on percussion. During these paroxysms the skin is often cold and moist. When the cause of the atelectasis, whether it be obstruction by plugs of mucus or pus or temporary exhaustion of the contractile powers of certain portions of the lungs, has been removed, the cyanosis and dyspnœa pass away and the general symptoms improve. These symptoms may arise at various periods during the course of broncho-pneumonia, and unless the atelectasis passes off within a few days a fatal issue is very apt to result.

Well-marked physical signs, especially dulness on percussion, are usually found at the bases of both lungs behind, and also between the scapulæ and the vertebral column. The earliest changes, however, in percussion and auscultation are often first detected in the highest part of the axilla. These signs of consolidation are rarely found in the early days of the disease, when the bronchitic signs are usually all that can be detected. The physical signs are markedly modified when atelectasis or emphysema is present.

In cases which recover, resolution takes place slowly and the lung gradually returns to the normal condition. Great weakness and prostration often last for a long time. Relapses are quite common.

COMPLICATIONS.—Pleurisy of a light grade is not an uncommon complication of broncho-pneumonia. Abscess and gangrene sometimes, though very rarely, arise. A case of the latter occurred at the Boston Children's Hospital in the service of Dr. Morrill.

A very frequent and important complication of broncho-pneumonia is tuberculosis.

In certain cases of the fulminant type of broncho-pneumonia the post-mortem examinations show extensive deposits of miliary tubercle, which in these cases is the cause of the accompanying acute inflammation. This condition is called tubercular broncho-pneumonia.

A frequent, short, hacking, and painful cough is a constant symptom from the beginning of the disease, and even after resolution has taken place this may continue for a long period. Infants and young children, even up to the age of seven or eight years, have often not learned to expectorate, so that we cannot, as in adults, judge of the character of the sputum. When the sputum is seen it corresponds to the pathological exudation which I have just described when speaking of the pathology of the disease. Vomiting is at times met with, and diarrhœa is not uncommon. In certain cases disturbance of the gastro-enteric tract is present from the very beginning, and the intestinal disease is apparently as important a feature of the attack as the pulmonary part. As the attack progresses the child loses much in weight, the face often looks pinched, and at times during the height of the disease there is a certain amount of delirium, which in combination with other grave symptoms, such as uncontrollable diarrhœa and a depressed temperature, is a serious symptom.

DIAGNOSIS.—The diagnosis of broncho-pneumonia should first be made from the bronchitis which ordinarily accompanies it. This has already been sufficiently referred to in speaking of the diagnosis of bronchitis.

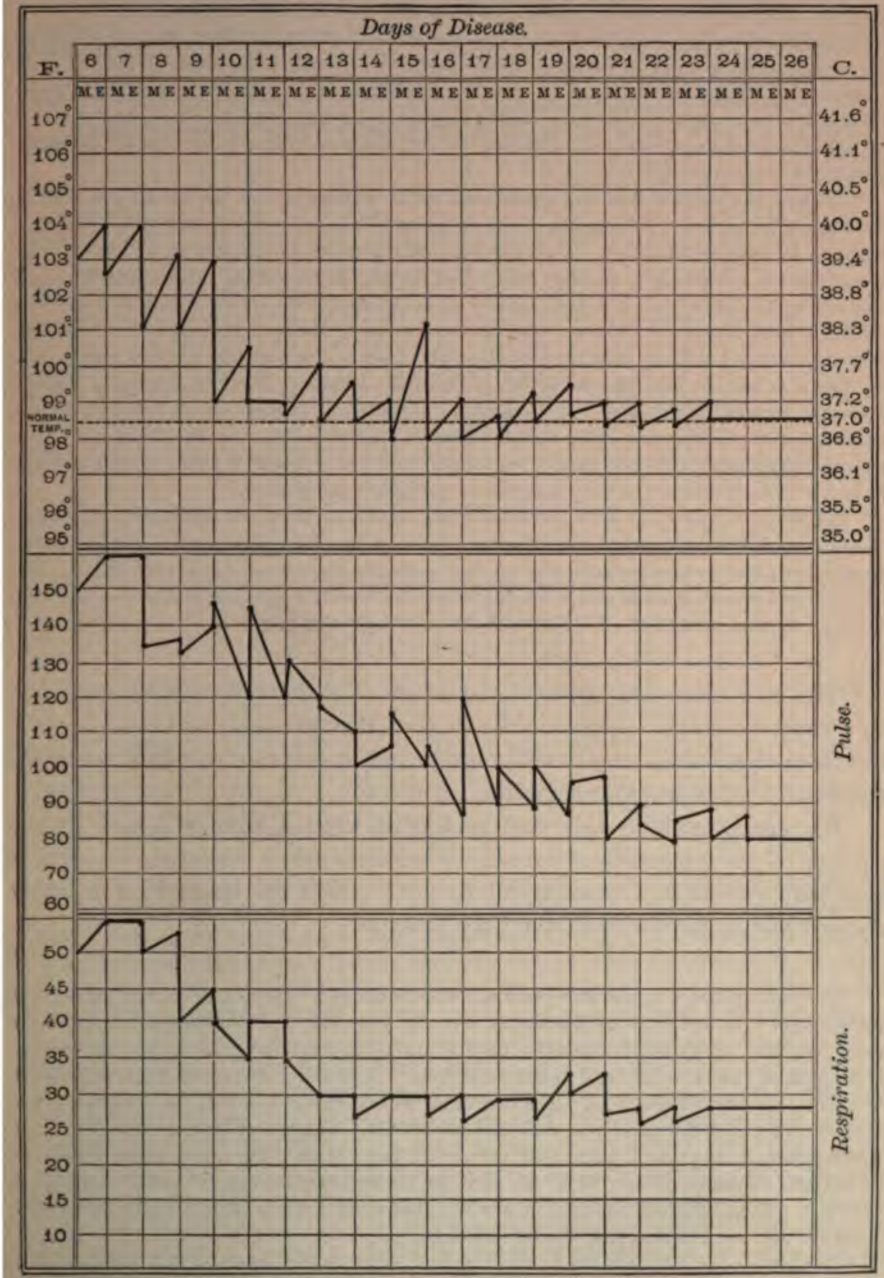
The differential diagnosis between the non-tubercular and the tubercular forms of broncho-pneumonia is important, but can rarely be made in the early stages of the disease, as the lesions are the same, and a bacteriological examination of the sputum in these cases can seldom be obtained.

The disease which should be especially considered in making the diagnosis of broncho-pneumonia is lobar pneumonia. The two diseases are perfectly distinct, in onset, course, duration, and termination, and can best be described when I speak of the diagnosis of lobar pneumonia (page 985).

PROGNOSIS.—Age is a very important factor in the prognosis of broncho-pneumonia. As Morrill has shown by a carefully prepared table, a large majority of the fatal cases of broncho-pneumonia occurs in the first two years of life. The prognosis varies according to the disease in the course of which it occurs. It is most grave when it occurs in pertussis, especially in infants, and the younger the child the more fatal the disease. Next to pertussis, the gravity of the prognosis is greatest in measles and diphtheria. When it occurs in such diseases as rhachitis and tuberculosis, or where the individual has not been well cared for, the prognosis is also very unfavorable. I have already referred to the temperature as a prognostic sign in broncho-pneumonia. According to Holt's observations, the highest mortality occurs among the cases of shortest duration, and the disease is universally fatal when its duration is shorter than four days. After this early period of danger is passed the prognosis becomes much more favorable, the lowest death-rate in Holt's cases being met with in cases terminating in from eight to fourteen days. When the disease lasts for more than two weeks the chances of recovery are lessened every day that the temperature remains raised. The cases in which there is a very high temperature, 41.1° C. (106° F.), are usually fatal. Where the disease is protracted, death generally occurs from exhaustion.

TREATMENT.—The treatment of broncho-pneumonia is that of the special disease to which it is secondary. The strength should be carefully supported from the time that the disease is first detected until convalescence has been completely established. The patient should be carefully nursed, as the nursing is the most important part of the treatment of broncho-pneumonia and requires much judgment and intelligence. The atmosphere of the room should be equable, the temperature from 20° to 21.1° C. (68° to 70° F.), and especial attention should be paid to the ventilation. The heat and ventilation obtained from an open wood fire are especially valuable. As few drugs as possible should be given, since there is no drug which will cut short the disease, and most of the drugs commonly used in the treatment of pulmonary affections are, as a rule, of more harm than benefit in broncho-pneumonia. The vitality of infants and young children is so easily lessened by a disease so severe as broncho-pneumonia that the respiratory power is likewise quickly diminished, and we should avoid, except where they are especially needed, such drugs as opium. Ipecac in minute doses seems to facilitate the removal of the mucus. During severe paroxysms an atmosphere of steam or the administration of oxygen is indicated, according as the symptoms seem to be produced by a tenacious exudate or by unaerated lung-tissue. In cases where cyanosis and dyspnœa are urgent, if these depend upon mechanical obstruction with its resulting atelectasis, an emetic is occasionally demanded. In some cases, also, where much exhaustion arises from incessant coughing, small doses of tinctura opii camphorata may be used with caution, and discontinued as soon as possible. When the urgent symptoms are caused by the heightened temperature, much relief well up to nine days ago, when she began to complain of headache and pain in her chest. At that time she vomited, and two days later began to cough and to be rather somnolent.

CHART 33. (CASE 461.)



Acute broncho-pneumonia. Recovery in thirty days.

Her bowels were regular. On physical examination the child is found to be rhachitic, as shown by a rosary, enlarged epiphyses of the wrists and ankles, and marked bowing of the legs. On entering the hospital her pulse was 160, her respirations 60, and her temperature

39.4° C. (103° F.) in the morning and 40° C. (104° F.) in the evening. She seemed very sick, had considerable cough, but no expectoration; there was some dyspnœa, and at times she was somewhat cyanotic. On examining the chest the percussion was found to be resonant, but throughout both lungs there were moist râles. Nothing abnormal was detected on examining the heart and abdomen. She was treated with milk and brandy.

On the following day she was in about the same condition, and her pulse, respirations, and temperature were as on entering the hospital. In certain circumscribed areas in both backs slight dulness was detected on percussion, with moist râles around the edges of these areas.

On the third day the pulse had fallen to 136, the respirations to 40, and the temperature to 38.3° C. (101° F.).

To-day, the ninth day of the disease, the pulse is stronger and the child's condition is very much improved. The dyspnœa has disappeared almost entirely, there is no cyanosis, and she is more comfortable. On examining the front of the chest you notice that the resonance on percussion is normal, and I find no abnormal sounds on auscultation. On examining the back you will find certain circumscribed areas of dulness, the borders of which I have marked in black. One of these areas is between the edge of the scapula and the vertebral column, another is at the right base in the posterior axillary region, and another is at the left base just below the angle of the scapula. Over these areas of dulness bronchial respiration is heard. Just outside of the areas of dulness can be heard in limited areas moist râles of various sizes, which I have indicated by black dots. You will notice that the physical signs in this case of broncho-pneumonia correspond to the areas where the lesions of this disease are usually detected on physical examination.

(Subsequent history.) Five days later the child was found to have much improved. During the following ten days the abnormal signs in the chest disappeared, but the pulse, respirations, and temperature did not become permanently normal for a week later. The child, after remaining weak and debilitated for some weeks, was finally discharged from the hospital in good condition. The chart of this case is seen on page 975.

This case apparently arose in the course of a slight bronchitis occurring in a rhachitic child. I have told you that the prognosis of broncho-pneumonia in rhachitis is usually unfavorable, but in this case the child possessed sufficient vitality not to succumb to the disease.

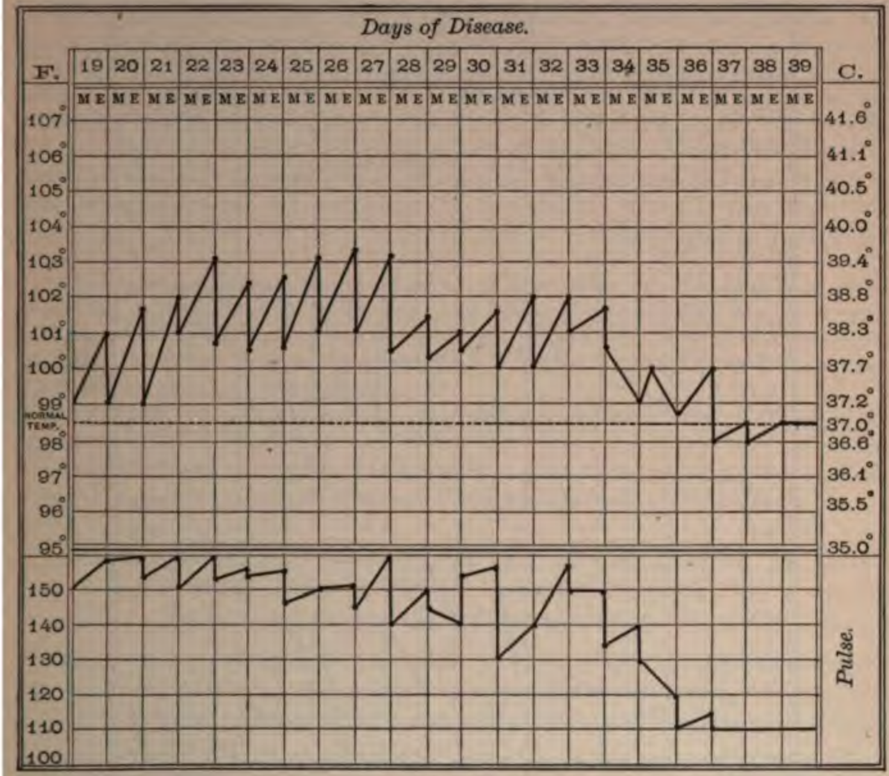
In connection with this case, and with what I have just said of the gravity of the prognosis of broncho-pneumonia when occurring in connection with rhachitis, I shall recall to your minds the case which I lately showed you in the wards of the City Hospital.

The child (Case 462) was two years and one month old. Its mother died of pulmonary tuberculosis. It had bronchitis when it was one year old, and the cough continued for three months. One week before entering the hospital it was attacked with a severe cough, and began to lose in weight and to have diarrhœa. A physical examination showed that it was a case of marked rhachitis. The breathing was rapid and labored, there was considerable cyanosis, and the child was dull and somnolent. Patches of dulness were found in various parts of the lungs, with moist râles of different sizes. The pulse varied from 140 to 150, the respirations from 80 to 90, and the temperature from 38.8° to 40° C. (102° to 104° F.). The symptoms increased in severity, the child grew weaker, and on the second day after it entered the hospital it died suddenly.

When broncho-pneumonia attacks a child with such marked rhachitis as was shown in this case, a fatal issue almost always results.

Here is a chart (Chart 34, page 977) showing the temperature and pulse of an infant (Case 463) eight months old, from the nineteenth day of an attack of broncho-pneumonia until convalescence was established.

CHART 34.



Acute broncho-pneumonia. Infant, 8 months old. Recovery in thirty-six days.

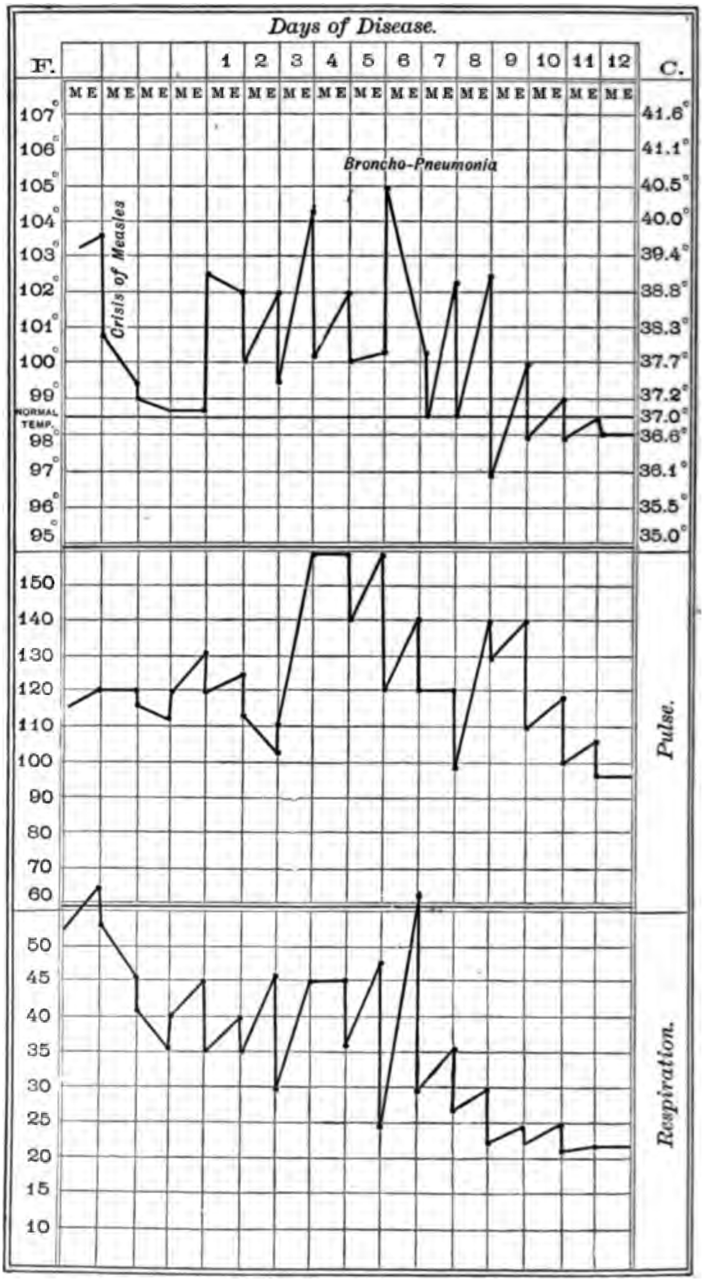
The infant, a male, had always been strong and well. The parents were healthy, but on the father's side a number of brothers and sisters had died of pulmonary tuberculosis. It was being nursed by its mother, who was strong and well. On December 16 the infant did not seem well, and on the following day, after having passed a restless night, bronchial respiration and râles were detected at the base of the right lung. The temperature on that day varied from 38.3° C. (101° F.) in the morning to 39.4° C. (103° F.) in the evening. There were no symptoms except a slight cough, and the respirations were accompanied by an expiratory moan. Until December 23 it took about a quart of milk in the twenty-four hours, but on the 24th it refused to take any food, and the temperature, which had been gradually coming down so as to reach almost 38.3° C. (101° F.) in the evening, began to rise, and dulness and fine râles were detected in the left lower back. The pulse at this time rose to 160, but was regular and strong; the respirations varied from 60 to 70. The alæ nasi showed more active dilatation, and there was slight twitching of the arms and hands. The cough became more frequent, and there was slight diarrhœa. These symptoms continued for several weeks, when they lessened in severity and the temperature fell to the normal.

The case was treated with brandy and digitalis, and finally recovered entirely.

When this child was eight years old he was attacked with purpura rheumatica, followed in a month by nephritis, and one month later by a severe attack of broncho-pneumonia, which involved extensive areas in both lungs and was accompanied by constant diarrhœa, delirium, cyanosis, and dyspnœa, resulting in death from exhaustion in the second week of the disease.

This next case, a girl, two years old (Case 464), had up to the present attack been strong and well. There is no tuberculous history in the family. She entered the hospital six days ago, and was placed in the isolating ward, as she was found to have measles. On entrance

CHART 85.



Broncho-pneumonia following crisis of measles. Child, 2 years old.

her temperature was 39.8° C. (103.7° F.), the pulse was 120, and the respirations were 62. An examination of the chest showed the heart to be normal. The percussion showed normal resonance and harsh puerile respiration, with numerous fine and coarse moist râles throughout both lungs. Nothing else abnormal was detected.

Five days ago the temperature fell to 38.1° C. (100.7° F.), four days ago to 37.3° C. (99.2° F.), and day before yesterday was just above normal. During these days the efflorescence rapidly faded, and she seemed better, though she occasionally had a harsh cough. Yesterday she had a slight rise of temperature, but there were no marked symptoms until to-day, when the temperature rose to 40° C. (104° F.), the pulse to 160, and the respirations to 50. She is, as you see, very restless, and has considerable dyspnœa. She is pallid and sometimes slightly cyanotic. On physical examination there is found diminished resonance over an area in the lower part of the left back. Over this area the breathing is bronchial, and there is also a number of moist râles. On the right side of the thorax, especially at the base of the lung, there are numerous coarse moist râles and harsh respiration, but no dulness.

This case illustrates the rapid development of a broncho-pneumonia during an attack of measles, occurring after the temperature produced by the measles had fallen to the normal and while the efflorescence was disappearing. The physical signs show the presence of small areas of consolidation in the left lung, and the usual diffuse bronchitis throughout the right lung and parts of the left lung.

(Subsequent history.) This chart (Chart 35) shows the course of the temperature, pulse, and respiration during the next twelve days. The pulse continued to be rapid and the respirations to be somewhat raised for some days after the temperature became normal. The abnormal signs in the chest disappeared, and the child made a rapid recovery.

Chronic Broncho-Pneumonia.—I have already described the pathological lesions which occur in chronic broncho-pneumonia. In a certain number of cases, after a child has had an attack of acute broncho-pneumonia the physical signs of consolidation may persist, although apparent recovery has occurred so far as the general symptoms are concerned. When this occurs the fever may return after a variable period, and the child, after having become still more emaciated, may die after a number of months of exhaustion. Instead of this fatal issue, the child, as has been shown by Delafield, may be left with a chronic form of the disease, which may last for many years and be accompanied by symptoms of cough, dyspnœa, and at times periods of fever. The most common termination of these cases is in acute general miliary tuberculosis. In certain cases, however, where only a small portion of the lung has been affected, the child may recover as it grows older. Broncho-pneumonia of a subacute or a chronic type is so apt to develop in the lungs of young children during the course of any disease of a prolonged nature, that frequent examinations of the lungs should be made, in order that the insidious development of these pulmonary lesions may not be overlooked.

The treatment of these chronic cases of broncho-pneumonia is essentially climatic. The child should be taken to a warm dry climate of high altitude, where it can live in the open air, and where it will not be subjected to frequent atmospheric changes.

ATELECTASIS.—Atelectasis is a collapsed and unaerated condition of the air-vesicles. It may be congenital or acquired.

Congenital atelectasis arises because the infant has not sufficient general vitality and respiratory power at birth to inflate fully all parts of its lungs. There may be an obstruction by mucus. There are in these cases areas of uninflated pulmonary vesicles of varying extent. These vesicles at the post-mortem examination can easily be artificially distended, and then cannot be distinguished from those which have been normally inflated.

The symptoms of congenital atelectasis are cyanosis, dyspnœa, rapid respiration, rapid, feeble, and often intermittent pulse, a temperature usually lowered, and dulness on percussion with lessened respiration over the atelectatic area. These are the typical physical signs of atelectasis, but in many cases some or all of these signs are absent and the areas of atelectasis are detected only at the post-mortem examination.

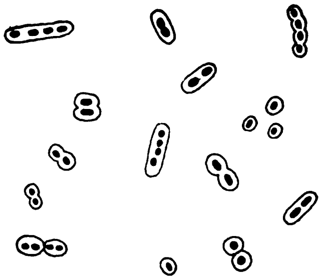
The prognosis in these cases varies according to the extent of the pulmonary tissue involved and the vitality of the infant. As a rule, the prognosis is very unfavorable.

The treatment of atelectasis is to stimulate the infant, and to endeavor to raise its temperature by means of a warm pack. In a number of cases I have found the administration of small quantities of oxygen to be of benefit. Artificial inflation of the air-vesicles has not proved to be an especially valuable form of treatment.

Acquired atelectasis is a symptom of some other disease, and I have already spoken of it sufficiently in connection with what I have said concerning broncho-pneumonia. Acquired atelectasis undetected during life is frequently found at the post-mortem examination of infants and young children dying of almost any disease.

LOBAR PNEUMONIA.—Lobar pneumonia is an acute self-limited disease of the lung, running a definite course and caused by the diplococcus pneumoniæ.

FIG. 140.



Diplococcus pneumoniæ.

ETIOLOGY.—Although lobar pneumonia may occur at any age, it is not met with so commonly in infancy and in early life as broncho-pneumonia. Exposure to cold, and especially to sudden atmospheric changes, apparently renders the individual more susceptible to the invasion of the micro-organism which causes this disease. Here is a specimen (Fig. 140) of this organism, taken from the sputum of a case of lobar pneumonia. It shows the morphology.

According to Delafield and Prudden, these germs during their development are distinctly spheroidal, but in their mature condition they often become slightly elongated and a little broader at one end than at the other, which gives them a lanceolate form. They are very apt to occur in pairs, and are frequently seen in short chains, but rarely in long chains. Very frequently when growing in the living animal the pneumococcus is surrounded by a distinct homogeneous capsule of varying thickness. The coccus itself is readily stained; the capsule is stained with difficulty.

PATHOLOGY.—The pathological condition which occurs in acute lobar pneumonia is an acute exudative inflammation which involves progressively the whole of one lobe, or the larger part of one lung, or portions of both lungs. There is no especial distinction between the lesions of lobar pneumonia as they occur in children and those which are met with in adults, except so far as the anatomical conditions differ according to the age of the individual. The stages of congestion, red hepatization, gray hepatization, and resolution take place in succession in the pneumonia of the child as in that of the adult. In the stage of congestion the lung is hyperæmic and œdematous and the air-vesicles contain fibrin, pus, granular matter, red blood-cells, and epithelial cells. The epithelium of the air-vesicles is swollen, and there are large numbers of white blood-cells in the capillaries. The large bronchi are congested. The small bronchi contain the same inflammatory products as do the air-vesicles. This stage lasts only a few hours, as a rule, but may be protracted for several days. When the exudation of the inflammatory products has reached its full development the presence of these products within the air-vesicles and bronchi causes the lung to be slightly enlarged, and at this time it is said to be in the condition of red hepatization. After the air-vesicles have become completely filled with exudation there follows a period during which the exudation first becomes decolorized and then degenerated. This is the period of gray hepatization. This happens at a variable time, which is usually shorter in children than in adults. The color finally becomes gray. The exudate then undergoes still further degeneration and softening, and is removed by the lymphatics. This is the stage of resolution. Resolution should begin immediately after defervescence and be completed within a few days, but it may not begin until a number of days after defervescence, and may be unusually protracted.

The bronchi are almost always affected in lobar pneumonia. The pneumonic process may occur in small patches, but usually involves an entire lobe. The lower lobes are the ones which are most frequently affected in early life, but the locality of the pneumonia is of pathological rather than of clinical importance, as the disease may attack any part of the lungs. It is generally a unilateral disease, but in some cases it may be bilateral.

SYMPTOMS.—The onset of acute lobar pneumonia is, as a rule, sudden, and in the infant or young child is frequently accompanied by vomiting and sometimes by convulsions; the latter, however, is uncommon as an initial symptom after the period of infancy. An initial rigor is uncommon. Pain is probably present, but cannot usually be located by the child with the same precision as by the adult, young children often referring the pain to the abdomen. Cough is a common symptom, not only in the beginning of the disease but also during its whole course, and often seems to be painful. It may, however, be absent for several days in the beginning of the attack. There is rarely any expectoration before the seventh or eighth year. In some cases during the height of the disease there is delirium. In the milder cases the delirium may be merely a slight wandering, but in the more severe cases the children may become much excited, and the delirium may be accompanied by contracted or dilated pupils, and even involuntary passages of urine and of fæces, with continual movement of the head, muscular twitchings, and other symptoms which may simulate closely those of cerebro-spinal meningitis. In place of the delirium and the excited condition there may be a condition of stupor which sometimes simulates the stupor of tubercular meningitis. In another set of cases the nervous symptoms markedly simulate those of the non-tubercular form of meningitis. Meningitis in any form, however, rarely occurs in the course of pneumonia. Violence of the symptoms is not common. Marked cerebral symptoms seem to depend more on the height of the temperature and the extent of the lung involved than on any especial part of the lung being affected, such as the apices. In infancy and in the early years of childhood, in place of these cerebral symptoms there may be simply an apathetic condition during the height of the disease, and the infant, although somewhat somnolent and restless, often shows no other nervous excitement. The course of the disease is usually shorter in young than in older children.

The rate of both the pulse and the respiration is increased, but the greatest increase is shown in the rate of the respiration. This is much higher *proportionately* to that of the pulse than is usually the case in other affections which would be likely to simulate lobar pneumonia. There is commonly dilatation of the alæ nasi, and it is seldom that this symptom is absent in pneumonia. The pulse varies from many causes, among which is the nervous condition of the especial child. It may be 120 to 150.

The temperature is a very important symptom in acute lobar pneumonia, and is almost diagnostic of the disease. In the initial stage it rises at once to 39.4° or 40° C. (103° or 104° F.); it remains high, with slight remissions of about two degrees in the morning, for a number of days, and then in a large number of cases falls to the normal within twenty-four hours by crisis. The time when the temperature falls and the crisis takes place varies. It may occur as early as the third or fourth day, but is usually between the fifth and the eighth day. It may, however, be delayed until the ninth or tenth day, and in rare cases still longer. When the temperature falls at the crisis of the disease it is very apt to be subnormal, and to remain so for a number of days. Sometimes after the temperature has fallen to the normal it may rise again, but, as a rule, another rise of temperature points towards the involvement of some fresh area of the lung or to some complication.

such as pleurisy. The fall of temperature at the time of the crisis is often accompanied by symptoms of great prostration and even collapse, and it is therefore important in young children to watch carefully for the crisis and to be prepared to combat these symptoms. The normal height of the temperature in acute lobar pneumonia, according to the extensive observations of Holt, is from 40° to 40.5° C. (104° to 105° F.). In children over three years of age the temperature curve resembles the adult type in being regular and falling by crisis, while under three years of age the proportion of typical cases is much less, and there is more irregularity in the course of the temperature, which may fall by lysis. The younger the individual the more likelihood there is to be a wide fluctuation in the range of the temperature, which has a tendency to be of the remittent type even in uncomplicated cases.

The physical signs of lobar pneumonia are the same as occur in adults. There is dulness on percussion over the affected area of the lung where consolidation has taken place, with bronchial respiration, increased vocal fremitus, and increased vocal resonance. In the initial stage of the disease fine râles are heard at times, but not so commonly in children as in adults. When resolution is taking place, moist râles of all sizes are heard. These are the typical signs of lobar pneumonia. In some cases the physical signs are entirely absent for a number of days, and the diagnosis has to rest upon the heightened temperature, the increased respirations and pulse, and the dilatation of the alæ nasi. In the early days of the disease the cough may be absent; this renders the diagnosis still more difficult. The cough may continue and the physical signs remain unchanged for a number of days after the temperature has fallen. The physical signs in some cases immediately disappear when the temperature becomes normal. Fine dry râles are not heard so frequently in the lobar pneumonia of young children as in that of adults.

In certain cases, where hepatization of the lung has taken place in the usual way and the crisis has come with a fall of temperature, resolution will fail to take place and the lung will remain solidified sometimes for a long period. Although an infection by the bacillus tuberculosis may be suspected in many of these cases, from their protracted course and from the prostration which usually accompanies them, yet such infection does not necessarily take place, and resolution often finally occurs. In these cases the lung is left apparently in the same normal condition as if this variation in the resolution had not taken place.

As an illustration of delayed resolution in lobar pneumonia I shall report to you the case of a little girl (Case 465), four years old, who, when she was perfectly well and strong, was suddenly attacked with vomiting, pain in the right side, and cough accompanied, according to her mother, by a reddish-brown sputum. Physical examination on the following day revealed nothing abnormal except a few fine moist râles at the base of the right lung behind. The pulse was 170, the respirations 60, and the temperature 39.4° C. (103° F.). On the following day the temperature still remained raised, and there was dulness on percussion over the lower right lobe behind, with bronchial respiration. On the following day the dul-

DIAGNOSIS.—The diagnosis of lobar pneumonia, when the typical temperature and the characteristic physical signs are present, is not difficult, but there are a number of atypical cases in which a doubt might easily arise for a number of days after the invasion of the disease. An early diagnosis from a pleuritic effusion and from other pulmonary affections is at times impossible.

The differential diagnosis between lobar pneumonia and a pleuritic effusion may be quite difficult in the early stages before the characteristic areas of dulness have been established. In both diseases dulness over a limited area, and bronchial respiration without any especial difference in the vocal fremitus and vocal resonance, and without evidence of a friction-rub or of râles, may make the two diseases simulate each other closely and compel us to wait for further developments before determining which disease is present.

From tubercular disease of the lung the differential diagnosis is usually not difficult, except in young infants, in whom the tubercular process with its corresponding symptoms may in rare cases simulate lobar pneumonia.

The disease from which a differential diagnosis should especially be made is broncho-pneumonia. Lobar pneumonia and broncho-pneumonia are so distinct, however, in their previous history, initial stage, course, and duration that, if care be taken to note closely all these stages of the two diseases and to arrive at a diagnosis from the evidence given by all the stages and not by any one stage, the diagnosis can, except in the very early days of the disease, usually be determined. Lobar pneumonia, in contradistinction from broncho-pneumonia, is a primary disease, characterized by a sudden onset and a regular temperature, the rise being sudden. This is accompanied by a corresponding rapidity of the pulse and respirations, dulness on percussion usually involving and limited to one lobe or one lung, with increased vocal fremitus and resonance, and bronchial respiration over the dull area. This is followed by a fall of temperature and by a rapid resolution. The duration is short and definite. Broncho-pneumonia, on the other hand, is usually secondary to a preceding bronchitis, occurring either alone or in the course of some other disease. It is characterized by a slow and insidious onset, except when occurring in the course of measles; it has an irregular temperature, the rise usually not being so sudden or so high as in lobar pneumonia, and the respirations and pulse slowly rising with the temperature. There is often an absence of change in percussion, the dulness if present showing itself in small patches and commonly in both lungs. There is also often an absence of marked vocal fremitus or vocal resonance, and of bronchial respiration, except where the patches of dulness are pronounced. Moist râles of all sizes may be heard in circumscribed areas throughout both lungs. The temperature is usually of a remittent type, and this condition lasts for weeks rather than days. The resolution is slow. The duration is often prolonged. If these pictures of the two diseases are borne in mind, an error in the differential diagnosis will seldom be made. In the doubtful cases, where the characteristic course of either disease is absent, it will usually be found that we are dealing with a case of broncho-pneumonia, which is an exceedingly variable disease, rather than with lobar pneumonia, in which some of the characteristic features of the disease are almost invariably present.

In making the diagnosis between pneumonia and meningitis it is of much aid to remember that the *slow* intermittent pulse, slow irregular respiration, and moderate temperature of meningitis are uncommon in lobar pneumonia, where in most cases the pulse is quick and regular, the respirations rapid, and the temperature high. It is not uncommon, however, to find irregularities and intermissions in the *rapid* pulse of pneumonia. The younger the individual, as I have already stated in my lecture on meningitis (page 612), the more likely are the symptoms of tubercular meningitis to be replaced by those of the non-tubercular form of the disease, which may often simulate closely the symptoms of pneumonia. The convulsions which occur in pneumonia do not differ from those which occur in meningitis or, in fact, in any other acute disease. A careful physical examination should be made at every visit, once or twice a day if possible, as in this way the masked symptoms of a pneumonia may at times be detected where they would be overlooked if only an occasional examination were made.

After the first four or five days, as a rule, the differential diagnosis between cerebral disease and pneumonia is not difficult.

COMPLICATIONS.—The complications of acute lobar pneumonia are not very numerous. At times a pericarditis may occur, with its resulting effusion, but these cases are rare. The most common complication is a pleuritic effusion, which, especially in young infants, is apt to be purulent. In many cases the onset of the disease and its initial symptoms are apparently characteristic of pneumonia, and yet a few days later it becomes evident that a purulent pleuritic effusion has either complicated the pneumonia or was the original disease, simulating in its symptoms the early stage of lobar pneumonia. Lobar pneumonia is at times a serious complication of other diseases, and adds materially to their gravity.

GANGRENE.—One of the rare complications of lobar pneumonia is gangrene of the lung. This lesion is never found as a primary disease, and is rare in children. It is usually met with in weak, debilitated children whose circulation is impaired.

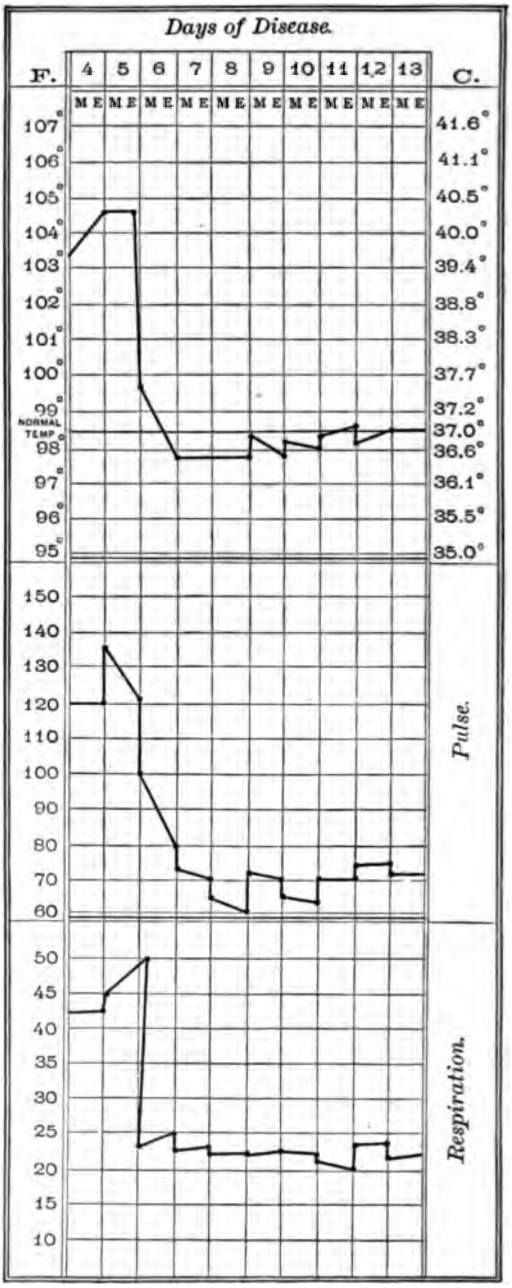
PROGNOSIS.—The prognosis of lobar pneumonia is very favorable. In young infants, or in those who are weak and debilitated, it is often fatal, but in comparison with broncho-pneumonia the percentage of recovery is very high. When the temperature rises to 41.1° C. (106° F.) the prognosis is usually grave. The convulsions which occur in the initial stage of the disease in infants are commonly not of grave import. Occurring late in the disease they make the prognosis very unfavorable. When delirium occurs, although it may be severe, it does not render the prognosis especially unvorable. The fulminant type of the disease which sometimes occurs is a ery fatal form.

TREATMENT.—As lobar pneumonia is a self-limited disease of short uration, the children are not so apt to die of exhaustion, and as a rule nly an expectant treatment is called for. Where the disease occurs in very oung infants it is safer to administer stimulants from the beginning. In hildren, however, it is often not necessary to use any drug whatever, and is safer to wait until there are indications that the disease will not run benign course before using drugs. Such indications are especially given y the temperature. Although at times a high temperature does not neces- arily indicate danger, since a temperature of from 40° to 40.5° C. (104° 105° F.) is part of the regular course of the disease, yet if the tempera- re rises above this point it is well to reduce it by means of bathing and to ive stimulants in the form of brandy. The child should be placed in a oom of an equable temperature of 20° or 21.1° C. (68° or 70° F.), and hould be given milk every two hours. There is no necessity for making ny external applications to the chest. The use of poultices is to be depre- ated, and in my experience is usually without benefit except in certain nstances for the relief of pain. The nursing is of especial importance, nd close watchfulness, especially at the time when the crisis is expected take place. At this time the temperature in infants and young chil- ren may fall with such rapidity to several degrees below the normal oint that collapse often takes place, the skin is cold and moist, and some- mes the child becomes unconscious. Under these circumstances the pulse feeble and intermittent, and in some cases death may occur unless active easures are taken for establishing reaction. The nurse should therefore e warned as the time for the expected crisis approaches to watch the hild both night and day, and to have remedies ready to be used in case rious symptoms should arise. These remedies should be the external pplication of heat by means of the hot pack, and the administration of randy by the mouth if the child can swallow, otherwise by rectal injection. have known of a case (Case 468) where a child died in the collapse follow- g the crisis of a lobar pneumonia. During the convalescence from pneu- onia the child should be protected from atmospheric changes, cold, and ampness for some time.

Lobar pneumonia may occur in the earliest days of life. I have met ith a case (Case 469) which on the third day of its life developed a lobar neumonia and died in twenty-four hours. The autopsy was made by Dr. . F. Whitney, and the characteristic hepatization was found.

Here is a boy (Case 470), eight years old, who entered the hospital on the fourth day of attack of lobar pneumonia. The attack began with vomiting and cough, but no pain, pectoration, or chill. An examination showed the right lung to be normal. On the left le of the chest an area corresponding to the lower lobe in the back was found to show solute dulness on percussion, bronchial respiration, and many fine moist râles. Nothing e abnormal was detected. The child was very restless, but on the following day, the fifth from the onset of the disease, the temperature fell by crisis to the normal point. This chart (Chart 36) shows the typical temperature, pulse, and respiration of a case of lobar pneumonia.

CHART 36. (Case 470.)

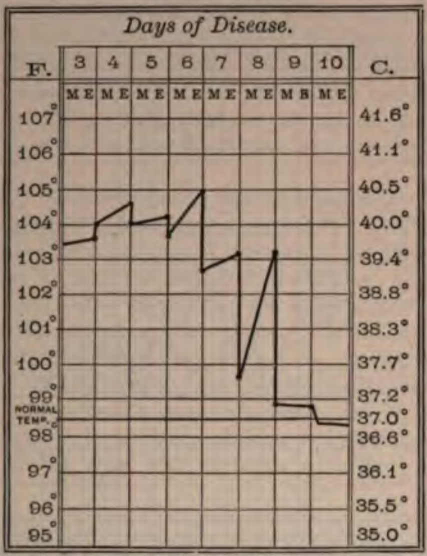


Lobar pneumonia. Male, 8 years old. Crisis on fifth day of disease.

(Subsequent history.) After the crisis the child improved rapidly, and the physical signs disappeared in ten days.

This little girl (Case 471), two and a half years old, entered the hospital on the third of an attack of lobar pneumonia. To-day is the tenth day from the onset of the dis-, and this chart (Chart 37) shows one of the variations in the crisis which is quite fre-ntly met with in young children.

CHART 37.



Lobar pneumonia. Irregular crisis on eighth day. Female, 2½ years old.

In this case the consolidated portion of the lung was the left lower lobe. The resolution rapid and convalescence normal.

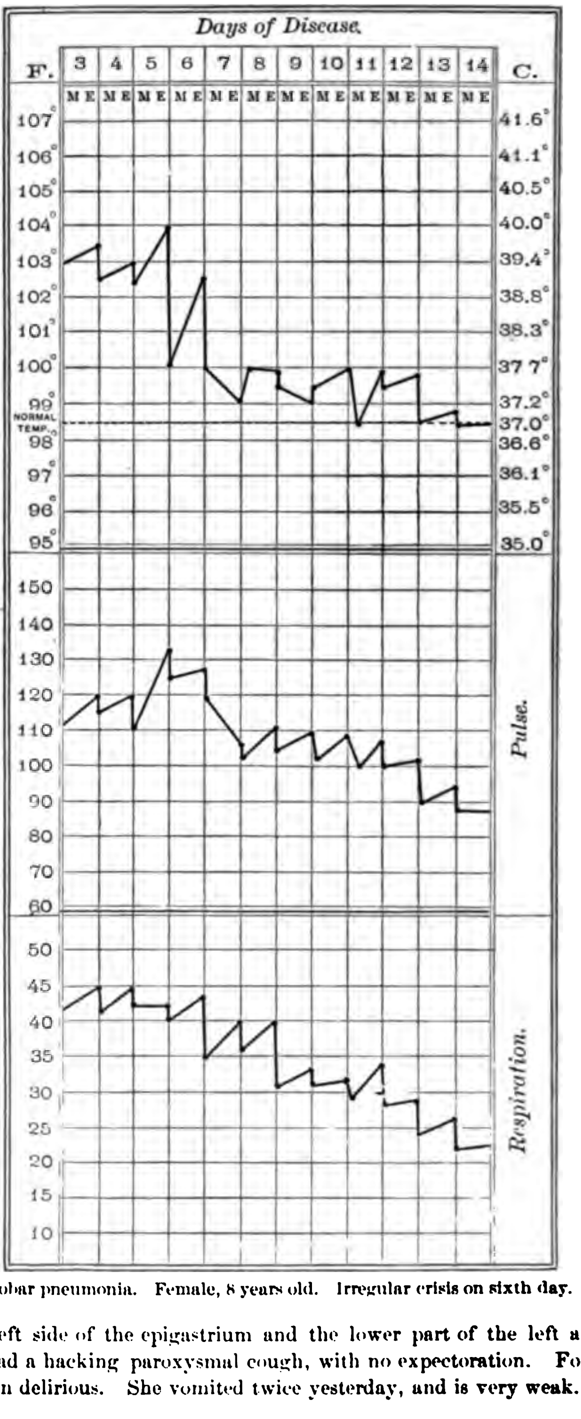
This little girl (Case 472), eight years old, had pertussis when she was fourteen months

CASE 472.

Lobar pneumonia. Female, 8 years old. The part of the lung involved by the pneumonic process own by black lines, and the area of diminished resonance and the fine râles are marked by black s.

old, scarlet fever when she was five years old, and measles when she was six years old. Five days ago she lost her appetite, was very feverish, and was attacked with acute pain referred to the left side of the epigastrium and the lower part of the left axillary region. She has since had a hacking paroxysmal cough, with no expectoration. For the past few days she has been delirious. She vomited twice yesterday, and is very weak. Her tongue

CHART 38. (CASE 472.)



Lobar pneumonia. Female, 8 years old. Irregular crisis on sixth day.

see, is heavily coated, the alæ nasi are working, her face is deeply flushed, and she spnœa to such an extent that she has to be propped up on pillows.

er respirations are 45, difficult and painful, her pulse 120, and her temperature 39.5° 3.2° F.). A physical examination detects nothing abnormal in the front of the chest he right back. There is absolute dulness in the left back, beginning at the fifth rib tending to the base of the lung and into the axillary region. Over this area of dul- iere is increased vocal fremitus and bronchial respiration. In this area, also, there ew moist râles Just above the upper border of the area of absolute dulness there ninished resonance and a number of fine râles. This is the fifth day of the disease. igh the general condition of the child seems to show no especial change, yet the al signs show that resolution has begun and that we may at any time expect the o occur.

ubsequent history.) On the following day the temperature fell to 37.7° C. (100° F.) morning, but rose again in the evening to 39.1° C. (102.5° F.). On the following ie seventh day from the beginning of the attack, the temperature fell to 37.2° C. .), and then varied from 37.7° C. (100° F.) to 37.2° C. (99° F.) until the eleventh hen it became normal. The chart (Chart 38) shows the pulse and respiration up to irteenth day from the beginning of the attack.

his case is one which illustrates the fact that the physical signs of resolution may mes appear before the temperature falls and the crisis comes; also that at the time crisis the temperature may fall, then rise again for twelve to twenty-four hours, and ll to the normal, as in this case. The child recovered completely.

his boy (Case 473), six years old, was taken sick four days before entering the ıl.

CASE 473.

Lobar pneumonia. Three invasions. Male, 6 years old.

ı entering the hospital his pulse was 128, his respirations 60, and his temperature C. (103.8° F.). A physical examination showed that there was absolute dulness ie entire upper lobe of the right lung. Over this area of dulness there were bron- espiration and increased vocal resonance. There was also an occasional high-pitched lobe of the right lung was involved in front, as I have indicated by this second black line below the one which I have just described. The temperature during the next two days remained between 39.4° and 40° C. (103° to 104° F.), but on the following day, the ninth from the onset of the disease, the temperature suddenly fell to 37.6° C. (99.7° F.) in the evening, but rose the next morning to 39.3° C. (102.8° F.), and in the evening rose to 39.9° C. (103.8° F.). A physical examination then showed that the whole of the lower lobe was involved, as I have indicated by the third black line. On the following day the upper lobe began to show evidence of resolution, and the temperature fell to 38.3° C. (101° F.). Two days later the temperature began to fall by lysis, the physical signs of the upper and middle lobes entirely disappeared, and the temperature reached the normal point on the fourteenth day from the time of the onset. On the seventeenth day from the time of the onset the lower lobe was also found to be in a normal condition, and from that time convalescence was uninterrupted.

Here is the chart (Chart 39), which shows the temperature, pulse, and respirations in this case from the fifth to the seventeenth day of the disease.

In some rare cases the infection in lobar pneumonia is so intense that a rapidly fatal issue may occur.

I have seen a little girl (Case 474), nineteen months old, who had been having so mild an attack of diarrhœa that she was playing about out of doors, suddenly attacked in the afternoon with convulsions and a temperature of 40.5° C. (105° F.). The convulsions continued during the night, and she soon became comatose. On the following day the temperature still remained at 40.5° C. (105° F.), the respirations were much accelerated, and the pulse was about 120. An area of absolute dulness over the left lower lobe behind, with bronchial respiration and increased vocal resonance and fremitus, rapidly developed. The child did not respond to treatment, and died in the evening.

TUBERCULOSIS OF THE LUNG.—Tuberculosis of the lung is an affection in which certain lesions are produced in the lung by the bacillus tuberculosis. Although this tubercular affection may attack any organ or any part of the body, yet whenever it occurs elsewhere it is almost invariably found in the lung. It is well, therefore, to speak of this especial manifestation of tuberculosis in connection with diseases of the lung.

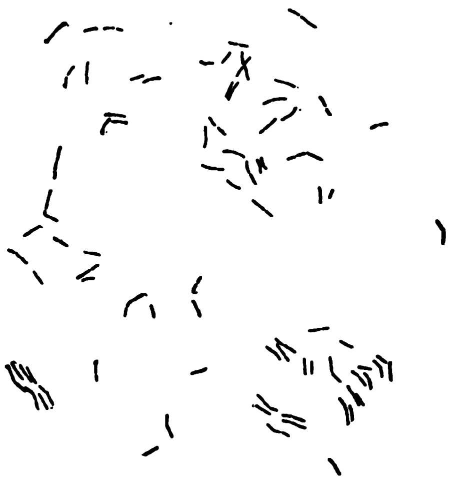
ETIOLOGY.—The cause of tuberculosis, as I have just stated, is an organism, the bacillus tuberculosis. Here is a specimen (Fig. 141, page 994) which shows the morphology of this organism.

These organisms are, according to Delafield and Prudden, slender, filamentous bacteria varying in length from one-quarter to one-half the diameter of a red blood-cell. They are frequently curved and bent, and may form short chains. This bacillus may retain its vitality for many weeks in a dried condition, but is killed by an exposure of fifteen minutes to a temperature of 100° C. (212° F.). In most cases it finds its way to the tissues by inspiration, although it may also gain access to the body by being swallowed.

PATHOLOGY.—The pathological conditions which result from infection by the bacillus tuberculosis are very numerous. The lesions in the child do not differ from those which occur in later life, and I shall therefore not describe them in detail.

The ordinary chronic tubercular lesions met with in adults are seldom seen in children, and it is rare for the tubercular process in children to begin at the apices of the lungs and gradually extend downward, as is common in adults. When this occurs, it is usually in the later years of childhood, when the conditions are beginning to approximate those of later life. The most frequent entrance of the tubercular affection to the lung is through the bronchial glands. This fact has been especially studied and described by Northrup. According to this investigator, in most cases the infection of tuberculosis in children is effected by the entrance of the tubercle-bacilli into the respiratory passages with the inspired air, and the lodging of them in the mucus of the air-passages or the alveoli of the lungs. They may then pass through at any point, and, being taken into the lymph-spaces, traverse the lymph-canals to the nearest glands and be retained there. These glands at the base of the lung receive and filter everything brought to them from the bronchial tract. The subsequent career of the bacilli depends upon the power of the tissues to withstand their further progress. They may die, or may remain inactive for a long period and later develop a tubercular process in the glands. These tubercular glands may finally break down and thus allow the bacilli to penetrate different portions of the lungs and produce their characteristic lesions.

FIG. 141.



Tubercle-bacilli taken from the sputum in a case of tuberculosis of the lung.

Tuberculosis of the lungs may occur in two forms: (1) *acute tubercular broncho-pneumonia*, and (2) *chronic tuberculosis of the lungs.*

Acute Tubercular Broncho-Pneumonia.—According to Osler, acute tubercular broncho-pneumonia is common in children from the sixth month to the fifth year, a large proportion of the cases, however, occurring after the second year. It is common in children who have been debilitated by previous illnesses, and occurs especially after measles, pertussis, scarlet fever, and diphtheria, being most frequent in the first two. It may, however, develop in perfectly healthy, well-nourished children, and also, as Osler has expressed it, may be a terminal process in cases in which local tubercular disease exists in other parts, such as the skin, bones, lymph-glands, or the uro-genital tract.

As in the other forms of broncho-pneumonia, the initial lesion is a bronchitis and peribronchitis, the distinguishing tubercular features being caseation and necrosis of the consolidation with the presence of the tubercle-bacilli. The accompanying phenomena of atelectasis and emphysema occur as they do in non-tubercular broncho-pneumonia. In some cases the non-tubercular broncho-pneumonia precedes the tubercular disease, this occurring particularly after measles, scarlet fever, diphtheria, and pertussis. According to Mosny, where the tubercular broncho-pneumonia follows the non-tubercular form, in addition to the lesions of the latter disease, there are found true tubercular processes, such as peribronchial nodules, tubercular infiltration, and caseous areas. Where the patient is the subject of a latent tuberculosis, such as may follow one of the infectious diseases, a non-tubercular broncho-pneumonia may also develop. In these instances, according to Mosny, the lesions may be seen surrounding the tubercular peribronchitic nodules, or foci of non-tubercular or tubercular broncho-pneumonia are found scattered through the apices of the lung.

SYMPTOMS.—The symptoms of acute tubercular broncho pneumonia are very similar to those of non-tubercular broncho-pneumonia. According to Osler, in most cases the onset of the disease simulates that of the ordinary non-tubercular broncho-pneumonia so closely that a differential diagnosis between the two diseases cannot be made until after death, and even then the post-mortem appearances may not be those distinctive of tubercular disease, and the pathological diagnosis can be determined only by finding the bacillus tuberculosis. The children may be attacked with cough, a heightened temperature, and the physical signs of broncho-pneumonia. The physical signs, as would naturally be expected, are usually found in the back and lower portion of the lung rather than at the apices, as in adults, on account of the usual nidus of the tubercular lesions,—namely, the bronchial lymph-glands. In some cases the onset of the disease is not so acute, and its course not so rapid. The child emaciates and has only a moderate fever, but later the development of such symptoms as sweating, chills, and hectic, together with the signs of softening and breaking down of the lung-tissue, leads us to suspect that we are dealing with tuberculosis of the lung.

DIAGNOSIS.—The diagnosis, as a rule, is to be made by taking into consideration the family history of the child, as the tissues of children whose parents are tubercular show an especial liability to infection by the bacillus tuberculosis. The diagnosis can be made positively only in those cases where a specimen of the sputum can be obtained and examined for the bacillus tuberculosis.

PROGNOSIS.—The prognosis is invariably unfavorable.

TREATMENT.—The treatment of tubercular broncho-pneumonia is the same as that of the non-tubercular form.

Chronic Tuberculosis of the Lungs.—Chronic tuberculosis of the lungs as it is ordinarily met with in adults is rarely seen in young children. During the first three months of life tubercular disease of any form is very rare, but in the latter part of the first year it becomes very common. The tubercular lesions which are found in the lungs in later life also occur in early life. Although cavities are not so commonly found in young children as in adults, it is not so much that they do not exist as that, their locality being more at the root and central portions of the lung, they are more difficult to detect on physical examination. It has been noticed that large cavities at the apex of the lung are rare in early life, but become more common as the child grows older. Tubercular disease of the lung is very irregular in the extension of its lesions in young children. Much more advanced lesions are usually found at the post-mortem examination than are detected during life. As I have already stated, the primary lesion of chronic tuberculosis of the lungs is commonly a tubercular broncho-pneumonia.

SYMPTOMS.—The symptoms of chronic tuberculosis of the lungs differ but little in the child from those seen in the adult, and are marked by the same irregularities in their course. This is due to the varied forms of the lesions. In young infants the symptoms are so often obscure and the physical signs of the serious pathological conditions which exist in the lungs are so frequently masked that the diagnosis is apt to be very doubtful. There is often a history of tuberculosis in the parents. The more common symptoms of chronic tuberculosis of the lungs are gradual loss in weight, strength, and appetite, irregular and moderate fever, hectic, and sweating. The physical signs are slowly increasing dulness in certain areas of the lung, especially in the back, accompanied by râles and other evidences of solidification. Later in the disease the characteristic signs of cavities may develop. Cough is usually present, though it is sometimes so slight in the beginning as not to be especially noticed by the parents. Hæmoptysis is rare in infants and in young children, but may be present in older children as they approach the age of puberty. As the disease progresses there is dyspnœa, usually of a moderate grade, with cyanosis, but in some cases considerable destruction may have taken place in the lung-tissue without the presence of any especial dyspnœa.

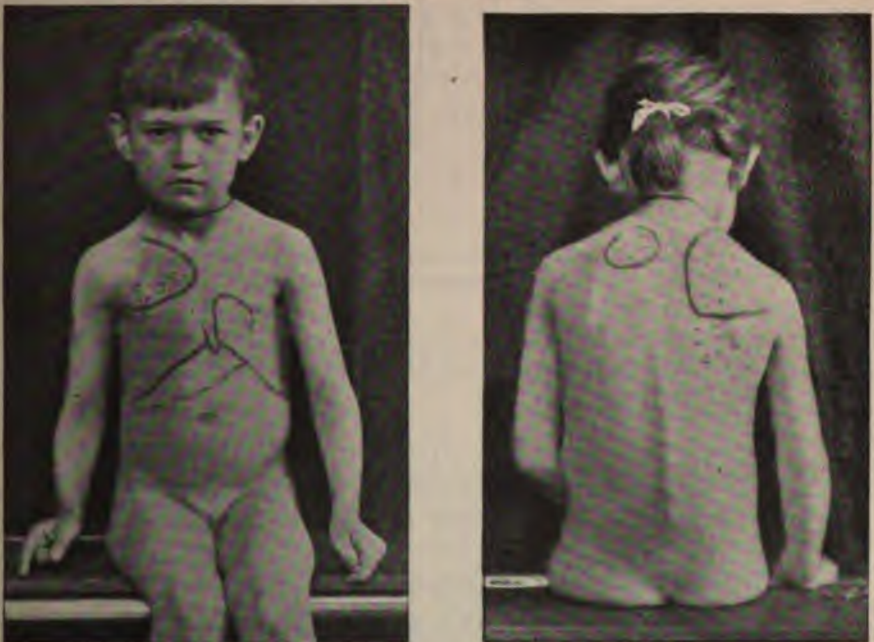
The course of chronic tuberculosis of the lungs is rather more rapid in children than in adults, and it is seldom that the long-protracted course of the disease so frequent in adults is met with in children. Sometimes, however, the child improves in its general health and may live for many years. In these cases the terminal phalanges of the fingers may become clubbed, and there is usually dyspnœa on exertion.

DIAGNOSIS.—The diagnosis is to be made from chronic empyema and from chronic non-tubercular broncho-pneumonia. The former disease can be readily eliminated by making an exploratory aspiration, but the latter can often be distinguished only by means of a bacteriological examination. In older children, where a specimen of the sputum can be obtained, the diagnosis is readily made by the detection of the bacillus tuberculosis. In younger children, in whom expectoration does not take place, the diagnosis is much more difficult, but if the children are carefully watched it is often possible to obtain a specimen of the sputum if the child happens to vomit, in which case particles of sputum may be coughed up with the vomitus and can be separated from it and examined.

Prognosis.—The prognosis of chronic tuberculosis of the lungs where the symptoms are at all advanced is very unfavorable, but the post-mortem examinations of so many individuals who have died of non-tubercular diseases show the presence of old tubercular lesions which have apparently ceased to be of grave import, that we must acknowledge that it is possible for many cases to survive the invasion of the bacillus tuberculosis.

Treatment.—The treatment of chronic tuberculosis of the lungs is essentially climatic, and the children should be removed at once, if possible, from a climate where the altitude is low and the atmosphere damp and subject to great variations. Too high altitudes are also to be avoided. Where the child cannot be removed to a more favorable climate, strict attention to its general hygiene and to its food will in some cases be followed by an apparent arrest of the tubercular process.

Case 475.



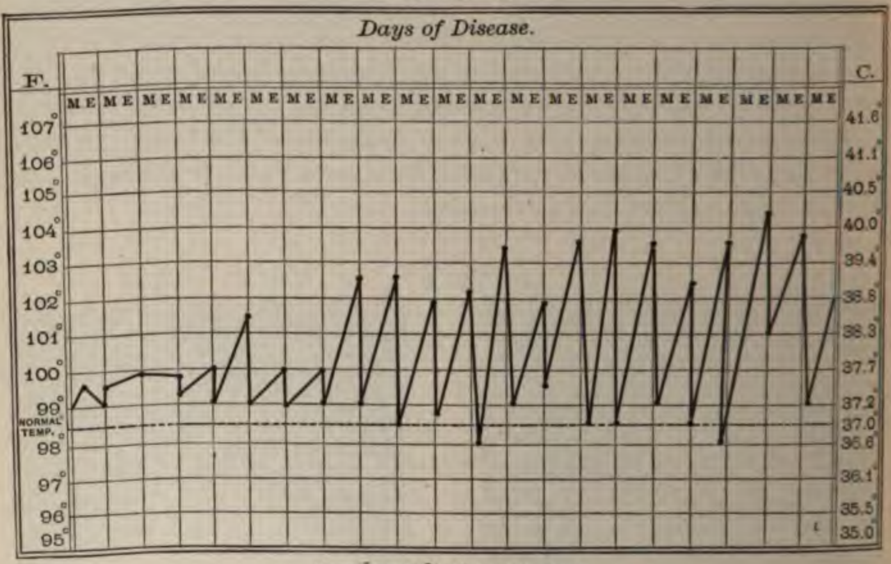
Chronic tuberculosis of the lung. Female, 8 years old.

This little girl (Case 475), eight years old, has a history of tuberculosis in her family. She had an attack of pertussis when she was six years old, and some months ago an attack of measles. Following the attack of measles she began to have headache, cough, and expectoration. She complained of pain in her chest and abdomen, and of chilly sensations, and has progressively lost in weight and strength. A physical examination shows the skin to be dry and harsh and the heart normal. The left lung in front appears to be normal. Behind over a small area at the upper part of the lung there are dulness, broncho-vesicular breathing, and some fine moist râles.

Over the right upper lobe in front and behind there is dulness, and the expiration is prolonged and high-pitched. Over the dull region are heard medium and fine moist râles. I have indicated the borders of the areas of dulness by black lines, and the râles by black spots. I have also shown the area of cardiac dulness, the lower part of the sternum, and the lower border of the ribs, by dark lines, and the edge of the liver, which seems to be somewhat enlarged, by an interrupted line.

This chart (Chart 40, Case 475) shows the irregular temperature which is commonly seen in cases of chronic tuberculosis of the lungs and is of a remittent type.

CHART 40.



Chronic tuberculosis of the lung.

The expectoration has not yet been examined for the bacillus tuberculosis, but the diagnosis is not doubtful, as the child is gradually failing and the signs of disorganization of the lung are slowly progressing.

Pertussis (Whooping-Cough).—Pertussis is a highly infectious disease affecting the respiratory tract and characterized by periods of spasmodic coughing, succeeded by a prolonged inspiration and accompanied by a peculiar sound called the "whoop."

The cause of pertussis is probably a micro-organism, but this organism has not yet been determined. It is supposed that the contagium can be carried by a third individual, but usually it is directly communicated from one person to another. This contagium is probably contained in the expectoration, and in this way houses where the disease is present may become a source of infection. The disease commonly occurs in epidemics during winter and spring. Sporadic cases occasionally appear, and in large cities the disease is often endemic. Pertussis seems to have some especial relation to measles, as children with the latter disease are liable to contract pertussis, and in like manner those with pertussis are liable to contract measles. Pertussis may occur at any age, and the disease has even been known to be contracted in utero. One attack usually protects from a second. Debilitated children with catarrh of the respiratory tract are more subject than others to the contagium of pertussis.

PATHOLOGY.—There are no pathological lesions distinctive of uncomplicated pertussis. The condition which characterizes the paroxysmal attacks is extreme congestion of the different organs, such as the meninges, the lungs, the heart, and the kidneys. In grave or fatal cases the lesions are those which arise either from mechanical accidents, as emphysema or hemorrhage in various parts, as the eye or the meninges, or from such complicating diseases as broncho-pneumonia with its accompanying bronchitis and atelectasis. The bronchial glands are often found to be enlarged.

SYMPTOMS.—The period of incubation of pertussis is variable, but is usually less than two weeks. The symptoms in the beginning, and often for several weeks, are simply those of a bronchial catarrh with a slight rise of temperature and a cough which, though sometimes spasmodic, is often indistinguishable from that of an ordinary bronchitis. After a period varying from a few days to two or three weeks, the cough becomes more severe and of a more decidedly spasmodic character, and the peculiar whoop which characterizes the disease appears. The cause of these paroxysms seems to be a spasm of the larynx. This is accompanied by a feeling of suffocation. The paroxysm begins with a number of short, spasmodic, expiratory coughs, succeeded by a long-drawn inspiration and by the peculiar whoop. During the paroxysm, especially in severe cases, the face and mucous membranes become cyanotic, the eyes protrude, the conjunctivæ are congested, and the child looks as though it would die of asphyxia. After a few seconds the child, with a convulsive cough, expels some tenacious mucus, and is then relieved, or the attack returns again, and again subsides, and the symptoms of asphyxia pass away. These paroxysms are often followed by vomiting. They may occur only four or five times in the twenty-four hours, or again much oftener,—at times thirty, forty, or fifty times. At the onset of the attack the children are usually very much frightened, and either run to the mother or nurse for aid, or go to some part of the room where they can be undisturbed during the attack. In certain children, after the severe paroxysms have lasted for some time, a small ulcer is formed on the frænum of the tongue. This is because the frænum is driven against the lower edge of the teeth during the paroxysms. During the course of the paroxysmal stage of pertussis it is quite common to have subconjunctival hemorrhages; rarely deeper-seated hemorrhages take place in the meninges and in the deeper parts of the eye. In protracted cases petechiæ sometimes appear in the skin. Epistaxis may also occur.

Examinations of the chest during the attack have shown that the pulmonary resonance is lessened during the expiratory stage and is clear during the prolonged inspiration. The auscultation usually shows diminution or absence of the respiratory murmur. Bronchial râles are heard occasionally.

Koplik has noticed an increase in the area of the relative cardiac dulness during the paroxysmal stage of pertussis, which is often accompanied by a slight blowing murmur limited to the apex of the heart. This may well occur from the engorged condition of the right side of the heart, which subjects the heart to a great strain and may thus result in dilatation. The heart-sounds are apt to be irregular during the paroxysm, and in protracted cases during the intervals the pulse is often irregular and quickened, while the respirations are not especially increased unless some complication has arisen. In severe cases of pertussis the kidneys are sometimes congested, as shown by the appearance in the urine of albumin, casts, and blood-cells. Sugar has also been found quite frequently.

After the disease has lasted for some weeks there is usually a certain amount of œdema of the face, especially under the eyes. The paroxysms are precipitated by nervous excitement or by an irritation in the throat or the respiratory tract, such as may result either from swallowing or from the inhalation of dust. The stage which is accompanied by the whoop and the more exaggerated paroxysms commonly lasts for three or four weeks, or even longer. The paroxysms then become less severe, and, although the cough continues, the whoop gradually becomes less frequent, and after three or four weeks more ceases entirely. When uncomplicated, the duration of the disease is usually three or four months. Slight changes in the atmosphere or exposure will give rise to a relapse. The relapses, however, are not, as a rule, of a severe type, and in these cases the cough seems to arise from renewed irritation of the sensitive mucous membrane of the respiratory tract rather than from a fresh infection by the specific germ. A persistent cough following an attack of pertussis may sometimes, according to Delafield, be caused by an insidious form of broncho-pneumonia.

The period of infection is supposed to last for a certain time after the whoop has ceased, and if the cough continues it is well to allow for a period of infection of three weeks after this cessation. It is possible, however, that the whoop may occasionally occur for long periods after the child has ceased to be a source of infection to other individuals.

COMPLICATIONS.—The complications which arise in pertussis are usually of a grave nature. The dangers from hemorrhages, unless in the form where they occur in the meninges, are not great. The complication of broncho-pneumonia is very serious, and often fatal. Severe and even fatal emphysema may occur in pertussis.

Convulsions may arise not infrequently in infants and may end fatally, in these cases usually being caused by general reflex disturbance, by cerebral congestion, or by some cerebral lesion. Spasm of the glottis may also very rarely cause death in greatly debilitated children. Excessive and obstinate vomiting at times becomes a serious complication, and may reduce the child's strength to a point which often gives rise to a doubt as to its recovery. It is an especially grave complication in infants who are already much debilitated.

DIAGNOSIS.—The diagnosis of pertussis cannot, as a rule, be made until the child whoops. Sometimes, however, where another child in the family has undoubted pertussis, a spasmodic cough may allow the diagnosis to be made before the whoop has developed. It is probable that a child may have pertussis without at any time developing the whoop.

In some children a simple catarrhal laryngitis will simulate pertussis quite closely; but, although in these cases there are paroxysms of spasmodic coughing, a pronounced "whoop" does not occur, and the symptoms do not progressively increase and last for a long period. The diagnosis of pertussis can usually be made by the swollen aspect of the face, the paroxysmal cough followed by the expulsion of tough mucus and vomiting, and the long duration of the attack.

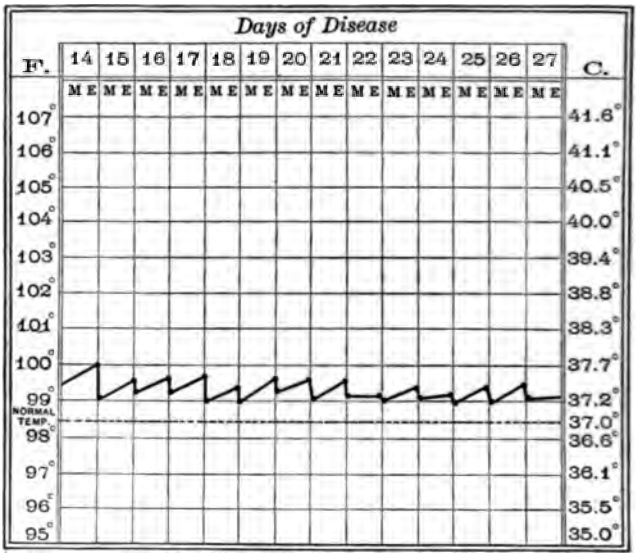
PROGNOSIS.—Pertussis is a very serious affection in young infants, and also in older children who are debilitated or poorly cared for. Where it is complicated it is one of the most fatal diseases which occur in early life. When it occurs in older children the prognosis is favorable, provided that they have previously been well and strong, that they are well cared for, and that no complications arise.

In some cases young infants, if their vitality is unusually good, and if they are carefully nursed and made to take a sufficient amount of food, show remarkable powers of resistance during attacks of pertussis.

A case of this kind that came under my care was that of an infant (Case 476), five months old. In March she suffered from an attack of epidemic influenza, which lasted about twelve days, and from which she finally recovered. She was then attacked with measles, and after the temperature had fallen to the normal point she was attacked with pertussis. After two or three days the cough increased in severity, and after two weeks the infant began to whoop. The attack lasted for two months, and she finally recovered. During the whole course of the disease she took over 600 c.c. (20 ounces) of modified milk in the twenty-four hours, and for a short time small doses of brandy were given. No drugs were administered. Here is a chart (Chart 41, page 1002) which shows the average range of temperature for two weeks when the disease was at its height.

TREATMENT.—In the treatment of pertussis we must take into consideration the age of the individual, the stage of the disease, and the presence or absence of complications. In the early months of life, after the disease has lasted for a week or ten days and has become more severe, the infant will usually show symptoms of general circulatory disturbance. The great strain thrown upon the heart during the paroxysms quickly affects the general strength of the infant, a marked interference with its nutrition soon appears, it loses in weight, and often it refuses its food. At times it will become somewhat cyanotic even between the paroxysms, and there is danger not only from the severity of the paroxysm, but also from the vitality of the infant being so much interfered with as to prevent its recovery. In cases of this kind the nursing is of the utmost importance. The infant should never be left alone, should always be taken up whenever a paroxysm is approaching, and should be assisted in various ways until the paroxysm is over. Holding the infant in different positions, sometimes bending the head and body forward at the end of the paroxysm so as to aid by gravity the expulsion of the tenacious mucus, is desirable. At times, also, the finger covered with a thin cotton cloth can be quickly introduced into the throat and the mucus withdrawn in this way. It is of the utmost importance that the infant should be surrounded continually by fresh air. For this purpose two rooms should be used, if possible, one of which should have all the windows thrown wide open, so that the air can be completely changed before the infant is brought into it, and the patient should be alternately taken from one room to the other, the temperature of the rooms being kept as equable as possible. The nutrition of the infant is so easily affected that the utmost attention should be paid to the administration of the food. Small quantities of a milk carefully modified to suit its digestion should be given at frequent intervals, preferably after the occurrence of a paroxysm, as it is then more likely to retain the milk in its stomach a sufficient length of time for it to be absorbed before the next attack. The amount of food which the infant retains in the twenty-four hours is an important factor in the treatment. In infants of from six to twelve months at least 600 to 750 c.c. (20 to 25 ounces) of milk should be taken and retained in the twenty-four hours. When the amount is lessened to 360 or 450 c.c. (12 to 15 ounces), the infant's nutrition, as a rule, suffers to such an

CHART 41. (CASE 476.)



Pertussis. Female, 5 months old.

extent that unless this amount can be increased a fatal issue is likely to result.

Stimulants, in the form of brandy or whiskey, should be given early in the attack. Where the cyanosis is a prominent feature and the pulse is irregular and intermitting, small doses of digitalis should be given. In these cases, also, the administration of oxygen is a valuable adjunct to the treatment. At the height of the attack, when the paroxysms are severe and especially frequent at night, the burning of cresoline in the room at night is in some cases beneficial. In the milder cases not accompanied by the more severe symptoms which I have just enumerated, belladonna or atropine often proves valuable. There is no drug, however, which is a specific for pertussis at any age.

For older children whose health has previously been good, there is no especial treatment, except that they should have as much fresh air, free from dust, as possible, and that food should be given them after they have vomited.

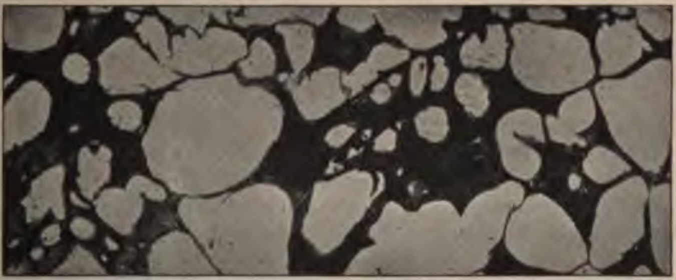
Where complications arise, the treatment is that of the complicating disease.

In cases which are protracted, a change of air, either to the country or to the sea-shore at suitable seasons, is often followed by an apparent shortening of the duration of the attack.

Prophylaxis.—Pertussis is so highly contagious a disease, and may be so serious an affection in certain children, that a rigid prophylaxis should be enforced. It is the duty of those who take care of children with pertussis to see that they are isolated during the whole course of the disease.

I have here a specimen (Fig. 142), made by Northrup, of a lung taken from an infant (Case 477) under one year of age, who died during a violent attack of pertussis.

Case 477. Fig. 142.



Emphysema following pertussis. Distended alveoli often coalescing.

The section shows extensive vesicular emphysema, with great distention of the walls of the alveoli.

I have here a little girl (Case 478), four years old, who is in the fifth week of an attack of pertussis.

The intervals between the paroxysms are usually one or two hours. She has just begun to cough, and you will have an opportunity of seeing her in one of the paroxysms.

PATHOLOGY.—There are no known pathological lesions which characterize the disease. In cases of long duration the lesions of chronic bronchitis are often found.

SYMPTOMS.—The symptoms of bronchial asthma are the same in the child as in the adult. The onset is usually sudden, and generally occurs at night. A catarrhal condition of the respiratory tract, especially of the bronchi, commonly precedes the attack for some days. The child is seized with distressing dyspnœa, mainly expiratory, the respiration being accompanied by a wheezing sound. The face is anxious, and if the attack continues for some time it becomes slightly cyanotic. The respirations are not especially increased in frequency. The pulse is rapid, and when the dyspnœa is very intense it is weak. The temperature is not raised by the asthma, and where the paroxysm is prolonged it may become subnormal. The physical signs are mostly diffuse, sibilant, and sonorous râles. The attack may last for a number of hours, or even for days. The paroxysms vary in their severity, and, as a rule, are followed by considerable exhaustion. The frequency of the attacks varies; they may occur often or only at intervals of months.

PROGNOSIS.—The prognosis of asthma with regard to the especial attack is good. Where the disease is not hereditary the children very commonly recover from it as they approach the age of puberty. In many cases the attacks seem to depend upon some local affection of the air-passages, and the cure of these local lesions will often be followed by recovery from the attacks of asthma.

TREATMENT.—In the treatment of asthma, the nose and throat should be carefully examined for local diseases, as the attacks may be caused by the different forms of rhinitis, adenoid growths, or enlarged tonsils. The children should be protected from unfavorable atmospheric influences, a high, dry, inland air usually being better suited to them than sea air. In some cases, especially of a mild form, the fumes of nitre paper will give considerable relief. In very severe attacks hydrate of chloral may be given, either by the mouth or by enemata. Antispasmodics, such as belladonna and lobelia, can also be used. There is no one drug which will relieve the paroxysms of asthma except morphine, which should be used with great caution. Iodide of potassium in gradually increasing doses is in some cases beneficial. Especial attention should be paid to the general hygiene and to the diet of the child.

PERIODIC CATARRH (Autumnal Catarrh; Hay Fever; Rose Cold).—Closely allied to asthma is an affection of the respiratory tract occurring periodically and characterized by great irritation of the mucous membrane of the eyes, nose, throat, and bronchi. The same causes that have been supposed to produce asthma seem to be of etiological importance in periodic catarrh. These attacks usually occur in the summer months, but are generally most severe in August and September.

The onset of the attack, in contradistinction to the paroxysms of asthma, is generally at some definite time of the year. The especial attack lasts for five or six weeks, or even longer. It is characterized by a severe acute catarrhal inflammation of the nose, eyes, throat, and bronchi. The coryza and lachrymation are in many cases excessive. As the disease progresses, the cough becomes very distressing, and the respirations are so impeded by the congested and swollen mucous membranes that sleep is interfered with, and the child's general nutrition is soon affected. There is no general remedy which controls the disease, and benefit usually can be obtained only by removing the child to a locality which is free from the causes that produce the disease.

The prognosis in children is good. The local treatment of the upper air-passages is the most likely means of obtaining a cure. If it is left untreated the disease occurs every year, so that just before the yearly attack begins it is well to have the children taken to the especial locality where it has been found that they do not suffer from the disease. In this way the impairment of their general health will be prevented, and it is possible that they will eventually cease to be affected by the disease.

Where the child cannot be removed from an irritating locality, temporary relief can be obtained from sprays of cocaine. As recommended by Wyman, the windows of the sleeping-room should be closed early in the evening and kept closed during the night. In this way the dust in the air is allowed to settle, and there is less danger that the irritating material, whatever it may be, will produce its effect when the child is asleep. As a rule, it is advisable to give the child quinine in tonic doses, beginning just before the date of the onset of the disease and continuing with it until the attack has almost run its course.

LECTURE L.

DISEASES OF THE PLEURA.

PLEURISY.—Inflammation of the pleura may be acute or chronic, and may be accompanied by an effusion, which may be serous, sero-purulent, or purulent.

Acute pleuritis, either with a simple exudation of fibrin or accompanied by fluid, is quite frequent in children. The effusion has a greater tendency to be purulent in children than in adults. It seems to follow exposure of various kinds and to be produced by a number of organisms. As a secondary affection it occurs especially after lobar pneumonia and pulmonary tuberculosis, also in the course of the acute exanthemata and in such diseases as rheumatism.

In regard to the micro-organisms which are supposed to produce pleuritis there is an evident difference in the intensity of the inflammation which follows their invasion. In the serous exudations the pneumococcus has been found most frequently, and seems to be most commonly present in the benign forms of the disease. Next to the pneumococcus the staphylococcus has been found to be present in the least virulent forms. The bacterium which has been found in the pleuritic effusions of the severest cases is the streptococcus. In those effusions which arise from tuberculosis the bacillus tuberculosis has been found.

PATHOLOGY.—Pleuritis is usually a unilateral disease, but may in rare cases be bilateral. The pathological conditions found in the pleurisy of children do not differ from those which occur in later life. Although localized areas characterized by the production of fibrin (dry pleurisy) are quite frequently found at the post-mortem examination, the diagnosis of this form of disease in infants and in young children is not often made during life. Where, however, large areas of the lung are involved in broncho-pneumonia, dry pleurisy quite frequently occurs, and small circumscribed areas are commonly met with in connection with lobar pneumonia. In the common form of pleurisy, where there is a production of fibrin and serum (pleurisy with effusion), a greater part of the pleura of one side of the chest is usually involved. According to Delafield and Prudden, while the inflammation is in progress the surface of the affected pleura is coated with fibrin, bands of fibrin stretch between the parietal and pulmonary layers of pleura, and in the pleural cavity there is serum in variable quantities. This serum is sometimes clear, sometimes is turbid from the presence of pus-cells and flocculi of fibrin. Both these forms of pleurisy, although differing in their clinical history, are anatomically essentially the same. In both we find, first, the exudation of fibrin and a few pus-cells either with or without serum; second, a gradual absorption of the serum and fibrin; finally, a formation of new permanent connective tissue in the form of adhesions or of thickening of the pleura. Through the whole process the tissue of the pleura is but little changed. The products of inflammation, although they originate in the tissues, do not infiltrate it, but make their way to its surface, accumulate there, and undergo different changes. Variations from the regular course of the inflammation are caused by the excessive formation of the fibrin, the serum, or the pus, and by the manner in which these inflammatory products are absorbed. It is still, however, undetermined whether acute pleuritis with a serous effusion is an entirely separate disease in children from a pleuritis with purulent effusion, or whether the difference between the two diseases is merely one of degree in the amount of pus-cells present. Clinically there is a certain amount of evidence in favor of the former supposition, as an acute pleuritis with serous effusion in young children usually runs a definite benign course and is reabsorbed without becoming purulent. Empyema in young children, on the other hand, is frequently, so far as can be determined, a purulent exudation from the beginning. It is therefore better in describing pleuritis in infancy and early childhood to speak of the serous effusion and the purulent effusion as two separate diseases.

SYMPTOMS.—The onset of acute pleuritis with serous effusion is in many cases violent, and attended by a high temperature, increased respirations, quickened pulse, restlessness, and even pain, which in young children is usually referred to the abdomen. In infants and in young children convulsions are quite common, while in older children the symptoms are more like those which occur in adults. There is a short, painful cough, with loss of appetite, and frequently vomiting and diarrhœa. These early symptoms are usually followed in two or three days by an exudation and by a decided lessening of the pain and dyspnœa. At the same time the temperature begins to have a decided morning remission. When the exudation is large, the children lie more comfortably on the affected side, and when they are nursing they nurse most easily from the right breast if the left pleura is affected, and from the left breast if the right pleura is affected. After the serous effusion has remained for a number of days it ordinarily begins to lessen in amount, absorption takes place, and by the end of a week or ten days it becomes entirely absorbed and the child recovers. In other cases it becomes chronic unless its absorption is furthered by aspiration.

The physical signs of pleurisy before the effusion has taken place are in infants and in young children quite difficult to detect. The friction-rub is often absent and the pain is difficult to locate. It is frequent, however, to find that there is tenderness on the affected side on palpation and percussion, as the child cries more when the affected side of the chest is compressed.

When the effusion has taken place, the chief physical signs are dulness on percussion, bronchial respiration, and, if the effusion is in considerable amount, displacement of the heart. The other physical signs, such as decreased vocal resonance and fremitus, which are commonly met with in the pleurisy of adults, are not, as a rule, sufficiently marked in infancy and early childhood to be of much value for diagnosis. Great difficulty may arise in auscultation from the finer sounds being obscured by the child's crying, but in the intervals when the child takes a breath and its cry must necessarily cease for a moment, valuable information can be obtained by the quick use of the stethoscope.

Where the effusion is sufficiently large to displace other organs, such as the liver and the spleen, the presence of the effusion is so evident from the usual signs that these displacements are not of especial value except so far as they show that the effusion is in large amount. After these large effusions have lasted for some time, and especially when they are purulent, I have met with decided bulging of the affected side.

DIAGNOSIS.—The diagnosis of pleurisy with serous effusion is to be made from lobar pneumonia and from empyema. The physical signs which in the adult are most useful in differentiating pneumonia from a pleuritic effusion are often misleading or absent in the young child. Thus, absolute dulness may occur in other conditions as well as in a pleuritic effusion, while bronchial respiration, such as is heard over a consolidated lung, may also be heard over a large effusion. The vocal fremitus may be absent in a pneumonic consolidation, and sometimes, though rarely, well marked over an effusion. Moist râles have been heard in children over an effusion, and fluid has been aspirated at a point where a friction-rub was heard. It is well known, also, that aspiration is not a conclusive means of diagnosis, for punctures have often been made where an effusion was present and yet no fluid was obtained. The change in the level of the effusion on change in position is of some value in diagnosticating a pleuritic effusion from pneumonia, but is often difficult, and at times impossible, to determine in young children. The most reliable means of diagnosis in infancy and in early childhood is percussion. The area of dulness which occurs in lobar pneumonia is quite different from that which is found in cases of effusion uncomplicated by previous adhesions. If adhesions are present, these typical changes are so interfered with that the percussion becomes as unreliable a sign as the others which I have just mentioned. The younger the individual, however, the less likely are extensive adhesions to be present, and the more valuable, therefore, is the evidence of an effusion given by percussion. As has been shown by Whitney, when the effusion is small there is absolute dulness (flatness) at the base of the thorax. A friction-sound may be heard over the dull area, and respiration may be quite distinct, and sometimes accompanied by râles. Under these circumstances the diagnosis of the condition as one of effusion must depend upon the outline of the area of dulness. In determining these small areas of dulness the lower border of the two sides of the thorax must first be carefully compared by percussion, bearing in mind that the lower border of the pulmonary resonance in early life corresponds to the position of the ninth dorsal vertebra on the right side and to that of the tenth dorsal vertebra on the left, as I have already explained to you in my lecture on development (page 122).

You must always remember that the percussion of an infant's or a young child's chest should be very light, as heavy percussion, owing to the delicacy of the thoracic walls in early life, is unreliable.

Palpatory percussion, for the same reason, gives more information when the child is crying than can usually be obtained by the sound, but, as I have said in describing the auscultation in these cases, quick percussion in the intervals of respiration is also a valuable aid to diagnosis.

Extended observations have been made on the line of percussion-dulness found in medium effusions by Ellis and Garland, and lately in small effusions by Whitney, of Denver. These investigators have shown that as an effusion increases in quantity its upper border undergoes a gradual series of changes, provided there are no adhesions.

Where the effusion is small in amount it can usually first be detected in the back. In these small effusions the upper border of absolute dulness begins at the vertebral column, extends outward horizontally for a distance which varies according to the size of the effusion, and drops in the neighborhood of the posterior axillary line by a curve more or less abrupt to the base of the thorax. As the effusion increases in size the line of dulness drops more anteriorly. Where the effusion is moderate, as where the lower half of the pleura is filled, in some cases, but not usually, a slight displacement of the heart may be noticed, and the percussion over the slightly compressed lung may give tympanitic resonance. The upper border of the area of dulness in these medium effusions is found to extend at first outward and then upward over the angle of the scapula, reaching its highest point in the axillary region. The line may then drop abruptly from the upper axilla to the base of the thorax near the apex of the heart. This line, which has been called the "letter S" curve, is characterized by having its highest point in the axillary line. When the quantity of fluid is still larger and exceeds a certain amount, the "letter S" curve is obliterated, and the resonance over the compressed lung becomes less marked. The displacement of the heart in this latter case is a most valuable sign of effusion in young children, and with careful, light percussion the gradual increase and decrease of the effusion where it is of any great extent can be determined by cardiac percussion.

You must always bear in mind the physiological dulness of the heart which I have already described as occurring in early childhood (page 123) under the lower third of the sternum. This dulness, however, is relative, and becomes much more marked and absolute where it is caused by a displaced heart.

The differential diagnosis from lobar pneumonia is greatly aided by understanding these areas of percussion-dulness which I have just described. Thus, where the diagnosis is to be made between pneumonia of a lower lobe and a small or medium pleuritic effusion, where an effusion is present the dulness will be in the lower part of the thorax, with normal or tympanitic resonance above it; where pneumonia is present the area of dulness will often correspond to the boundaries of the lower lobe only. In like manner the area of dulness of the effusion will differ in the axillary regions and in the front of the thorax from the areas of dulness produced by the consolidation of the different lobes of the lung.

The diagnosis from empyema is very difficult, as the younger the individual the more likely are the effusion to be purulent and the early symptoms to be similar to those of the serous form of exudation. After the first week or ten days of the disease, however, where the effusion is purulent, the usual signs of absorption which so commonly occur in a serous effusion are ordinarily not found, and aspiration of the pleural cavity will then determine which form of the disease is present.

Where no bacteria are found in the fluid, where there is no history of a preceding acute pneumonia or a neoplasm of any kind, or where there is little tendency to absorption of the exudate, and where the exudate is found to contain blood, the failure to find bacteria in the exudate may be regarded as pointing strongly towards a tubercular origin.

PROGNOSIS.—The prognosis of a serous effusion, as a rule, is very favorable in infants and in young children unless one of the more virulent forms of the pyogenic cocci is present, or unless the disease is secondary to tuberculosis elsewhere and is caused by the bacillus tuberculosis. If the serous effusion tends to become purulent, the prognosis is not so good, but still, provided appropriate treatment is carried out, it is favorable. If, as in rare cases, the pleuritic effusion occurs on both sides, the prognosis becomes grave. The possibility of the presence of tubercle should be considered in these latter cases.

I have had in my service at the City Hospital a boy (Case 480), thirteen years old, who was attacked with pleuritis and a serous effusion of the left side with displacement of the heart to the right. After one aspiration the fluid was quickly absorbed, but three weeks later he was attacked with pleurisy on the right side, followed by an effusion and displacement of the heart to the left. This effusion was absorbed without aspiration, and the boy was discharged from the hospital well and strong, with both lungs apparently in a normal condition.

Where the effusion is very large and the heart is much displaced, there is always the danger of a fatal issue from asphyxia, and the prognosis depends upon whether the effusion can be controlled by aspiration and the heart thus be kept in normal position. A case which illustrates the danger of these large effusions accompanied by displacement of the heart came under my care at the Children's Hospital:

A boy (Case 481), four or five years old, entered the hospital with a large effusion in the left chest. The heart was displaced to the right, and upward as far as the second interspace to the right of the sternum. He was cyanotic and gasping. On aspirating the chest and removing a large quantity of fluid, the heart reassumed its normal position under the sternum. On the following night the boy died suddenly, the effusion having rapidly accumulated and having again displaced the heart.

Cases of this kind should warn us that a pleuritic effusion of any extent in a young child should be watched continuously, and that aspiration should be performed where there is indication of an increase in the intrathoracic pressure.

Where the pleurisy is secondary to other diseases, such as rheumatism and scarlet fever, the prognosis is not so favorable: the effusion is not apt to be absorbed so readily, and is more likely to become purulent. The prognosis is also rendered more unfavorable in these cases by the prolonged pressure upon the lung, with its corresponding ill effects upon the general condition of the child. The dangers which arise from the development of tuberculosis must also be borne in mind.

TREATMENT.—The treatment of pleuritis during the early days of the attack, before an effusion of any considerable extent has appeared, should be directed to the relief of the pain by a flannel bandage closely applied to the thorax, so as to allow the ribs to move as little as possible in respiration. Sometimes an occasional dose of tinctura opii camphorata will also be needed to make the child comfortable. After the effusion has increased, the child should, if possible, be kept in bed. There are some cases, however, where a child with a considerable effusion in its pleura will feel well and bright, and will play about its nursery without showing any especial symptoms of discomfort. I have met with instances of this kind where, excepting that it was pale and had a poor appetite, the child seemed bright and active, and yet it had a pleuritic effusion large enough to displace the heart.

In mild cases, after the effusion has attained its maximum, you should carefully examine the child each day, to see whether there is a rapid increase in the fluid, which by displacing the heart and causing dyspnœa would render necessary immediate relief, or whether the fluid has begun to be absorbed. In the latter case an expectant treatment is all that is required. In the former case, or if absorption of the fluid is delayed for two or three weeks, the chest should be aspirated. A bacteriological examination of the fluid removed should then be made, to determine which form of organism is present in the exudate. If one of the more benign forms of bacteria is present, such as the pneumococcus, or if the fluid is found to be serous, no further treatment will be required, unless there be a reaccumulation of the fluid, in which case a second aspiration will be indicated. If, however, the streptococcus is found in the exudate, the case must be watched very carefully, as it is more likely to become purulent and to need radical treatment.

As the unfavorable symptoms in a pleuritic effusion arise mostly from intra-thoracic pressure, relief from the pressure by aspiration is indicated rather than by the use of drugs, which cannot be depended upon.

The point of aspiration should usually be in the fourth or fifth interspace in the axillary line, or a little farther back.

Purulent Pleuritis (Empyema).—Empyema is a purulent effusion into the pleural cavity. In the first three or four years of life it is much more common than a serous effusion.

The cause of these purulent effusions is the same, so far as we know, as that of serous effusions. The same organisms are present in the two forms, and they are also frequently present when purulent pleurisy is secondary to a number of diseases, the most prominent of which is lobar pneumonia.

Usually the whole pleura is involved, encysted empyemas in infants and young children being rare.

The disease when primary may be acute in its onset, and may simulate closely the initial stage of lobar pneumonia. In other instances it is slow and somewhat insidious in its development. The pulse and respirations may be quickened, but after the early days of the disease they are often very little raised. There is nothing characteristic in the temperature of an empyema, and the diagnosis usually can be made only from the knowledge that the younger the individual the more likely is pus to be present. This, however, can be determined definitively only by means of the aspirator.

The physical signs are the same as in a serous effusion. The absorption of a purulent exudate without surgical interference is very rare. I have occasionally met with cases where one aspiration was all that was necessary, and where, seemingly, complete absorption took place.

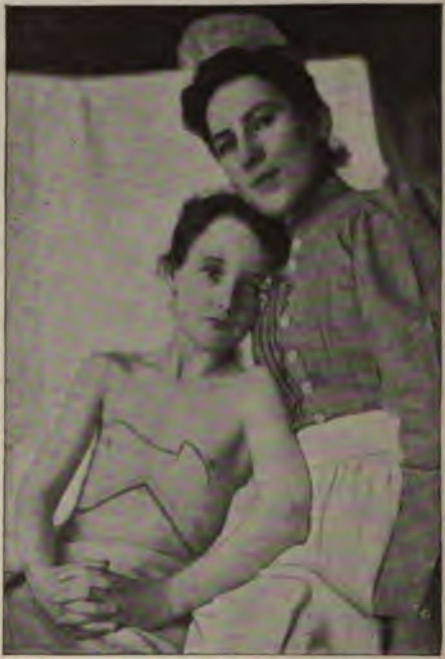
Where cases of empyema are left untreated, a spontaneous opening usually takes place through some portion of the thoracic walls, but the exudate may also find its exit through the lungs by opening into one of the bronchi or perforating in other directions. I have met with cases where the diaphragm was perforated and the point of exit of the pus was in the region of the umbilicus. Where perforation does not occur, the pus is partially absorbed, adhesions are formed, and sometimes great deformity of the chest follows, which may result in a marked degree of lateral curvature of the spine as well as in great contraction of the chest.

After the first aspiration, unless absorption occurs within a week, and especially if one of the more virulent forms of bacteria is found in the exudate, a radical operation is the best method of treatment. Where this is performed early in the disease, the prognosis is very good in infancy and early childhood, unless the empyema is of a virulent form or is secondary to some incurable disease, such as is caused by the bacillus tuberculosis. The pleural cavity should be thoroughly drained by means of drainage-tubes. In many cases, especially in children over two or three years of age, resection of one or two ribs gives the best results. Although in some cases a rapid cure in two or three weeks follows the operation, yet the recovery is often prolonged for many months, even where strict antiseptic precautions have been taken at the time of the operation.

As the treatment of empyema is essentially surgical, I shall not enter into its details.

This little girl (Case 482), eleven years old, was attacked six weeks ago with a chill followed by vomiting. She then had a short, dry cough. For the past few days she has complained of pain in the lower part of the right chest. She has been feverish, has lost in weight and in appetite, and her respirations have been painful. She lies most comfortably on her back and on her left side. A pleuritic friction-rub has been heard in the right axillary region. Her lips and cheeks are slightly cyanotic.

CASE 482.

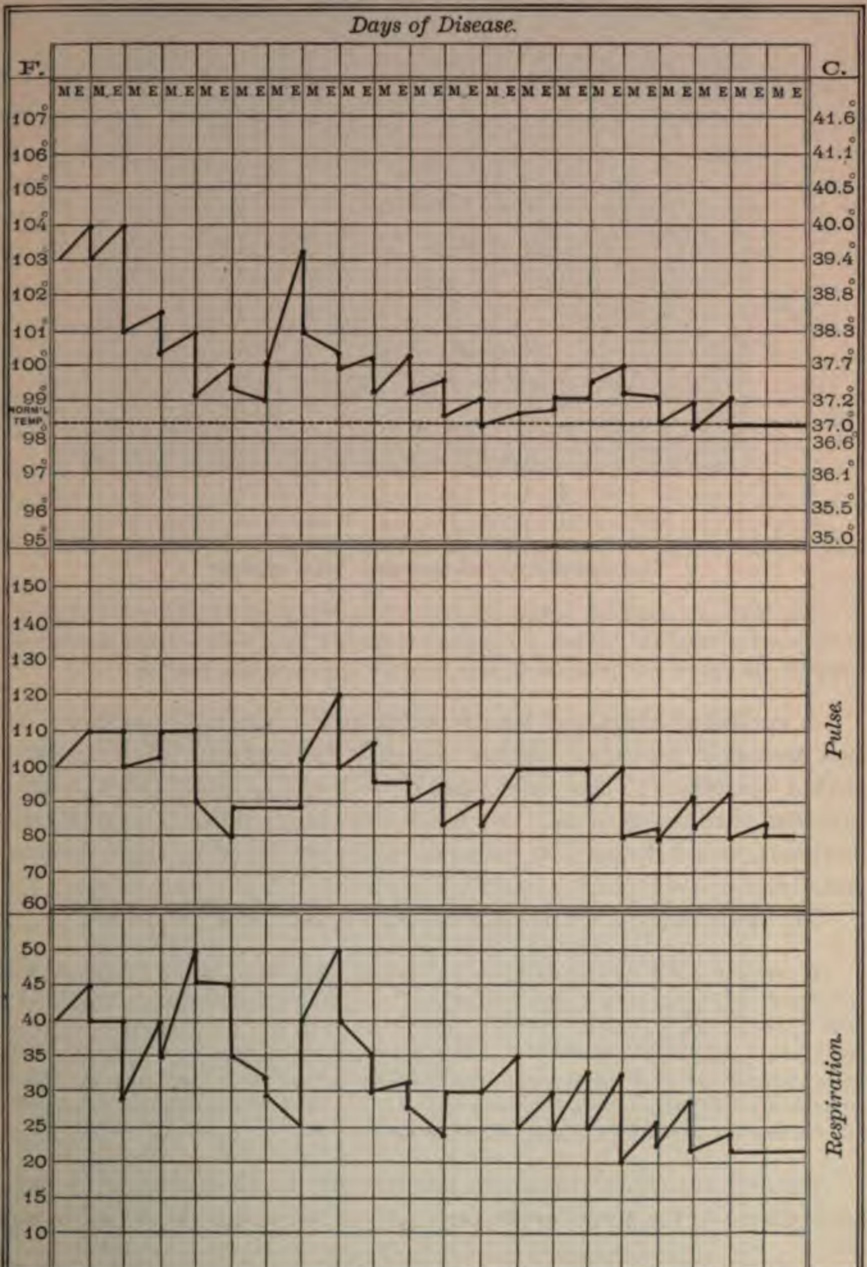


Acute pleurisy with serous effusion. Female, 11 years old. The line of the upper border of the effusion, the area of cardiac dulness, and the margins of the ribs are marked in black.

Her tongue is somewhat coated. The alæ nasi are working, and, as you see, the dyspnœa is so marked that she has to sit almost upright in bed. The percussion and auscultation of the left lung show nothing abnormal. The resonance is fair over the upper part of the right front and back. There is absolute dulness from about the fifth dorsal vertebra in the right back to the base of the lung. This dulness extends into the axillary region, where it reaches its highest point, and then gradually descends to the right parasternal line on a level with the fourth costal cartilage. Over this area of dulness respiration is markedly diminished. No friction-rub is heard. The vocal and the tactile fremitus are diminished. The impulse of the heart is found in the fourth interspace, 1 cm. ($\frac{3}{8}$ inch) to the left of the mammary line. The heart-sounds are normal. There does not appear to be any displacement of the liver. An examination of the urine shows it to be acid, to have a specific gravity of 1022, to be of normal color, and to contain no albumin. The chlorides are normal. The physical signs are those of a pleuritic effusion of the right side with displacement of the heart to the left.

(Subsequent history.) During the following week the area of absolute dulness gradually decreased, and an exploratory aspiration showed the fluid to be serous. Three weeks from the time when she entered the hospital, and nine weeks from the beginning of the attack, the dulness on percussion gradually disappeared, auscultation showed the respiration to be normal, and the heart resumed its normal position. This chart (Chart 42) shows the temperature, pulse, and respiration while she was in the hospital.

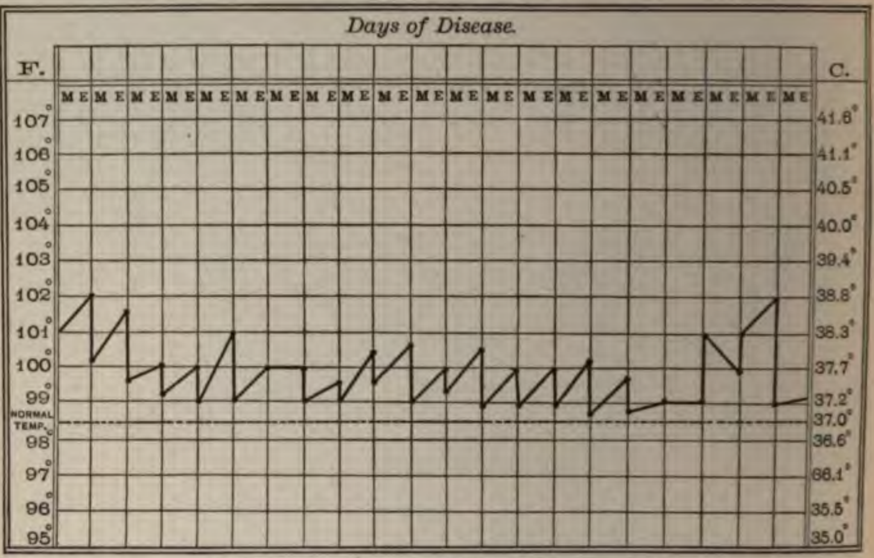
CHART 42. (Case 482.)



Acute pleurisy with serous effusion. Female, 11 years old.

This chart (Chart 43) shows the temperature for twenty-one days in a case (Case 483) of serous effusion in the pleura, where in the beginning 165 c.c. (5½ ounces) of fluid were withdrawn from the chest.

CHART 43. (CASE 483.)



Acute pleurisy with serous effusion. Male, 4 years old.

The fluid reaccumulated, so that absolute dulness was found over the whole right side of the chest in front and behind, but aspiration did not have to be resorted to again, and complete absorption took place thirty days from the beginning of the attack.

A number of cases have been reported in which a purulent effusion has been treated by aspiration and has seemingly disappeared entirely without a radical operation. These cases should be borne in mind when treating purulent pleuritic effusions. An infant (Case 484), seven weeks old, with empyema, at the Boston City Hospital, in the service of Dr. Doe, recovered entirely after one aspiration.

The initial stage of empyema often closely simulates pneumonia.

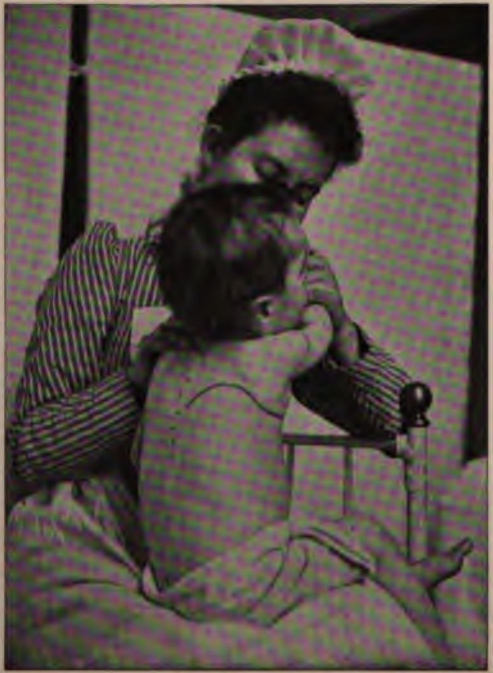
A case illustrating this fact has come under my observation, where a boy (Case 485), three years old, and previously well, was attacked with pain in the left side, with a chill and with dyspnœa. A physical examination made on the third day of the attack showed the right lung to be normal. On the left side of the chest there were absolute dulness, diminished respiration, and increased vocal resonance, and fine râles were heard from the fifth rib to the base of the lung, both in front and behind. Two days later an exploratory aspiration showed that the physical signs were caused by an empyema.

Another case which illustrates the difficulty in diagnosticating a purulent effusion in the pleura in the early days of the disease is the following:

A girl (Case 486), four years old, was suddenly attacked with cough, and pain in the right side. The temperature was 40.5° C. (105° F.). The respirations were quickened, and the pulse was rapid. Nothing abnormal was detected on physical examination. On the following day the general symptoms disappeared, and the temperature fell to 38.8° C. (102° F.). In another day the temperature fell to 37° C. (98.6° F.), and the child seemed

CASE 488.

I.



Purulent pleuritis of right side. Female, 1½ years old. The black line shows the upper border of the effusion.

II.

Scar left after operation for purulent pleuritis.

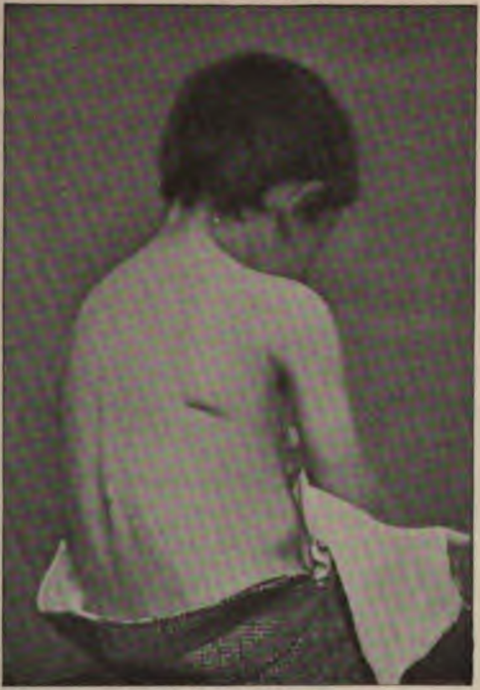
bright and well. On the following day, however, the temperature rose to 40° C. (104° F.), absolute dulness was detected in the right axillary region, and an exploratory aspiration showed the presence of pus.

Here is a little girl (Case 487), three years old, who two years ago had an attack of some pulmonary disease accompanied by fever. Since then she has been delicate and has coughed a great deal. Her cough has increased in the last few weeks, but she has not lost in weight nor had any other abnormal symptoms. She is pale, and the cervical, axillary, and inguinal glands are enlarged. Her fingers are markedly clubbed. She shows a peculiar lateral curvature of the spine, which cannot be made to disappear by traction. The right side of the thorax expands normally, the left side scarcely at all. There are hyperresonance over the right lung, no râles, and compensatory respiration. The left lung is apparently atelectatic, and shows dulness everywhere except in a small triangular area at the inferior angle of the scapula. This deformity of the thorax is probably the result of an empyema which occurred two years ago and was not properly treated.

Here is an infant (Case 488), one and a half years old, who entered the hospital with a history of an acute attack, characterized by fever, cough, and dyspnœa. Physical examination showed nothing abnormal on the left side of the chest, but on the right side there was absolute dulness, with bronchial respiration. No râles were heard anywhere in the lung.

The upper border of the area of dulness I have marked with a black curved line, and you see how, beginning at about the fourth dorsal vertebra, it gradually rises as it approaches the axillary line and then falls as it approaches the right parasternal line. The child's right arm has been turned upward and forward, in order that the ribs shall be sufficiently separated for the introduction of the trocar preparatory to an operation for the radical cure of the empyema. An exploratory aspiration has already shown the presence of a purulent effusion in the pleura.

CASE 489.



Recovery from empyema. Male, 10 years old, showing scar eight years after operation.

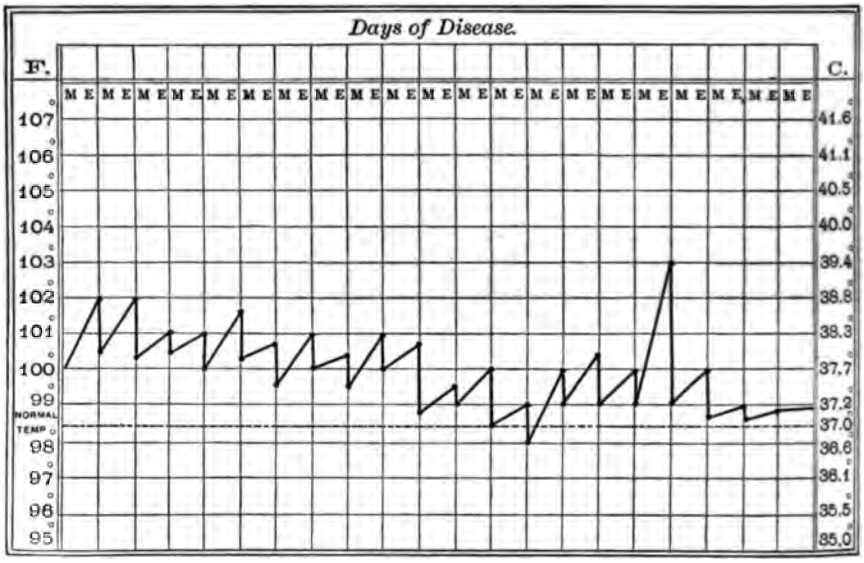
(Subsequent history.) The child was operated upon by Dr. Burrell, an incision being made in the mid-axillary line on the right side, and about 1.4 cm. (½ inch) of the seventh rib was resected. Nearly two quarts of pus were evacuated, and the pleural cavity was washed out with a boracic acid solution, a drainage-tube inserted, and a baked dressing applied.

This picture (Case 488, II.), taken some months later, shows the scar which was left after the operation.

This boy (Case 489, page 1017), ten years old, had a purulent effusion on the right side when he was two years old.

On entering the hospital with a history of having been sick for a number of weeks, there was found to be absolute dulness over the whole right side of the chest, with displacement of the heart to the left. Aspiration showed the dulness to be produced by a purulent effusion in the pleura. I made a permanent opening, and after thirty-six days the boy recovered, and was discharged from the hospital with the lung apparently normal. I show him to you to-day in order that you may see how the scar looks after a number of years. You see that the right side of the chest is equally expanded with the left, and that no deformity has resulted from an extensive empyema.

CHART 44. (Case 489.)



Purulent pleuritis. Male, 2 years old.

This chart (Chart 44) shows the temperature during the twenty-one days previous to the removal of the drainage-tube.

DIVISION XVII.

DISEASES OF THE HEART AND PERICARDIUM.

LECTURE LI.

DISEASES OF THE HEART.

Cardiac disease in infancy and early childhood may be divided into congenital or acquired, developmental or inflammatory, functional or organic, acute or chronic. In this early period of life cardiac disease has certain characteristics in which it differs essentially from those which are met with in later life. One of these characteristics is that there is a more decided tendency to recovery than at a later period. Another is that, owing to the undeveloped condition of the infant and young child, interference with the growth of other organs and parts of the body may more easily result from diseases connected with the circulation than is possible in the case of the fully-developed adult. Thus, there are certain anatomical facts connected with the ossification of the sternum which become of great importance in connection with cardiac disease. Deformities of the thorax may result from the continued pressure of the enlarged heart on the soft and pliant sternum and costal cartilages of the young subject. These deformities do not arise merely where the individual is rhachitic, but may also depend upon the stage of development at which the cardiac disease begins. The deformity is more or less pronounced in inverse ratio to the age and in direct ratio to the time during which the cardiac disease has existed. The shape and extent of the deformity are also dependent on the degree of ossification which has taken place in the sternum. In young infants, where the entire sternum, as I have described in a previous lecture (page 71), is in a cartilaginous condition, the intra-thoracic pressure from an enlarged heart may cause a bulging of the whole front of the thorax. This may occur during the first year, and even up to the third year. As the child grows older, the manubrium and the second piece of the sternum become ossified and offer more resistance, while the third piece of the sternum, still remaining in a semi-cartilaginous condition, may be tilted. This may occur in children in whom the cardiac disease has not developed until the fourth, fifth, or sixth year. I have had under my care a child seven years old who at the age of five years had articular rheumatism with resulting cardiac hypertrophy, and who presented this displacement of the third piece of the sternum. No other signs of rhachitis were detected. The middle period of childhood is also a peculiarly unfortunate one for the occurrence of cardiac disease, because the heart grows so rapidly at this period that it requires a proportionately greater amount of intra-thoracic space for the normal performance of its function than it does later.

In addition to the injury which may be done to the thoracic walls by an enlarged heart, we must consider the interference with the normal uniform expansion so necessary for the growing pulmonary tissue, and the consequent loss of the elasticity which plays so prominent a part in the establishment of the equipoise which should exist in a perfected respiratory apparatus.

The occurrence of *diseases of the blood-vessels* is rare in infancy and early childhood in comparison with later life. *Aneurism* is rare. A *narrowing of the isthmus aortæ* is more common, and is one of the most marked of the congenital defects of the blood-vessels. Sometimes there is an *absence of the isthmus aortæ* during fœtal life. The compensation for this defect takes place by an increased action of the left ventricle and the establishment of a collateral circulation between the subclavian artery and the thoracic and the abdominal aorta. These malformations exert in varying degrees an influence on the heart, as the infant grows older, from increased blood-pressure.

CONGENITAL DISEASES OF THE HEART.—Congenital diseases of the heart are somewhat obscure in their etiology, but usually they result either from an interference with the normal development of the organ or from endocarditis, or from a combination of both. In order to understand these congenital lesions you must remember the chief points in the mechanism of the fœtal circulation, which I explained to you in a previous lecture (page 19). I then told you that the parts of the fœtal circulation at birth which were of most importance in reference to diseased conditions of the heart and great blood-vessels later were the foramen ovale and the ductus arteriosus. I also told you at what period after birth they disappeared. Where these remains of the fœtal circulation, which are normal during intra-uterine life and for a short period afterwards, continue as the infant grows older, they become abnormal and interfere with the equilibrium of the circulation.

Where the development of the heart has been interfered with in intra-uterine life, there results another set of malformations, the chief of which are an open ventricular septum, a transposition of the great vessels connected with the heart, and various malformations of the valves of the heart. Where, again, an inflammatory condition has taken place in intra-uterine life (fœtal endocarditis), various other morbid conditions result, the most common of which are connected with the pulmonary artery, causing stenosis or atresia, a narrowing of the conus arteriosus, and various malformations of the tricuspid valve and other orifices of the heart.

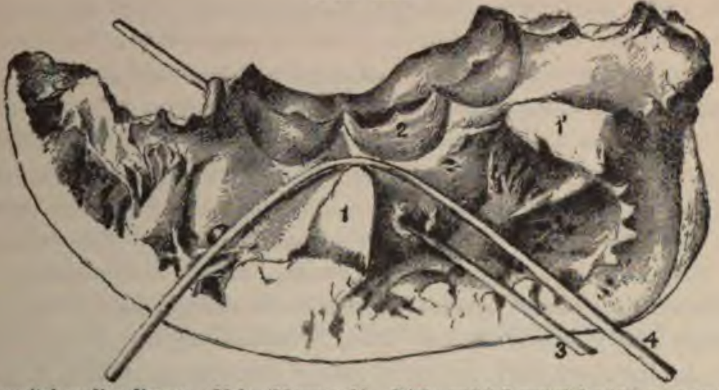
The form of inflammation of the endocardium which occurs in intra-uterine life is the chronic or sclerotic variety. Verrucose endocarditis is rare. (Osler.)

A deficient filling of the left side of the heart in early life, such as occurs in cases of atelectasis, fœtal pneumonia, or fœtal endocarditis, especially where stenosis of the pulmonary artery has resulted, may delay the closure of the foramen ovale and of the ductus arteriosus, which under these circumstances act as safety-valves. This is true also of the delay in the closing of the intra-ventricular septum, which is often of great aid in preserving the equilibrium of the circulation. In congenital cardiac disease it is usually the right side of the heart that is affected. The most common congenital cardiac lesions are an affection of the pulmonary artery, an open foramen ovale, an open ventricular septum, and an open ductus arteriosus.

The lesions most commonly found in connection with the pulmonary artery are stenosis of the pulmonary orifice, atresia of the orifice and of the artery, and stenosis of the conus arteriosus. Stenosis of the pulmonary orifice usually results from fœtal endocarditis, though it is possible that it may be the result of faulty development. The complete obliteration of the orifice of the beginning of the pulmonary artery is common, though not so frequent as stenosis of the orifice, and is probably of developmental origin.

I have here a specimen (Fig. 143) which was taken from a child (Case 490) with congenital cardiac disease under the care of Dr. Northrup. It illustrates this malformation of the pulmonary orifice.

FIG. 143.

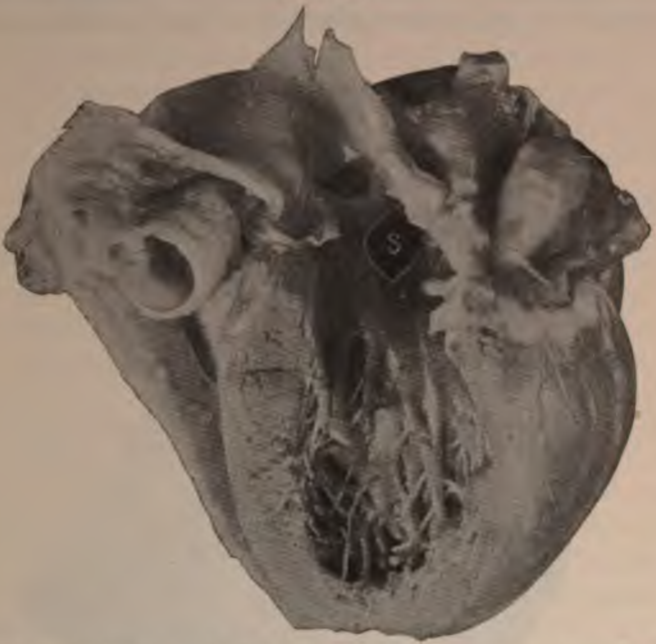


Congenital cardiac disease. Male, 4½ years old. Right and left ventricles laid open by two cuts. Stenosis of pulmonary orifice. Incomplete septum ventriculorum. 1 and 1′, septum ventriculorum cut across; 2, aortic valves; 3, probe passing through narrowed pulmonary orifice; 4, bent probe passing through right ventricle to left through opening in septum ventriculorum.

The specimen was taken from a boy four and a half years old, who during life had shown cyanosis, clubbed fingers, and at times severe dyspnœa. The physical signs in connection with the heart were a fine wave perceptible to the eye at the left third interspace, a soft, purring thrill over the base of the heart, cardiac pulsation 1.4 cm. ($\frac{1}{2}$ inch) outside of the left mammary line, and cardiac dulness from the right sternal margin to the left mammary line, tremely rare. I have already shown you this specimen (Fig. 6, page 42) of an infant's heart with an open foramen ovale.

Here is another specimen (Fig. 145), which shows a small opening in the ventricular septum.

FIG. 145.



S, unclosed ventricular septum. Female, 10 months old. Warren Museum, Harvard University.

In this case there was also an open foramen ovale, but no other malformation. The infant, after showing the usual progressive signs of congenital cardiac disease, died suddenly. There was no history of cyanosis.

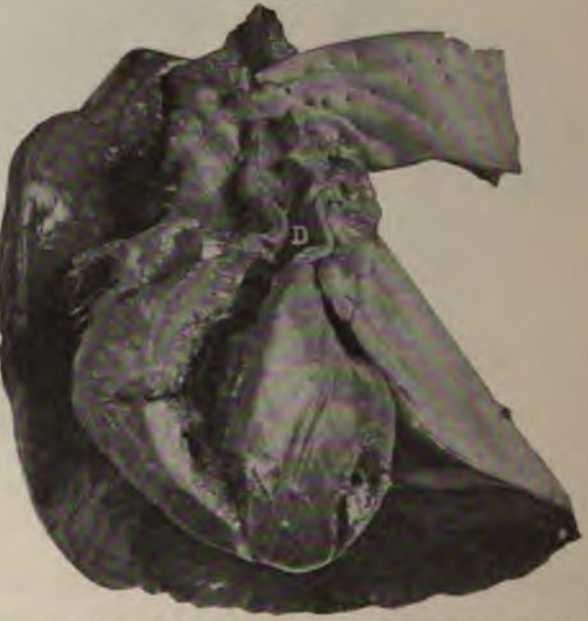
The fourth common congenital cardiac imperfection, an open ductus arteriosus, which I have just referred to, is shown in this specimen (Fig. 146, page 1024).

This heart, which has been left attached to the lung, was taken from an infant (Case 491), sixteen days old, who was apparently healthy at birth and presented no symptoms of cardiac disease.

When the infant was five days old it was noticed that it would sometimes become slightly cyanotic. At this time its temperature rose to 39.4° C. (103° F.). A physical examination showed nothing abnormal, and nothing abnormal was seen on inspection. The area of cardiac dulness was normal, and no murmurs were detected. A day or two later the temperature became normal; the cyanosis increased somewhat, but was intermittent and of a very slight degree. At times the skin would become cool. A few days later there was slight intestinal disturbance. When sixteen days old, without any other symptoms having developed, the infant died suddenly. The post-mortem examination made by Dr. Cutler showed this widely open ductus arteriosus. The foramen ovale is also open. There are no other lesions, such as stenosis of the pulmonary artery, open ventricular septum, or lesions of the valves. The heart is of normal size. As you see, there are no signs of the obliterative endocarditis usually found at this age in the ductus arteriosus.

The ductus arteriosus, as I have explained to you in a previous lecture (page 21), should gradually be obliterated within the first two weeks of extra-uterine life. Interference with this normal involution is not very uncommon, rarely occurs alone, and is usually found in connection with lesions of the pulmonary artery or narrowing of the isthmus aortæ. Sometimes the process of obliterative endarteritis, which has been shown by Dr. J. C. Warren to be the method by which the closure of the lumen of the ductus arteriosus is accomplished, extends to the aorta and causes stenosis of the isthmus aortæ. Again, the duct, in closing and retracting, pulls the aorta and tends to narrow that vessel, thus increasing the arterial tension. During fœtal life stenosis of the isthmus aortæ does not produce much disturbance in cases where the ductus arteriosus can carry the blood to the descending aorta. At birth, however, in these cases, unless the ductus arteriosus remains pervious, serious symptoms arise, and, if life be prolonged, hypertrophy of the left ventricle takes place, and the arterial blood has to be conveyed to the descending aorta by means of a collateral circulation which is established between the branches of the subclavian arteries and the branches of the thoracic and abdominal arteries. Premature closure of the ductus arteriosus during fœtal life has been met with, but is a rare condition. Very rarely the ductus arteriosus may be entirely absent.

FIG. 146.



D, open ductus arteriosus. Male, 16 days old. Warren Museum, Harvard University.

Imperfections of the tricuspid orifice are more rare. Lesions of the mitral valve are very rare in intra-uterine life. Those of the aortic orifice are rare in comparison with those of the pulmonary orifice, but are of the same nature,—that is, they may be developmental or inflammatory.

The duration of life where there are intra-uterine lesions of the aortic orifice is not nearly so long as where the pulmonary artery is affected.

Transpositions of the aorta and pulmonary artery are very commonly met with in connection with other congenital defects, such as spina bifida or hydrocephalus, but may occur in infants who are otherwise normally developed. In these cases the duration of life is almost invariably short.

Lesions of the valves vary greatly in their extent and kind.

On the boundary line between developmental and inflammatory conditions of the heart is a class of cases in which small hæmatomata are found on the valves. These hæmatomata appear just before or just after birth, and in the process of disintegration through which they pass may cause a contraction of the valvular tissue, and thus eventually produce the same symptoms that usually result from the more common valvular imperfections.

Although these various abnormal conditions may be found alone, yet they generally occur in combination with each other, and all kinds of transpositions and malformations of the vessels are at times met with.

There are various malformations of the heart which occur at an early period of fœtal development, and which are of pathological rather than clinical interest. Of these I might mention cases where there are one auricle and one ventricle (*cor biloculare*) or one ventricle and two auricles (*cor triloculare*), as well as a case which has come under my notice, where the heart had a double apex, the right apex lying in the fourth interspace to the right of the sternum, and the left apex lying in the fourth interspace to the left of the sternum.

SYMPTOMS.—Although in some cases the symptoms of congenital cardiac disease are very indefinite, and the disease may be masked for a number of months, yet in a large number of cases they soon become evident. The typical symptoms of congenital cardiac disease are cyanosis and attacks of dyspnœa amounting at times to suffocation and atrophy. As the disease progresses, the fingers often become club-shaped, the nails blue, and the skin cool. In connection with these rational signs there is usually an evident pulsation in the cardiac region, with bulging of the præcordia. Where the obstruction caused by the lesions is sufficient to produce hypertrophy and dilatation of the heart, an increase in the area of cardiac dulness is found. Diffuse cardiac murmurs are heard often over the whole chest, but usually have their maximum intensity towards the upper part of the sternum, and are commonly systolic in time.

The most common symptom is cyanosis. Remember that cyanosis may arise from incomplete oxygenation of the blood, and not merely from the mixture of the venous and arterial currents. Where cyanosis is present to any extent there is usually some malformation of the pulmonary artery or its valves. Well-marked congenital malformations may be present with no symptoms whatever. There may be an entire absence of cyanosis; there may be no increased area of dulness and no murmurs; and I have met with instances where the infants seemed to be thriving, and showed neither labored breathing nor physical signs of disease up to within a few hours of deat and yet where a number of cardiac malformations were found at the autops Although, as a rule, the symptoms occur at a very early period of extr uterine life, yet quite frequently they are so mild in their character th they are not noticed especially, as is the case when they appear only whe the infant is much excited or is crying. Again, the cardiac symptoms ma not be prominent enough to attract attention until the infant is old enoug to exert itself sufficiently, as by creeping or walking, to interfere with t equilibrium of its circulation. At times another disease, especially bro chitis or pneumonia, may precipitate the cardiac symptoms. Again, it quite common for endocarditis to develop in a heart in which a congenit malformation is present, and the diagnosis between a congenital and acquired cardiac affection then becomes necessary, and is accompanied many difficulties. As an illustration of how congenital cardiac disease c be masked for a number of weeks, I shall report to you a case which h lately come under my care.

This infant (Case 492) was apparently healthy at birth, and a careful physical exam nation showed nothing abnormal in the thorax. There was no cyanosis noticed, the sk being of a normal color. When it was sixteen days old it refused to take the breast, a in the afternoon seemed somewhat cold, was slightly cyanotic, and had a temperature 35.2° C. (95.5° F.). An examination of the heart detected nothing abnormal. A fe drops of brandy were given to it, and after several hours the skin became warm, the resp rations normal, and it took its food as usual. Early in the following morning the quic ened respiration returned, the temperature rose to 37.7° C. (100° F.), it refused to take i food, failed rapidly, and died in the afternoon.

The examination of the heart by Dr. Mallory showed a large open foramen ovale a an absence of the upper part of the intra-ventricular septum below the aortic valve. T beginning of the aorta for a distance of 1 cm. (⅜ inch) was dilated into a spherical pouc from which were given off (1) the aorta without any branches before the intercostals, th supplying only the lower part of the body, (2) a large vessel to the right lung, and (3) large vessel to the left lung. From the upper part of the right ventricle was given off large vessel which divided 1.4 cm. (½ inch) above the pulmonary valve into a large ves on the right side and two smaller ones on the left. The large vessel apparently cor sponded to the innominate, and the other two vessels to the subclavian and common carot of the left side. By these vessels blood was supplied to the head and upper extremit There was no communication between the arterial and pulmonary vessels, as the duct arteriosus was absent. The cause of the dilatation of the beginning of the aorta wa thickening and narrowing of the vessel for 8 mm. (¼ inch) just beyond the dilatation. T heart was enlarged, but not especially hypertrophied.

There was a general streptococcus invasion, for which no cause could be found. T cord had come away at the usual time without leaving any abnormal condition in t neighborhood of the umbilicus.

DIAGNOSIS.—Although from what I have just told you concerning t symptoms it is usually possible to make a diagnosis of congenital cardi disease, yet when we consider the variety of lesions which may occur, a the combination of different lesions which may be present, you will und stand that a diagnosis of the especial lesion is, as a rule, impossible.

Bearing in mind the mechanism of the fœtal circulation (Diagram page 19) and the connection which an enlargement of the heart has wi especial lesions, we can sometimes arrive at an approximately correct diagnosis; but no reliance can be placed upon the locality or sound of the cardiac murmurs, as such murmurs may be produced by very trivial lesions, and may be absent where the lesions are most pronounced.

Prognosis.—Where the lesion is connected with the pulmonary artery, and there is an open ventricular septum to act as a safety-valve, the equilibrium of the circulation may be retained to such a degree that the child will live for a number of years. Where the only malformation is an open foramen ovale, life may be prolonged for many years. Where, however, other malformations are present, especially of such a grade as to overcome the compensatory power of the heart, death generally takes place at an early period. Where there is transposition of the main arterial trunks, the infant usually lives but a short time. Infants and children with congenital disease of the heart are very apt to die suddenly.

Death ordinarily results from some affection of the lung, sometimes from hæmoptysis, and it is quite common for tuberculosis to develop in these cases of congenital cardiac disease.

In some rare cases the compensatory power of the heart is so great that the equilibrium of the circulation is maintained, and adult life may be reached.

Treatment.—The treatment of congenital disease of the heart is essentially hygienic and symptomatic. The infants should be carefully protected from atmospheric changes which would be likely to produce bronchial irritation, as in many cases bronchitis appears to play an important part in interfering with the maintenance of the equilibrium of the circulation and in destroying compensation. In a number of cases I have found that the administration of digitalis in small doses and with the greatest caution is valuable when hypertrophy has begun to fail and dilatation to increase. Where the dyspnœa is distressing, a few drops of aromatic spirit of ammonia will often give relief. Stimulants are usually indicated.

Freedom from excitement and over-exertion should be constantly enforced, but the child should be kept in the open air as much as possible.

Here is another infant (Case 493), three months old, in whom the most striking feature of its congenital cardiac disease is extreme wasting. It has a cardiac murmur at the base of the heart, and is, as you see, slightly cyanotic. You will notice that the cyanosis sometimes affects the mucous membrane of the mouth, and that the nails are blue. A harsh systolic murmur can be detected at the base of the heart. At times the infant has serious attacks of dyspnœa and suffocation, but by simply placing it on its right side immediate relief is obtained from these symptoms, this procedure evidently bringing into action a safety-valve by which some overtaxed portion of the circulatory mechanism is temporarily freed from its burden.

ACQUIRED DISEASES OF THE HEART.—Acquired diseases of the heart may be functional or organic, acute or chronic.

Functional.—Functional affections of the heart do not usually occur until the later years of childhood. Functional cardiac disturbances may signs of cardiac disease, such as murmurs, thrills, and dulness, as they are very similar to those with which you have been made familiar in your study of the adult's heart. The importance of recognizing the relative size and position of the heart at different ages I have already spoken of in my lecture on development (page 122), and I shall therefore refer you to what I said on that occasion. There are certain differences, however, between the symptoms of cardiac disease in infancy and early life and those in later life. In young children murmurs are more apt to be diffuse than in adults, often being heard over the entire chest; and the rate and rhythm of the heart are so easily disturbed by nervous influences as to be of little diagnostic value. Progressive emaciation is a symptom which is apt to appear speedily. An enlarged heart dependent on adhesions from a preceding pericarditis is more common in early life than in adults, while compensation, as I have already told you, is much more readily acquired.

I have had children with cardiac disease presented for treatment at my children's clinic one year with cardiac symptoms so severe that they had to be carried; they were emaciated and cyanotic, the area of cardiac dulness was increased, and souffles were present; yet these same children would return and be shown to the next class of students in the following year, walking up-stairs without dyspnœa, looking well nourished, of good color, with much less enlargement of the area of cardiac dulness, and with the cardiac souffle scarcely perceptible, showing that the cardiac compensation was complete.

As an illustration of this class of cases you will perhaps remember the little girl (Case 495) who was brought to the clinic by her mother to be shown as a child who was then well, but whose chances of living had seemed at one time very slight.

When first seen she was about five years old. She had never had any of the acute diseases, such as scarlet fever, diphtheria, pertussis, articular rheumatism, or in fact any disturbance except slight pains in her limbs. For the previous six months she had lost in appetite and weight, got out of breath very easily, suffered from palpitation, and in the beginning of her sickness was confined to her bed for a week or ten days with a high fever and pain referred to her left side. On examination she was found to be somewhat cyanotic. The area of visible cardiac pulsation was much increased. The apex of the heart was in the sixth interspace, 3 cm. (1⅛ inches) to the left of the mammary line. The area of absolute cardiac dulness extended to the right parasternal line, from the third to the fifth cartilage, and 1 cm. (⅜ inch) to the left of the mammary line on a level with the left nipple; the vertical area of dulness to the left of the sternum extended from the second to the sixth interspace. There was a loud mitral systolic murmur. The lungs were normal.

The chief points of treatment in this case were the careful administration of nourishing food and the enforcement of rest. She was always carried up and down stairs for almost a year. She grew worse for a time; she became irritable, and for some time when the cyanosis and orthopnœa were most marked she had a cough, and once or twice hæmoptysis. By the following winter, however, the general symptoms were much improved, and in another year the dyspnœa, cyanosis, palpitation, and pain had passed away. The apex of the heart was found to be in the fifth interspace in the mammary line, and the area of dulness very little greater than normal.

Cardiac symptoms dependent on organic lesions may arise, and yet no physical signs of such lesions be detected during life.

Mechanical.—The mechanical conditions in cardiac disease play a very interesting and important part in many diseases in infancy and early childhood, and by their results often interfere seriously with the general physical condition and normal development of the child. These abnormal conditions may result in hypertrophy or dilatation from over-exertion; they may occur at puberty; they may arise from direct mechanical interference with the heart's action, as from adhesions or from undue pressure on the cardiac cavities, as in pulmonary disease, pertussis, renal disease, and narrowing of the aorta. In all these diseases there is a greater liability that acute dilatation may take place in early life than that it may occur at a later period. You should therefore always remember to examine the heart carefully during the course of all these diseases. The processes which suddenly cause great increase of the blood-pressure in the lungs may lead to acute dilatation of the right ventricle, while where there is a diffuse renal disease, as in scarlet fever, acute dilatation of the left ventricle may take place, and be followed by hypertrophy, as I have explained to you in my lecture on scarlet fever (page 569, Case 245). In all these diseases this acute dilatation may take place rapidly and disappear almost as rapidly, a phenomenon which is somewhat characteristic of cardiac disease in early life.

I have already referred to the great changes which take place in the heart, and to its rapid growth, at the time of puberty. At this period the general growth of the child is apt to be very rapid, and symptoms of cardiac weakness commonly occur, especially in girls. These symptoms are debility, lack of energy, palpitation, and dyspnœa on exertion. There may also be signs of slight cardiac dilatation, and murmurs, probably hæmic in their nature. This period, therefore, is one in which cardiac disease from any cause, such as rheumatism, is of more serious import than at a later period, when the heart is not taxed by too rapid growth.

These cases should be treated by mild physical exercise, care being taken that the children do not over-exert themselves. Complete rest for two or three hours every day should be enforced. Under this treatment, combined with nutritious food and possibly a tonic of iron or nux vomica, the signs of cardiac disturbance usually soon disappear.

I must again remind you of the importance of mechanical interference with the action of the heart arising from adhesions. Adhesions of the pericardium or in its neighborhood are so latent in infancy in their symptoms that they are often overlooked until the mechanism of the heart has become so seriously interfered with as to present the symptoms of disease of the heart itself, such as dilatation or hypertrophy.

Inflammatory.—The chief inflammatory lesions of the heart are *endocarditis* and *myocarditis*.

Endocarditis.—The most common cardiac disease which occurs in children is endocarditis. Endocarditis may be acute or chronic, primary or secondary.

ETIOLOGY.—The elaborate investigations of J. H. Wright, W. R. Stokes, and others have shown that acute endocarditis is of bacterial origin. Weichselbaum has contributed more to our knowledge of this disease than any other investigator. He has proved that there is no essential difference between the various forms of endocarditis, either histologically or pathologically, and that no one species of bacteria is exclusively concerned in the production of the disease. Sometimes the streptococcus pyogenes is found, sometimes the staphylococcus pyogenes aureus, and sometimes the diplococcus pneumoniæ. We therefore no longer need make a distinction between simple endocarditis and ulcerative or verrucose endocarditis. There is merely a difference in the degree of the malignant nature of the especial organism which has produced the disease, or in the vulnerability to infection of the individual.

PATHOLOGY.—While the same lesions of endocarditis may be found in children as in adults, yet in infancy, although marked acute cardiac symptoms and murmurs frequently arise, the autopsy almost invariably fails to show any endocardial lesions or growths. In two thousand autopsies at the New York Foundling Asylum, Dr. Northrup and Dr. O'Dwyer never found an acute inflammatory lesion except in one case, which showed the lesions of acute malignant endocarditis. Where the lesions of endocarditis are found in children, the connective tissue and the basement substance are, according to Delafield and Prudden, principally concerned in the inflammatory process. The endocardium which forms the valves is that which is most frequently inflamed, but other portions of it are by no means exempt, In some cases there is swelling of the valves, which are thickened, their surfaces remaining smooth, the basement substance is swollen, and there is a moderate production of new connective-tissue cells. In other cases the growth of connective-tissue cells is very much more marked, the basement substance is broken up, and little cellular fungus-masses, called vegetations, project from the free surface of the endocardium. In still other cases the cellular growth in some places forms vegetations, and in others degenerates, and thus portions of the valves are destroyed. This is *simple acute ulcerative endocarditis.*

In some cases the children recover, and the valves seem to return to their normal condition, while in others the valves are left permanently damaged.

Chronic endocarditis may succeed an acute endocarditis, or the inflammation may be chronic from the onset. It affects most frequently the aortic and mitral valves and the endocardium of the left auricle and left ventricle, similar changes in the right side of the heart being much less frequent. In these cases the endocardium may be thickened and tense, and its surfaces smooth or covered with small, hard vegetations or ridges, or there may be a growth of connective-tissue cells in the endocardium, with a splitting up of the basement substance.

While endocarditis may be primary, simply arising from the infection the latter of which may amount to orthopnœa, increase. There is apt to be cough from an accompanying bronchial irritation, produced most frequently where there is obstruction at the mitral orifice, and, following a general venous stasis, enlargement of the liver, hæmoptysis, and œdema of the face, legs, and arms appear. Children show such a wonderful recuperative power that even in these advanced cases under proper treatment the serious symptoms may gradually pass away, and often such complete cardiac compensation takes place that they are left with no symptoms of cardiac disease except a murmur.

In endocarditis relapses are common and there is a great tendency to recurrence. Embolism may take place, and sometimes the first symptom of cardiac disease which has been noticed is a hemiplegia following a lesion of the mitral valve. Anæmia is a very common symptom, especially where endocarditis accompanies rheumatism. Congestion of the lungs, with resulting hæmoptysis, may arise where there is insufficiency of the mitral valve. Although when the valves are affected murmurs are usually present, yet sometimes where there are lesions of the valves murmurs cannot be detected. In endocarditis murmurs are most frequently heard in the region of the mitral valve, and insufficiency of the mitral valve is the most common of the inflammatory cardiac lesions in childhood.

DIAGNOSIS.—The diagnosis of endocarditis depends upon the physical signs. These signs are an increase in the area of cardiac dulness and a change in the cardiac sounds. The change in the area of cardiac dulness must be differentiated from that which occurs in a pericardial effusion, of which I shall speak later (page 1056).

The change in the cardiac sounds may be produced by changes in the blood or by organic lesions of the valves. The differential diagnosis between these two conditions is the same as in adults, and therefore I shall not dwell upon it. In insufficiency of the mitral valve the murmur is systolic, and is transmitted to the axilla and the back. In some cases the murmur of mitral insufficiency is closely simulated by a valvular sound produced in the course of pericarditis. Stenosis of the mitral valve is much less common than insufficiency. It is represented by a presystolic murmur heard in a limited area in the region of the heart's apex, and is sometimes accompanied by a reduplication of the cardiac sounds at the apex and by a thrill. Pain is said to be more common in connection with this lesion than with other cardiac defects. Lesions of the *aortic valve* are almost invariably associated with rheumatism. Stenosis of the aortic orifice is very apt to be associated with a mitral lesion. There is nothing especially characteristic in childhood of these lesions of the aortic valves, and the same may be said of lesions of the tricuspid valves.

Where endocarditis has become chronic and compensation has only partially taken place, the children are atrophic, anæmic, and have a tendency to imperfect circulation and to bronchitis. In some cases the fingers become club-shaped.

Prognosis.—The prognosis of acquired endocarditis in early life is very favorable. I have already referred to the great recuperative powers of the child, and in many cases, especially where it is the first attack, such complete compensation takes place that the child practically recovers. If it is the walls of the heart that are affected, the heart may regain its normal size and position. If the valves alone, or the valves and the walls, are affected, reaction can still take place. Death may, however, occur at the height of the attack, or the child may die later from exhaustion and sometimes suddenly from heart-failure.

Treatment.—The treatment of acute endocarditis during the early days of the attack is essentially rest in bed, and is otherwise symptomatic. From the very beginning, however, we must bear in mind that our treatment should be directed to establishing compensation. We should also remember that the younger the child the more likely it is that we shall have to contend with a resulting atrophic condition and anæmia. The child should be encouraged to sleep, in order that the circulation may be kept as quiet as possible and thus relieve the work of the disabled heart. The heart-beats of a young child during sleep are often reduced twenty in a minute, and thus sleep affords the best opportunity for compensation. The treatment which I have found most efficient in all forms of cardiac disease is absolute rest in bed for days or even weeks until compensation has become complete. Later the general health of the child should be carefully attended to by means of good food, pure air, and exercise of a mild type, never excessive. The surface circulation should be promoted by baths and gentle massage. Digitalis and iron are of great value, the former in aiding the establishment of compensation, the latter in combating the anæmia. If at any time during the course of the disease the attacks of dyspnœa are excessive, nitroglycerin can be given in doses proportionate to the age of the child; 0.0003 gramme ($\frac{1}{200}$ grain) can be given to a child three or four years old.

Although the more advanced pathological condition usually spoken of as "ulcerative endocarditis" rarely occurs in young children, yet it is at times met with. Its symptoms are obscure, and the diagnosis is rarely made during life.

I have here the organs of a child who has lately died in my wards with this disease.

This boy (Case 496), four years old, was attacked one month ago with fever, thirst, and pain in his knees. Later his feet became painful and swollen, and other joints were successively involved. He complained of pain in the back of his neck and along his spine. One week before entering the hospital he began to have moderate but incessant choreic movements, and he showed much incoördination of mastication and articulation.

A physical examination showed the lungs to be normal, the area of cardiac dulness somewhat increased to the left of the mammary line, and a murmur at the apex, with the first sound transmitted to the axilla and the back. On the following day a pericardial friction-sound was heard just above the left nipple, accompanied by præcordial pain. Two weeks later the choreic symptoms disappeared, and the temperature became normal. The area of cardiac dulness did not extend under the sternum, but was found to correspond to the impulse of the heart, which was 1.4 cm. (½ inch) outside of the left mammary line. During the last week of its life the child became very weak, had marked dyspnœa, and showed signs of effusion in the right pleural cavity, but presented no other symptoms. It died yesterday.

The post-mortem examination was made by Dr. Councilman.

Both pleural cavities contained a considerable accumulation of blood-stained fluid. The anterior mediastinum was deeply injected and reddened, and the mediastinal lymph-glands beneath the sternum were enlarged. The apex of the pericardium was tightly adherent to the left pleura, and about this point the tissues were thickened, deeply injected, and œdematous. The right lung was adherent to the pleura by comparatively fresh adhesions. Here and there over the pleural surface of the lung is a slight fibrinous exudation. The lymphatics over the surface of the pleura are greatly dilated. The upper lobe of the right lung is congested. Small nodular masses can be found beneath the pleura, and on section, as you see, there is a distinct lobular consolidation throughout the upper lobe of the right lung. The solid portion of the lung is of a dark-red color and comparatively smooth on section. Muco-purulent matter can be squeezed from the larger bronchi. The small consolidated areas are more or less separated from one another, and between them are cavities in the interlobular septa. The appearance of the lung is somewhat similar to that presented in bovine pleuro-pneumonia. The bronchial glands are enlarged and reddened. The left lung was not so adherent as the right. All over the posterior portion of the pleura there was a slight fibrinous exudation. This lung has been somewhat compressed by the accumulation of fluid in the pleural cavity, but otherwise shows about the same condition as the right lung, the consolidation being in the posterior portions principally. The pleural cavity, as you see, is obliterated by the adhesions. The parietal pericardium is greatly thickened, and in and between the connective-tissue adhesions there is a thick fibrinous exudation. The heart is somewhat enlarged. At the apex of the left ventricle, at a point corresponding to the adhesions of the pericardium, the myocardium feels soft and is somewhat whiter than the remainder of the tissue. The interior of the right heart contains tolerably firm, fresh clots. The myocardium of the right side of the heart is pale and soft. Along the free border of the right auriculo-ventricular valve there are a few fresh vegetations. The left side of the heart is dilated. The edge of the mitral valve is thickened and eroded. There appears to be a slight loss of substance in the thickened portion of the valve, and the edges are irregular and eroded. The muscular substance of the heart is generally pale. Beneath the endocardium there are small, whitish points. Similar points are seen on the papillary muscles and on the inner side of the auricle. The aortic valves are intact, except for a few fibrinous deposits just at the edges of contact. The coronary arteries are normal.

The spleen is enlarged and comparatively soft. The mesenteric lymph-glands are enlarged and also slightly soft. The liver is large, dark red in color, and the lobules are prominent.

The left jugular vein is filled by a firm, adherent thrombus, which extends downward into the subclavian vein, the innominate, and the superior vena cava, and completely obliterates those veins.

A microscopic examination of the lungs shows a distinct lobular pneumonia. The alveoli contain very little fibrin, but are filled with large, pale cells. Among these are a few leucocytes, but usually the leucocytes are conspicuously absent. The consolidation is quite general, comparatively few of the alveoli in the most affected portions being free. At numerous places in the lung there are wide passages, apparently lymphatics, filled with fibrin and large, pale cells similar to those in the alveoli. The bronchi are in most cases free. The lung consolidation does not appear to take its point of departure from the bronchi.

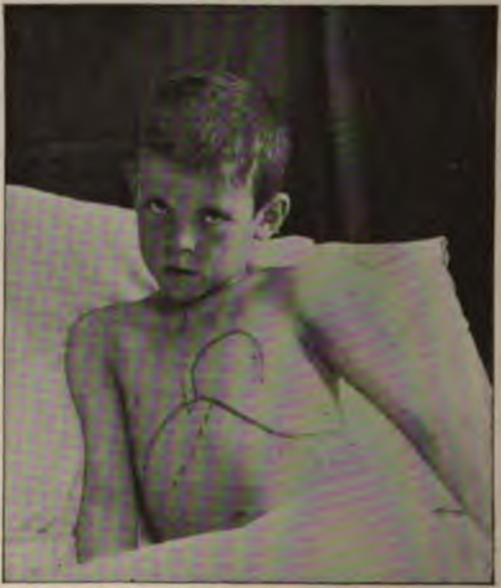
Typical masses of streptococci are found in the alveolar contents and in their walls. The lymphatics of the pleura are enlarged, and correspond to the large passages just described in the lung. Sections of the bronchial and cervical lymph-glands show acute swelling of the glands, with micrococci here and there in the sinuses.

A microscopic examination of the heart showed the vegetations on the mitral valve to be distinctly verrucose. Here and there on the ends of these vegetations were small masses of fibrin. Only in places was there a direct infiltration with leucocytes. Streptococci were found on the edges of the vegetations, chiefly in the fibrin. In but one place were they found within the tissue. Sections of the myocardium embracing the pericardium showed a fibrino-purulent exudation on the pericardial surface. Numbers of streptococci were found in the fibrinous exudation and in a few places on the edge of the cardiac muscle. Sections from the left ventricle showed a marked adhesion with the pleura and an acute inflammation with a few streptococci in the tissue. The thrombi in the large cavity showed no evidence of organization, and no streptococci were found in them, but there were numbers of them in the perivascular tissue, which showed slight purulent infiltration.

Sections of the liver, kidney, and spleen showed no pathological condition save a slight, cloudy swelling, and no organisms were found in these tissues.

Cultures made at the autopsy gave a pure culture of streptococci from the lungs, from the pericardium, and from the bronchial lymph-glands. In the spleen only a few colonies were found. The other organs were sterile.

CASE 497.



Acute endocarditis. Mitral insufficiency. Male, 8½ years old.

As Dr. Councilman says, the most interesting part of the autopsy is the manner of infection. He thinks that the heart must have become infected before the lungs, so that apparently this is a case of primary endocarditis of the malignant form. It seems very probable to Dr. Councilman that the path of the infection was from the heart to the pericardium, thence to the mediastinum, producing the thrombus of the veins, and probably thence to the lung, possibly by means of the thrombosed veins. The thrombi seem to have been due to an inflammation of the wall of the vein, produced by the streptococci in the perivascular tissue. From this point they could have got into the veins, the infection being carried thence into the lungs. The pneumonia in the lungs is entirely different from the ordinary broncho-pneumonia of infants, which is due to aspiration, and in which the chief seat of the disease is in the bronchi and the surrounding lung-tissue. In this case, however, the bronchi are less involved than other portions of the lung.

I have here in the wards a number of cases which illustrate the various types of cardiac disease.

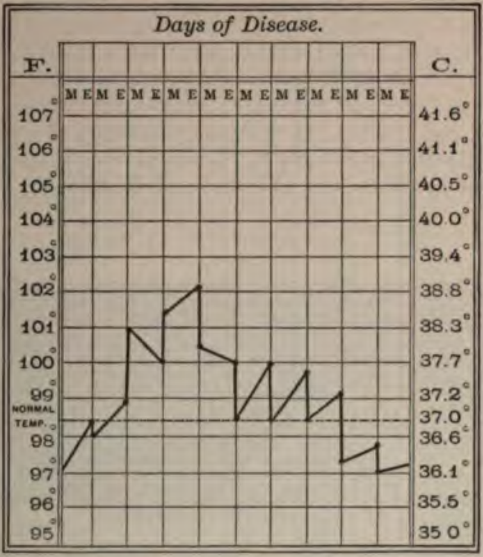
This boy (Case 497, page 1036), eight and a half years old, was well until nine weeks ago. He had never had any diseases, except measles and varicella when he was four years old. Nine weeks ago he was attacked with chorea, which lasted for about seven weeks and was succeeded by symptoms of dyspnœa on exertion, loss of appetite, and slight cough. There has been no history of rheumatism in the case. You see that he is cyanotic, but otherwise looks comparatively well.

There is no œdema, and an examination of the lungs detects nothing abnormal. The impulse of the heart is in the left mammary line in the fifth interspace. The cardiac area of absolute dulness is as I have indicated with this black curved line. I have also marked the lower border of the ribs with a plain black line, and have shown the slightly enlarged liver and spleen with a broken line. The dulness does not extend beyond the middle of the sternum, but is increased in the vertical line as high as the second interspace and extends slightly beyond the left mammary line. There is a marked systolic murmur, heard most loudly at the apex, and transmitted to the axilla and the back, also to the base of the heart.

This appears to be a case of acute endocarditis arising during an attack of chorea. The prognosis is good, as the child is already improving.

(Subsequent history.) The child was treated simply by rest in bed, and a month later his general symptoms improved, the areas of splenic, hepatic, and cardiac dulness were much decreased, and the cardiac murmur was not so distinct. Two weeks later compensation was apparently established, he had gained in weight, his color became better, and he left the hospital in good condition.

CHART 45. (CASE 498.)



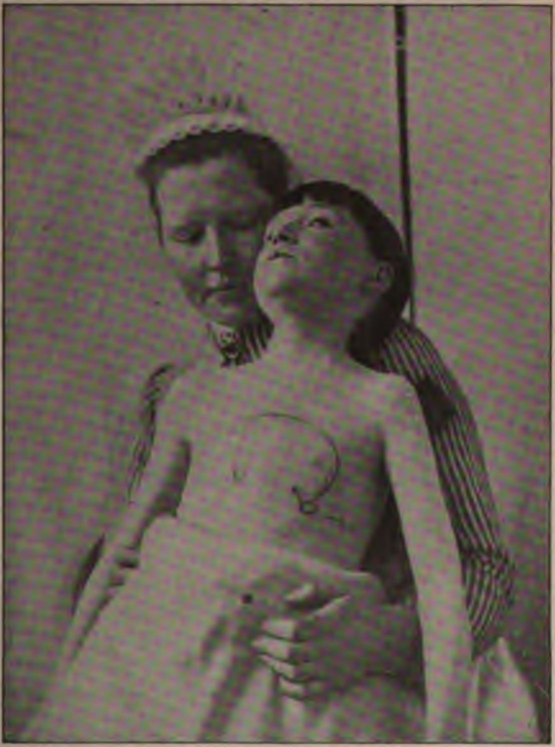
Acute endocarditis. Female, 9 years old.

I have here a girl (Case 498, I., page 1038), nine years old, who, although she has always been a delicate child, never had any especial disease until two weeks ago, when she was attacked with fever, palpitation, cough, and a rapid, irregular pulse. On entering the hospital she was cyanotic, the face and extremities were cold, and there was considerable prominence over the cardiac region. The resonance of the lungs was normal, but there were a few moist râles at both bases. The impulse of the heart was in the fifth left interspace, 1.4 cm. (½ inch) outside of the mammary line, and there was a marked thrill with a systolic murmur transmitted to the axilla and heard distinctly in the back. The liver was slightly enlarged. Here is the temperature chart (Chart 45), showing the temperature during the acute inflammatory stage of the endocarditis.

toms of dilated heart with a lack of compensation as you see in this little girl. While he was sleeping in the same position which she has assumed since I have been describing her case to you (Case 498, II., page 1038), he died suddenly. He had been subject to sudden violent attacks of dyspnœa, and once or twice while in the hospital had an attack of angina pectoris.

This next child (Case 500), a girl, thirteen years old, has a history of pertussis some years ago, but has not had any other disease except an attack of rheumatism two years ago. Since then she has occasionally had attacks of dyspnœa when at play and when going upstairs. She has also at times had œdema of the feet. One week ago she complained of pain in the cardiac region, so severe as to interfere with her sleep. On entering the hospital she had a temperature of 38.5° C. (101.2° F.), a pulse of 104, and respirations 65. An examination showed nothing abnormal except in the cardiac region.

CASE 500.



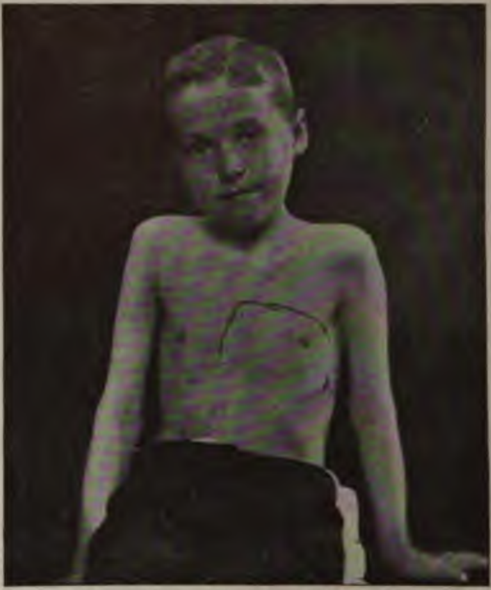
Chronic endocarditis following rheumatism. Mitral stenosis and insufficiency. Anæmia. Female, 13 years old.

The impulse of the heart is in the fifth left interspace in the mammary line. The area of absolute cardiac dulness is enlarged, and I have represented it by a black curved line. You see that it extends beneath the sternum, and at the junction of the upper border of the fourth rib extends a short distance to the right of the sternum. The upper boundary, as I have said, is the upper border of the third rib, and is about 5.3 cm. (2 inches) outside of the mammary line. There is a presystolic murmur at the apex, which is confined to a limited area. There is also a systolic murmur at the apex transmitted to the axilla and the back; the pulmonic second sound is accentuated.

(Subsequent history.) Four weeks later, after being treated by complete rest in bed, the cardiac symptoms almost entirely disappeared, the area of cardiac dulness was much diminished, and the murmurs were less distinct. Two weeks later the child left the hospital, much improved in her general health, but in a very anæmic condition.

This next boy (Case 501), nine years old, had an attack of rheumatism when he was six years old.

CASE 501.



Chronic endocarditis following rheumatism. Mitral insufficiency. Pericarditis sicca. Dilated heart. Pneumonia. Pleurisy. Male, 9 years old.

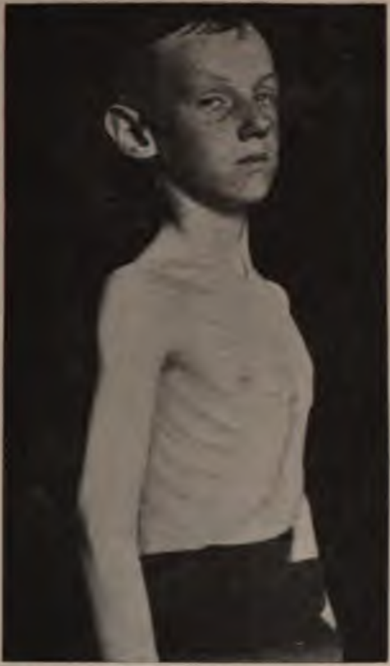
Four weeks before entering the hospital he began to have swelling of the feet, and four days before entrance swelling and pain in the cardiac region and much dyspnœa and general discomfort. His respirations and pulse were much quickened, and his temperature was raised. On entering the hospital, a physical examination showed that he had pneumonia of the left lung. A pericardial friction-sound was also heard in the second left interspace. The cardiac area of absolute dulness extended as far as the right parasternal line, as high as the third rib, and 5.3 cm. (2 inches) beyond the left mammary line. There was a soft systolic murmur at the apex. The pneumonia involved the whole of the left lung, and was complicated by a moderate pleuritic effusion. Resolution took place, however, and the fluid was absorbed. The cardiac symptoms improved as soon as the pneumonia and pleurisy disappeared, but the physical signs of the dilated heart have not yet changed. For some weeks I shall enforce absolute rest in bed, as this morning he was suddenly attacked with extreme dyspnœa, cyanosis, and collapse, which followed his getting out of bed and dressing himself contrary to my directions.

I have indicated the area of absolute cardiac dulness in black, which shows an enlarged heart, as you will understand better when I describe the dulness produced by a pericardial effusion. There is no visible impulse of the heart, and the beat can scarcely be found on palpation. The increased area of dulness is therefore practically caused by dilatation rather than by hypertrophy, and this supposition is substantiated by the symptoms of lack of compensation which he has shown.

This boy (Case 502, page 1041), eleven years old, had measles when he was an infant, diphtheria when he was three years old, and pertussis when he was four years old. He had always been well until one and a half years ago, when, after indefinite pains in his joints, accompanied by no swelling and not sufficiently severe to confine him to bed, he began to have dyspnœa on exertion, and cardiac pain. He is somewhat cyanotic, and has lately lost a great deal in weight. There is no œdema, and nothing else abnormal is detected except in the examination of the heart, which shows the area of absolute dulness to be somewhat increased. A loud presystolic murmur is heard at the apex, limited in its extent and accompanied by a thrill. He has also had a cough. He seems to represent a case of stenosis of the mitral valve. There is, as you see, decided enlargement in the cardiac region to the left of the sternum.

(Subsequent history.) After remaining in the hospital for two months and being treated by rest, compensation was established, and he left the hospital in good condition.

CASE 502.



Chronic endocarditis. Mitral stenosis. Bulging of left side of sternum. Male, 11 years old.

Since then he has returned from time to time with a renewal of the symptoms of cyanosis, dyspnœa, and lack of compensation.

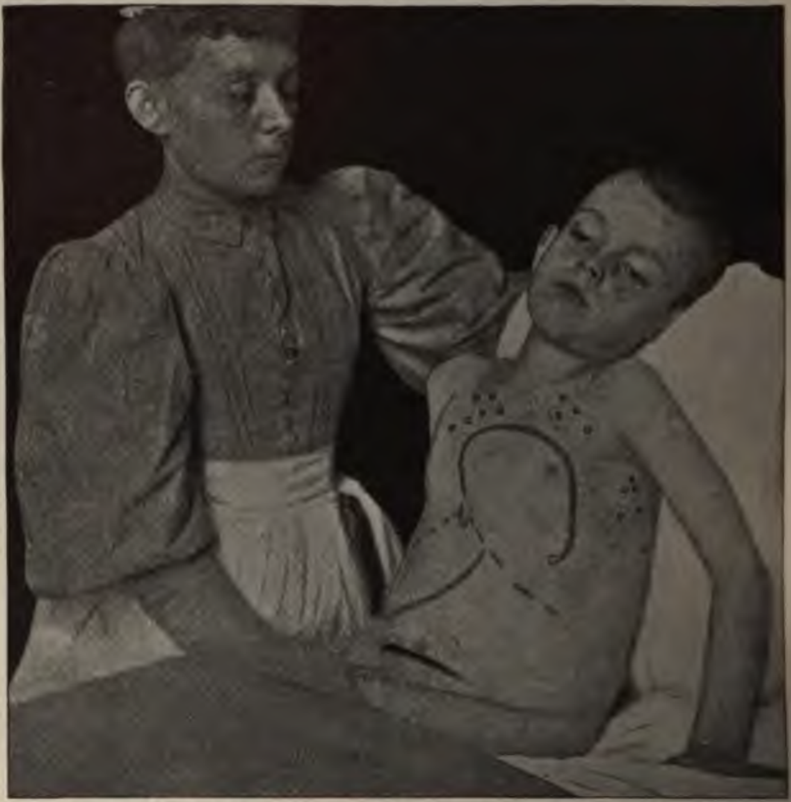
This next boy (Case 503, I., page 1042), ten years old, is interesting as illustrating certain characteristics of cardiac disease in early life.

Two years ago he entered the hospital with marked œdema of the face, body, and limbs, ascites, a slight amount of fluid in both pleural cavities, and œdema of the lungs. There was no definite history of rheumatism nor any other cause for the cardiac disease which was causing these symptoms, and which had apparently developed insidiously, though if he had been under closer observation a definite period of onset would probably have been discovered. The impulse of the heart was found to be 1.4 cm. (½ inch) outside of the mammary line in the fifth left interspace. The area of cardiac dulness was somewhat increased. There was a loud systolic murmur at the cardiac apex transmitted to the axilla. The second pulmonic sound was much accentuated. Here is a picture (Case 503, II., facing page 1042) taken at that time, and showing the marked œdema of the legs and the much distended abdomen. He was treated by complete rest in bed for five weeks, and in the beginning digitalis was administered until the urine, which was lessened in quantity, had increased
[illegible] lungs had disappeared. On entering the hospital the ascites was
abdominis. Under this treatment the child rapidly improved,
ared, the liver returned to its normal size, the area of cardiac
ased, the cardiac murmur became less marked, and six weeks
hospital complete compensation was established and he
rell. This picture (Case 503, III., facing page 1042),

was taken just before he left the hospital, and, as you see, is in marked contrast to the picture taken on his entrance.

CASE 503.

I.

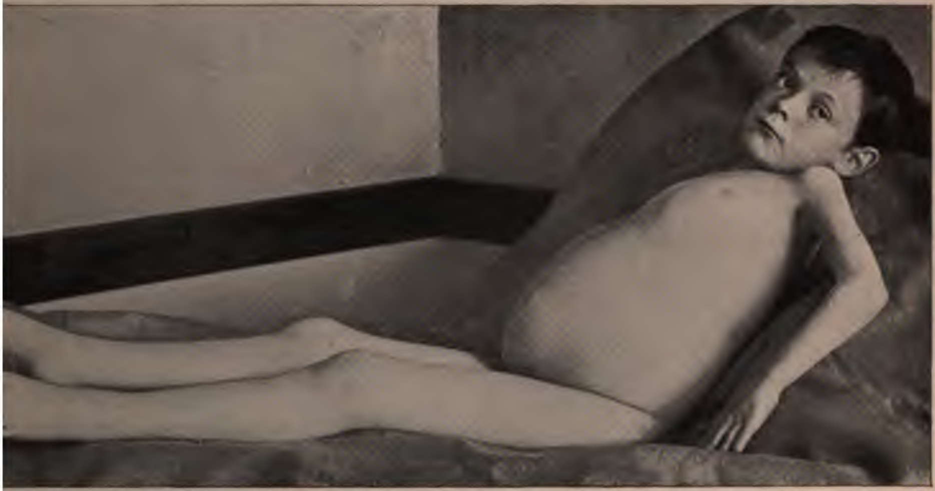


Chronic recurrent endocarditis. Mitral insufficiency. Disturbance of compensation. Dilated heart. Enlarged liver. Œdema of lungs. Ascites. Male, 10 years old.

Since leaving the hospital the boy is reported to have been very well, except that he could not play or work hard. Two weeks ago he was attacked with fever, præcordial distress, and cardiac pain; later he began to have œdema of the feet and dyspnœa. Since then he has been growing progressively worse, and his case illustrates a fresh attack of endocarditis supervening on an old chronic endocarditis (endocarditis recurrens) and resulting in a disturbance of the previous compensation. You see that he has orthopnœa to such an extent that he is unable to lie down in bed, and that he has to be continually watched by a nurse, as he frequently has attacks of excessive paroxysmal dyspnœa which are liable to prove fatal. There are cyanosis of the lips and hands and marked general œdema. The skin of the nose and extremities is cold. The impulse of the heart is felt in the sixth left interspace 2.8 cm. (1 inch) beyond the mammary line. The area of cardiac dulness extends beneath the sternum, and at the third intercostal space extends 1.4 cm. (½ inch) to the right of the sternum, thence upward in a curved line across the upper part of the sternum to the second rib, and then, keeping outside of the mammary line, descends and joins the point of cardiac impulse. There is a loud systolic murmur, heard most distinctly at the apex, but transmitted over the whole cardiac area and through the axilla to the back. The second pulmonic sound is accentuated. The aortic sounds are weak. There are numerous moist râles heard in all parts of the lungs. The percussion of the lungs is resonant everywhere except in the lower parts, where there seems to be a slight amount of fluid in both

Case 503.

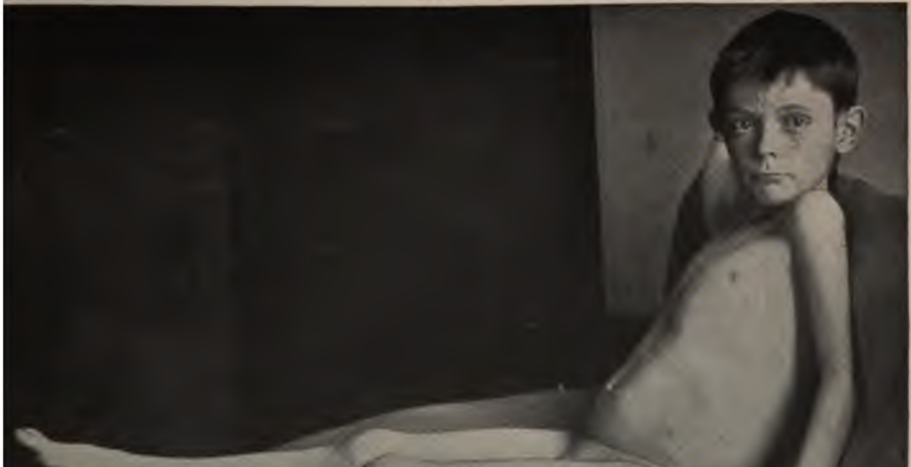
II.



Chronic endocarditis. Mitral insufficiency. General œdema and ascites. (Before treatment.)

Case 503.

III.



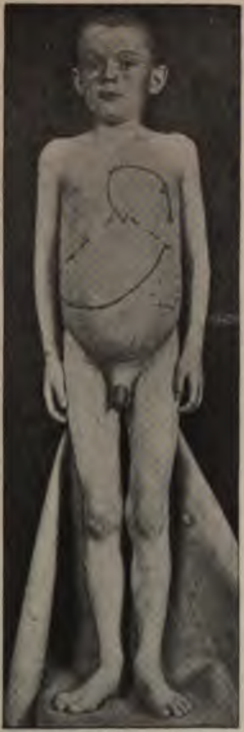
Chronic endocarditis. Complete compensation. (Six weeks after treatment.) Male, 10 years old.

pleural cavities. The liver is enlarged so that it extends 7.8 cm. (3 inches) below the margin of the ribs. Ascites is present, the fluid rising to about the line of the umbilicus. The spleen is normal in size. The child is passing only a small amount of urine, which contains a trace of albumin. I have marked the cardiac and hepatic areas of dulness and the upper border of the ascites by black lines, the margin of the ribs by broken lines, the point of cardiac impulse by a black ring, and the œdematous râles in the chest by smaller black rings. The prognosis in this case, although from the child's present condition very serious, as he is liable to die suddenly at any time if extra blood-pressure should be brought to bear upon the dilated and crippled heart, is not entirely unfavorable, as he has previously shown such great powers of compensation and recuperation. As there is no great distention of the abdomen, I shall not at present remove the ascites by paracentesis, but shall have the child carefully watched, and, if the ascites increases, shall have it removed at once. He is taking infusion of digitalis, 3.75 c.c. (1 drachm), every three hours, and diuretin, 0.36 gramme (6 grains), once in six hours as a diuretic. His diet is milk.

(Subsequent history.) Within forty-eight hours rapid relief was obtained from the urgent symptoms, and at the end of three weeks the œdema of the lungs, the general œdema, and the ascites had disappeared entirely. The urine became normal in quantity and free from albumin. One week later he was well enough to be out of bed for an hour each day, and at that time this picture was taken (Case 503, IV.), which shows the heart and liver to be still enlarged.

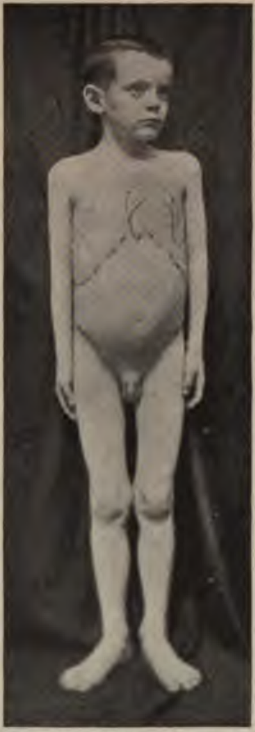
CASE 503.

IV.



Chronic endocarditis. Mitral insufficiency. Returning compensation. Enlarged liver. Enlarged heart.

V.



Chronic endocarditis. Mitral insufficiency. Broken line indicates enlarged heart. Black line indicates area of cardiac dulness with returned and complete compensation.

Some weeks later the liver regained its normal size, and still later the cardiac area of dulness was found to be much reduced and in the vertical line almost normal. This picture (Case 503, V.) shows the enlarged heart, which remained longer than the enlarged liver, and is represented by a broken line; the area of dulness of the heart as it appeared when he left the hospital is shown by a black curved line.

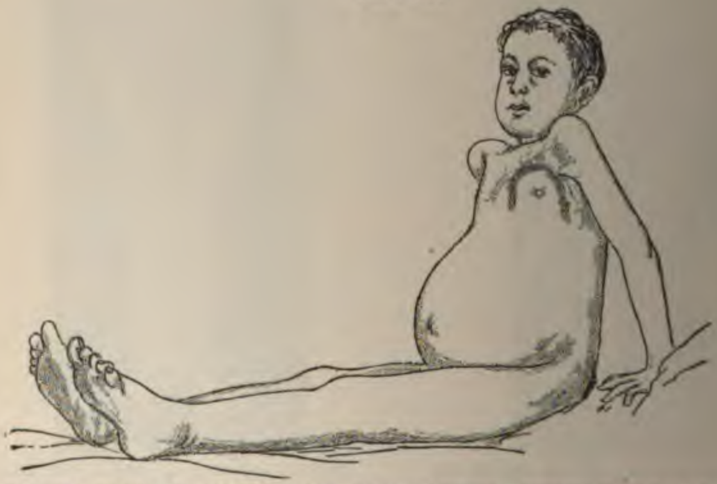
This next case, a girl (Case 504), nine years old, is instructive as showing the difference between the cardiac area of dulness produced by an enlarged heart and that produced by a distended pericardium. She had pertussis when she was three years old, and measles when she was seven years old. This was followed by an attack of rheumatic fever, which lasted six weeks. So far as I can ascertain, she had no cardiac disturbance at that time, and recovered completely from the attack of rheumatism. Two months later she had another attack of rheumatism, which was accompanied by pain in the cardiac region. She then apparently recovered, but one year later had a recurrence of the cardiac disturbance, which was, however, of short duration. From that time she remained well until four months ago, when she had a severe attack of bronchitis, and since then she has been failing in strength and has suffered from dyspnœa. Three weeks ago she began to have œdema of the feet and of the abdomen, and this has been progressively increasing. She is very anæmic, and, as you see (Case 504, I., facing page 1044), the œdema of the face and legs is marked. The distention of the abdomen is found to be produced by ascites. An examination of the heart shows the apex-beat to be in the sixth interspace, 4 cm. (1½ inches) beyond the mammary line. The area of absolute dulness extends from the third left costal cartilage downward across the sternum to 2.8 cm. (1 inch) beyond the right parasternal line in the fifth interspace. It also extends to the left and downward outside the mammary line until it joins the impulse of the heart in the sixth interspace. This area of dulness is not that which we meet with in a dilated heart alone, as I shall presently explain to you. On the contrary, it suggests that there is fluid in the pericardium.

In connection with the general œdema and absence of symptoms of pericarditis there is probably present the condition called hydropericardium. The liver is also enlarged. The pulse is regular, 140. There is a systolic murmur at the apex of the heart.

She is being treated by absolute rest in bed, a milk diet, infusion of digitalis, and diuretin.

(Subsequent history.) After she had been in the hospital for forty-eight hours the hydropericardium disappeared, the skin became less tense, the urine increased in amount, and there was rapid improvement in all the general symptoms. Two weeks later the œdema and ascites disappeared entirely, as is shown in a picture (Case 504, II., facing page 1044) taken at that time. The enlarged heart at that time is indicated by a broken line, while

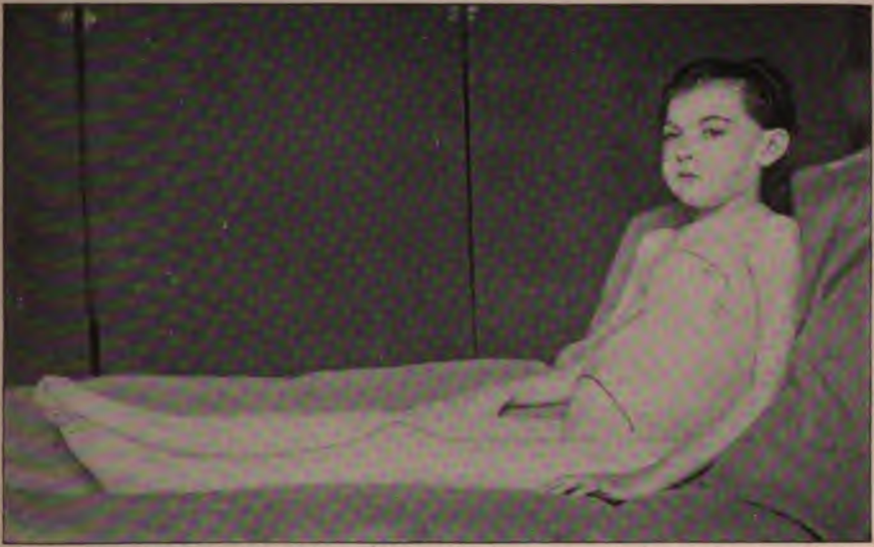
Case 505.



Chronic endocarditis. Greatly dilated heart. General œdema. Extreme distention of abdomen with ascites. Female, 11 years old.

Case 504.

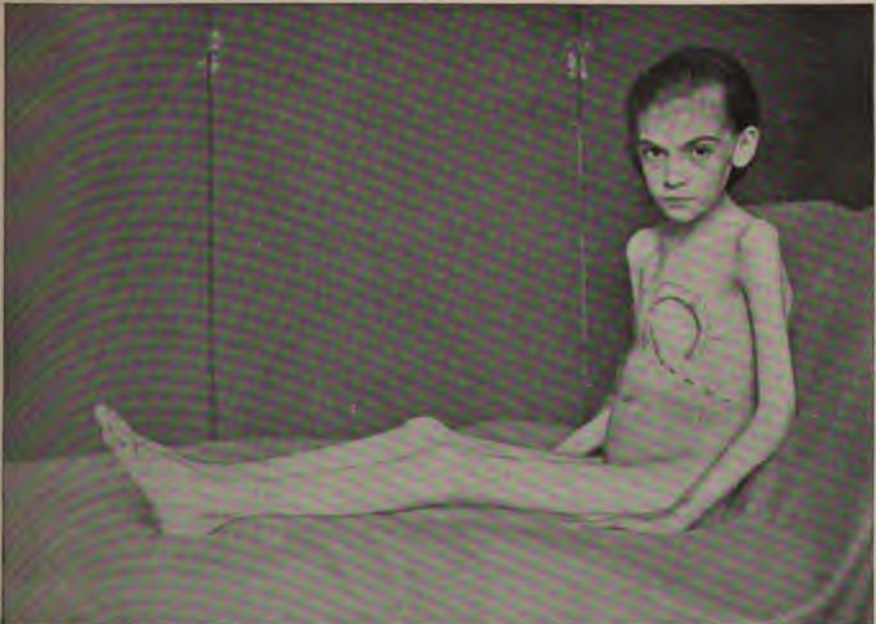
I.



Chronic endocarditis. Mitral insufficiency. Hydropericardium. General œdema and ascites. The line of ascites and of the cardiac area of dulness marked in black. (Before treatment.)

Case 504.

II.



Chronic endocarditis. Enlarged heart. Emaciation. (Two weeks after treatment.) Female, 9 years old.

the area of dulness, which was found some weeks later when compensation was established, is represented by a black line. The child was left in a very anæmic and emaciated condition, but the liver resumed its normal size, the area of cardiac dulness gradually became smaller, compensation was finally established, and she left the hospital in good condition.

Here is a girl (Case 505, page 1044), eleven years old, who has just been admitted to the hospital.

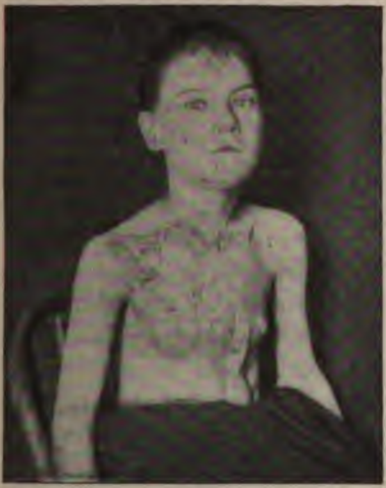
She shows, as you see, extreme dyspnœa, orthopnœa, cyanosis, marked general œdema, and great distention of the abdomen produced by ascites. Percussion of the chest shows extreme dilatation of the heart over an area which includes the entire sternum from the second interspace and extends 7.8 cm. (3 inches) to the left of the mammary line, the impulse of the heart being in the seventh interspace. There is also extensive œdema of the lungs. The pulse is weak and feeble. It is very evident that there is an entire lack of compensation in this case, and that, unless the heart is quickly relieved, cardiac failure will take place and the child will die. I have therefore told the mother that paracentesis abdominis must be performed at once.

(Subsequent history.) The mother refused to have paracentesis performed, and took the child home: it died suddenly on the following day from heart-failure.

I have already referred to the deformities which may arise in the chest from the pressure of an enlarged heart during a period when the thoracic walls are still pliant and undeveloped.

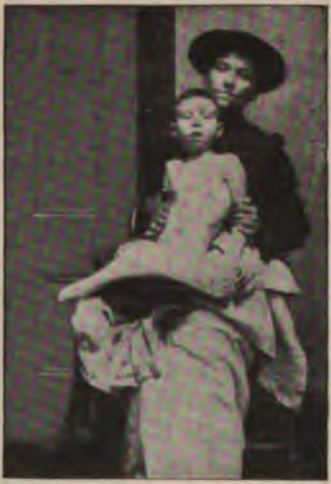
Here is a little girl (Case 506) who six years ago had an attack of rheumatism followed by endocarditis, and, although compensation has taken place and she is fairly well and strong, you see the displacement of the sternum and of the costal ends of the left ribs which has resulted from the cardiac enlargement.

CASE 506.



Displaced sternum and costal cartilages from enlarged heart. Female, 10 years old.

CASE 507.



Malformation of left side of thorax from cardiac disease.

Here is another case (Case 507) of cardiac disease, in which the endocarditis with its resulting cardiac dilatation and hypertrophy occurred at a still earlier period of life, and, as you see, there is great deformity of the left side of the thorax produced by the intrathoracic pressure.

LECTURE LII.

DISEASES OF THE PERICARDIUM.

THE anatomy of the infant's pericardium, so far as I have been able to determine by the dissection of sixteen infants of various ages, appears to approximate so closely that of the adult that there is nothing distinctive to note concerning it. The amount of fluid which normally occurs in an infant's pericardium, although of variable quantity, is probably under 5 c.c.

The chief diseases which affect the pericardium are hydropericardium, hæmopericardium, pneumopericardium, and pericarditis. The first three are very rare in early life, and therefore need be merely referred to. Absence of the pericardium may occur, and may be complete or partial.

PERICARDITIS.—The most common disease of the pericardium is pericarditis. It can occur at all ages, but the earlier the age the less often is it met with. It has been found in the fœtus and in the new-born, and well-marked adhesions of the pericardial surfaces have been observed in an infant which died thirty-six hours after birth.

ETIOLOGY.—There are a number of organisms which seemingly give rise to pericarditis. The most common of these is the micrococcus lanceolatus. In the new-born pericarditis may be the result of a septic condition following infection of the cord. At times it follows periostitis and ostitis in young children, here also probably being associated with septic infection. Traumatism may also be a cause of pericarditis. Rheumatism, especially after the third or fourth year of life, gives rise to as much periendocardial disease as at a later period. The inflammatory lesions may arise before the rheumatism has appeared elsewhere, and the intensity of the arthritic pain and the number of joints affected do not correspond to, or rather do not influence, the frequency of the pericardial complication. Inflammation of the pericardium is also frequently associated with pneumonia. It may be secondary to any of the eruptive fevers, but occurs most frequently as a complication of scarlet fever. When it occurs in this latter disease it appears usually in the second or third week of the attack. The pericardium also shows an especial tendency to invasion by the bacillus tuberculosis following tuberculosis of the pleura.

PATHOLOGY.—Pericarditis may be circumscribed or diffuse, and there appears to be no essential difference between the pathological conditions affecting the pericardium in early life and those which occur later. The pericarditis sicca of the adult is comparatively unusual in the child, in whom, as a rule, an effusion of greater or less extent almost always takes place. The effusion may be sero-fibrinous, hemorrhagic, or purulent. Not only is the tendency to effusion in the child greater than in the adult, but its formation is characterized by greater rapidity and it is more likely to be purulent than in the adult. A pericardial effusion tinged with blood is not uncommon in early life, and is not necessarily so significant of tuberculosis as is a pronounced hemorrhagic effusion. The white, opaque thickenings of the inner pericardial surface so frequently found in adults are rare in children, but have been found at all ages, and where there is a deformity of the chest, as in certain cases of rhachitis, they have been especially noticed. Tuberculosis of the pericardium as a primary disease is even more rare in the child than in the adult. Tuberculosis secondary to tubercle of the pleura may occur, especially when the left pleura is affected. The younger the subject the less likely are there to be adhesions between the pericardium and the pleura, an important fact, to be taken into consideration later when I shall speak of the diagnosis of pericardial effusion in infancy.

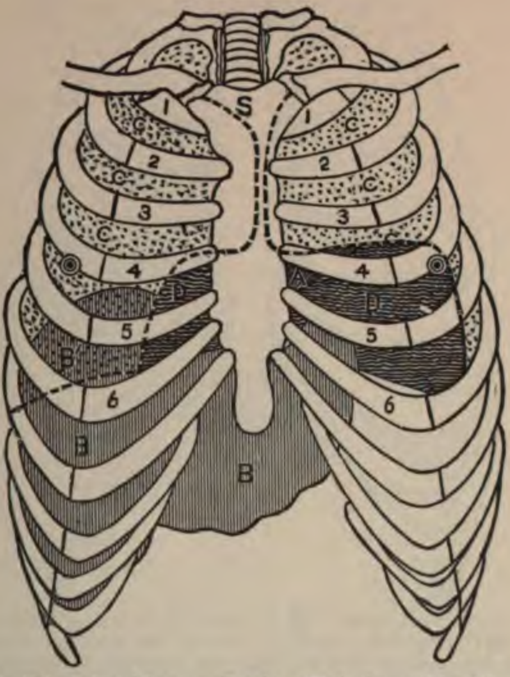
SYMPTOMS.—Pericarditis may be acute or chronic, primary or secondary.

The subjective symptoms of acute pericarditis in infancy are very indefinite, and throughout childhood this latency of the early symptoms is so marked and occurs so frequently that it may be said to be characteristic of the symptomatology of pericarditis in early life. It is so difficult to locate pain when it occurs in the infant, and a tumultuous action of the heart with general circulatory disturbance is so commonly the result of a diseased condition outside of this central organ, that it is impossible to formulate a practical general symptomatology for the onset of the disease. When, however, the disease has progressed, dyspnœa and orthopnœa become marked. Large effusions appear to affect the functional activity of the heart more rapidly in children than in adults, and to occasion earlier the signs of disturbance of the circulation. Diminution in the amount of the urine in cases of pericardial effusion, with a corresponding increase in the urine as the effusion decreases, has been noticed in children. The usual physical signs supposed to be characteristic of pericarditis are often very misleading, and where a pericardial friction-sound is absent the determination of a case of pericarditis in a young child may present great difficulties. Owing to the flexible thorax of the child, there is a greater opportunity for the neighboring parts to yield before the pressure of an effusion, and we are more likely to have bulging of the intercostal spaces, and on inspection a visible alteration of the cardiac area, than in adults. Because of the small size of the child's thorax, the heart and pericardium are much nearer to the anterior surface of the thoracic cavity than they are in adults. This occurs both normally and in diseased conditions, especially where there is flattening, and thus levelling, of the chest. Under these latter conditions the heart and pericardium are brought in such close contact with the thoracic wall that on palpation you can feel the heart's impulse, and on auscultation the heart-sounds, in a much more advanced stage of a pericardial effusion than would be possible in the adult with a proportionately large amount of fluid. It has also been noticed in early life that on auscultation the sounds in pericarditis and endocarditis simulate each other quite closely. Percussion is the most important physical sign, when the initial friction-sound has escaped detection, both for determining whether an effusion is present and as a guide to the prognosis and treatment. In effusions of exactly the same amount the area of dulness may differ, owing to the difference in the elasticity of the lungs and to the presence or absence of adhesions. The greater the elasticity of the lungs and the fewer the adhesions the more regular will be the outline of absolute dulness and the greater its significance as compared with that of the relative dulness; while the reverse of this proposition is true of the relative dulness. By *absolute dulness* I mean *entire absence* of resonance. By *relative dulness* I mean *diminished* resonance. The absolute dulness is determined by the retraction of the borders of the lungs, which withdraw from the chest-walls as the effusion gradually distends the pericardium. Relative dulness is due to the distended pericardium, and this to a greater or less degree compresses the lungs, which may be held in position by adhesions. The relative dulness, therefore, with its necessarily irregular outlines, varying according as adhesions are present or not, is most useful in studying complicated cases, while the absolute dulness should be relied upon in determining the outlines of the typical uncomplicated cases. The older the individual the more likely are adhesions and pulmonary diseases to be present. These will either prevent the retraction or alter the elasticity of the lung. The infant, being less likely to have had previous lesions of the lung and pericardium, gives us the best opportunity for studying the outlines of a pericardial effusion, and the area of absolute dulness is the most valuable physical sign of effusion in infants and in young children. It is evident, therefore, that we must acquire a precise knowledge of the uncomplicated cases before we shall be prepared to diagnosticate those in which adhesions are present or which are complicated by pulmonary disease. There is a great probability that many of the clinical observations on pericardial effusions made on adults by various competent clinical observers have been rendered of little practical value by the presence of adhesions, as has been proved by the difficulty in making a diagnosis by rules deduced from these observations. The number of clinical observations on infants corroborated by post-mortem examinations is not yet large enough to provide us with sufficient data from which to make precise deductions, but the experiments on which are based the diagrams of pericardial effusion which I am about to show you were made on sixteen infants, in none of whom did adhesions exist. In all of these presumably typical cases absolute dulness was found to the right of the sternum. An instance of how the area of dulness varies in complicated cases was given by a case in which, although the pericardium was much distended with fluid, it failed to show dulness to the right of the sternum, and the autopsy revealed adhesions binding the lung tightly to the right edge of the sternum. In this case the effusion was behind the lung, and this permitted resonance to be obtained over an area where in an uncomplicated case with the same amount of effusion there would have been dulness.

In addition to the difficulties in making a differential diagnosis arising from interference with the contractility of the lungs, such complications as pneumonia of the right lung, especially its middle lobe, pleuritic effusion on the right side, an enlarged liver, and an enlarged heart must be considered. Where this pneumonia, or pleurisy, or hepatic enlargement is present, an effusion into the pericardium cannot be diagnosticated by means of percussion, but these diseases can usually be readily determined by their especial symptoms. The differential diagnosis, on the contrary, from an enlarged heart, especially a dilated heart where the murmur may be absent, can often be made only by means of percussion.

Experiments with artificial effusions on the cadavers of healthy individuals should, therefore, first be made, and later further investigations be carried out, when possible, on individuals in whom the various conditions which interfere with the typical percussion outlines of a typical case are present. It is doubtful if these latter investigations will be carried out for many years, owing to the apparently insurmountable difficulties of producing these different abnormal conditions artificially. We can, however, learn much from the uncomplicated cases. Various methods of introducing fluids into the pericardium have been tried, and have failed to give satisfactory results. Although by dividing the sternum in the median line the pericardium can be entered without perforating the pleural cavity, yet when this method is employed the results of percussion are rendered void, since under these conditions air enters not only the anterior mediastinum but also the pericardium. The method which I finally devised and found to be most satisfactory in its mechanism was as follows. The infant was placed in the position of orthopnœa; that is, the trunk was bent upon the lower limbs at an angle of about 120°. Tracheotomy was performed, and a clamped rubber tube was attached to the glass tracheal tube. The lungs were then inflated through this tube until on careful percussion the absolute area of cardiac dulness corresponded to that of a normal expiration. Under these conditions the area of absolute dulness, as you see in this diagram (Diagram 14, page 1050), begins at the junction of the upper border of the fourth left costal cartilage, and extends downward and outward to the left in a curved line, with the convexity outward and keeping 2 or 3 cm. ($\frac{3}{4}$ or $1\frac{1}{8}$ inches) within the nipple, until it joins the dulness of the left lobe of the liver. From the same starting-point at the fourth cartilage it extends downward in the left parasternal line, or rather within that line, towards the middle of the sternum, until it reaches the liver. The absolute dulness, therefore, is determined not by the shape of the heart itself, but by the marginal lines of the lungs, varying according to their expansion or retraction. This is a point which it is well to understand,—namely, that the pericardium itself, whether it is distended with fluid or not, does not by its own shape, as has been delineated so often in the plates illustrating pericardial effusions, aid us materially in determining the shape of the area of absolute dulness in a pericardial effusion. This area is marked by the retracting or rather dium it is difficult to withdraw it, the point acting like the barb of a fish-hook. A short piece of rubber tubing fitted tightly to the neck of the trocar can, as soon as the point and shoulder have entered the pericardium, be pushed up tightly against the under side of the diaphragm, thus holding the trocar in position, and the diaphragm, being firmly compressed between the shoulder and the rubber tube, prevents the entrance of air. The trocar is connected by means of a piece of rubber tubing, which is also provided with a clamp, to a simple wash-bottle graduated for cubic centimetres and containing melted cacao butter. Before introducing the trocar the cacao butter is allowed to fill the tubing and the trocar so as to displace the air. As soon as the trocar has entered the pericardium the tracheal tube is unclamped, in order that the lungs may be free to retract before the fluid. When sufficient fluid, as indicated by the graduated bottle, has entered the pericardium, the cacao-butter tube and the tracheal tube are again clamped, the thorax is carefully percussed, and the line of absolute dulness is marked in ink. After twenty-four hours the sternum is removed from above downward, remaining attached below, and we find the lungs in position surrounding the hardened fluid. I have represented in this diagram (Diagram 15) the relations of the lung and the pericardium where a small amount of fluid has been introduced.

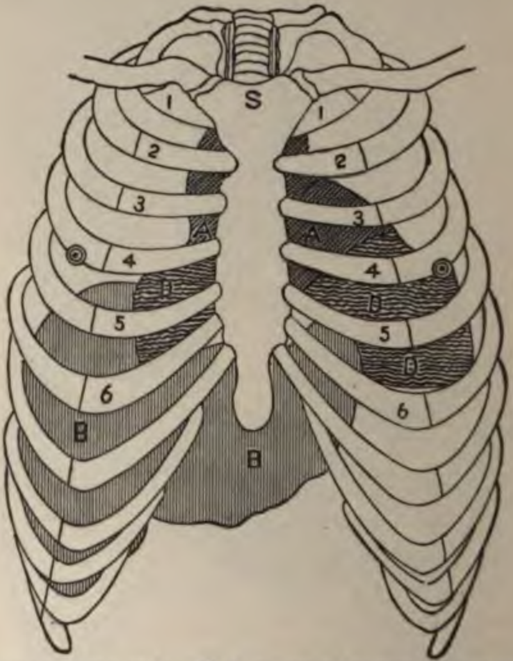
DIAGRAM 15.



Small amount of liquid introduced into sac (Rotch). ■ A, the portion of the area of absolute dulness which is still caused by the physiological dulness of the heart; ▥ B, liver; ▩ B′, that portion of the liver which is covered by the right lung; ▒ C, lung; ■ D, effusion; A + D, area of absolute dulness found when the effusion is small; S, sternum; ◎, nipple; 1, 2, 3, 4, 5, 6, ribs; --- (broken line), border of lung.

By replacing the sternum and verifying by means of needles penetrating the lines marked in ink, we can determine accurately the shape of the area of absolute dulness with this amount of effusion, which represents the results obtained when from 70 to 80 c.c. ($2\frac{1}{3}$ to $2\frac{2}{3}$ ounces) of fluid were introduced into the pericardium of an adult. There is a slight increase in the vertical as well as in the transverse area of dulness. The curved line which bounds the area of dulness starts at the sixth rib, to the right of the sternum, passes upward to the junction of the fourth cartilage with the sternum, impinges on the lower part of the third left interspace, and then descends just outside of the mammary line to the sixth rib, to pass inward until it meets the dulness of the left lobe of the liver. This line forms, as you see, an irregular semicircle, with a shorter radius to the right of the sternum and a longer one to the left. It is important to understand what causes this area of absolute dulness. This you can best do by referring to this next diagram (Diagram 16), where with the same amount of effusion the lungs have been removed, leaving the heart and the distended pericardium exposed to view.

DIAGRAM 16.



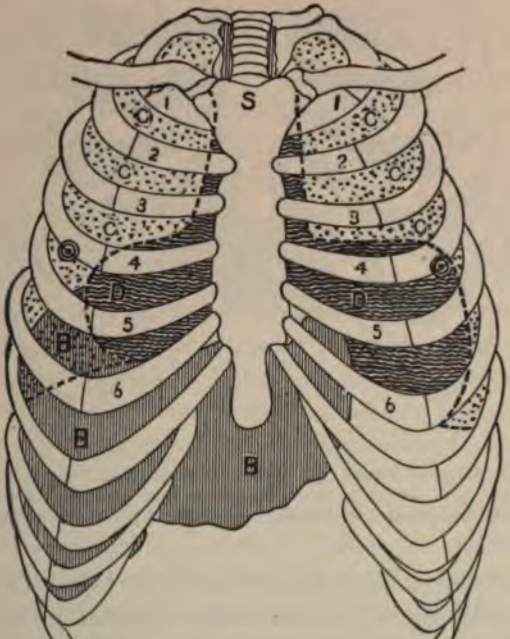
The lungs have been removed (Rotch). ■ A, portion of the normal heart enclosed in the pericardium; ▥ B, liver; ■ D, effusion as it appeared in the sac, the cacao butter being in small amount, and the lungs having been removed after the butter had hardened; S, sternum; ◉, nipple: 1, 2, 3, 4, 5, 6, ribs.

It will be seen on comparing the diagrams that a small section of the dull area, corresponding to the junction of the fourth and fifth ribs with the left side of the sternum, is formed by the heart itself, which is free from effusion at this point, while the rest of the dulness is produced by the effusion. On examining also the hardened cacao-butter cast of this effusion, it was found that the layer of fluid was very thin all over the upper portion of the effusion in the region of the fourth rib and fourth interspace, while the mass of the effusion, as shown by the greatest thickness of the cacao butter, was, as would be expected from the laws of gravity and the shape of the pericardium, in the lower part of the sac on each side of the sternum in the fifth interspaces, the cast riding the arched diaphragm like a saddle, and the larger part of the mass being on the left side. These points should be carefully noted, as they are significant for diagnosis and treatment.

The same result as to the area of dulness was obtained with a proportionately small amount of fluid in an infant about two weeks old; and of sixteen injections, of infants of various ages, the areas of dulness were identical in all, and in all the lungs were normal and there were no pulmonary or other adhesions.

This next diagram (Diagram 17) represents the position assumed by the margins of the lungs and the resulting area of absolute dulness where the pericardium was distended with a large amount of fluid so as to cover the entire heart.

DIAGRAM 17.

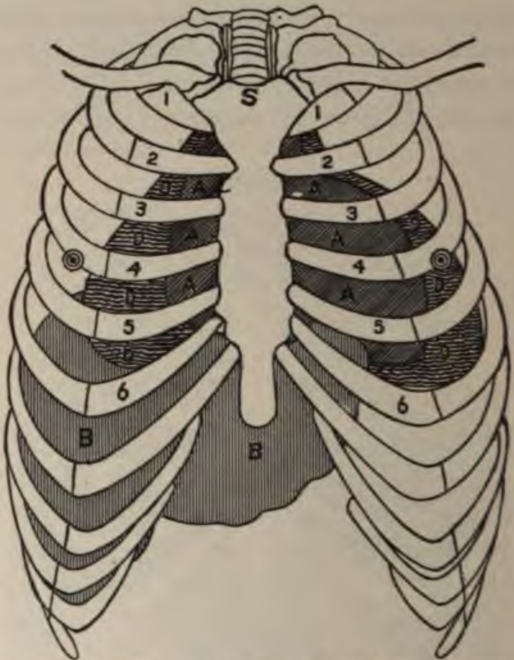


A large amount of liquid has been introduced into the sac (Rotch). ▥ B, liver; ▩ B′, that portion of the liver which is covered by the right lung; ⁙ C, lung; ■ D, the area of absolute dulness caused by a large effusion; S, sternum; ◎, nipple; 1, 2, 3, 4, 5, 6, ribs; --- (broken line), border of lung.

Here the transverse area of dulness produced by the much distended pericardium has increased so that it extends farther to the right of the sternum in the fourth and fifth interspaces, and then, rising to the third interspace, it occupies a small area on either side of the sternum under the third, second, and first ribs and the second and first interspaces, the upper lobes of the lungs having retracted from beneath the sternum. As the effusion increases the lungs retract still more, and the upper curved lines of the effusion on either side of the sternum present areas with still greater diameters.

This next diagram (Diagram 18) represents this same large effusion with the lungs removed, and also shows the relations of the heart and great blood-vessels to the ribs and sternum before the pericardium has been distended with fluid.

Diagram 18.



The lungs have been removed (Rotch). ▨ A, normal shape of the heart in its pericardium; ▨ B, liver; ▨ D, effusion; A + D, the shape which the pericardium assumed in a case where considerable fluid had been introduced into the sac; S, sternum; ◎, nipple; 1, 2, 3, 4, 5, 6, ribs.

As ordinarily seen on the injected subject, the heart would of course not appear as in the diagram, as it really was suspended in the pericardial sac with the effusion surrounding it and causing the entire area of dulness represented by A and D.

The fact that on opening the abdomen the diaphragm remains arched, and that the lung, by means of the tracheal clamp, retains its position and does not collapse, warrants us in assuming that we can fairly judge of the position of the fluid during life by this method of investigation, especially as the contractility and distensibility of the lung appeared to be perfectly retained after death, except in very cold weather, when it was found necessary to warm the cadaver. It might have been objected to these experiments that the fluid was introduced at the bottom of the pericardial sac, while during life it might originate at the base of the heart. The fluid was, therefore, in several cases introduced where the pericardium is reflected over the great vessels; but even when it was in very small amount and insufficient to cause any increase of dulness, it immediately ran down the side of the heart to the bottom of the pericardium. Even when it was mechanically retained at the base of the heart by inverting the cadaver, the resulting cast had its broadest part towards the diaphragm.

DIAGNOSIS.—From what I have told you regarding the latency of the general symptoms of pericarditis in childhood and the difficulty of interpreting the local symptoms, it will be readily understood how important it is to recognize any especial symptoms which may characterize the disease. Instances have been reported where a distended pericardium was mistaken by experienced diagnosticians for an effusion into the left pleura.

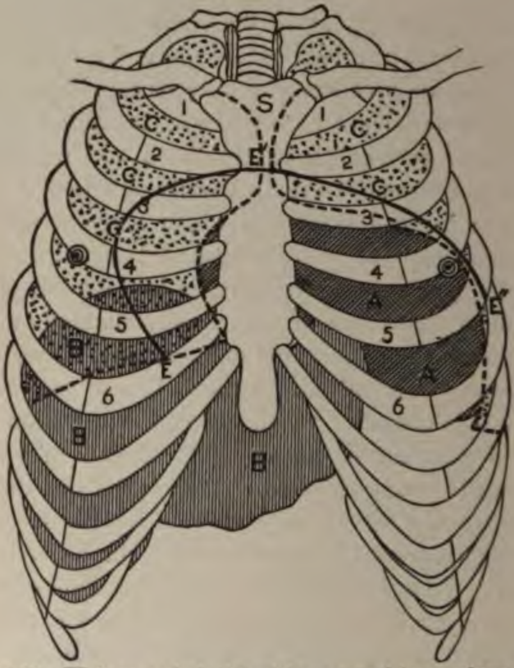
The condition, however, which most closely simulates a pericardial effusion, both in its general symptoms and in its physical signs, is a dilated heart.

The most distinctive of all the physical signs of pericarditis is the friction-sound, when it is present. When, however, an effusion has taken place, the friction-sound may not be heard. This absence of a friction-sound is especially frequent in young children. The heart's impulse may be clearly perceptible, even when a considerable effusion is present, owing to the thin layer of fluid which covers the heart in the area between the left nipple and the sternum. We are therefore forced by the similarity which at times exists between the general symptoms, in the inspection, palpation, and auscultation of a dilated heart and of a pericardial effusion, to depend upon percussion in making a differential diagnosis. In order, however, to make a differential diagnosis between this area of percussion and that produced by an enlarged heart, it will be necessary to consider the possible area of dulness which may be produced by an enlarged heart, and, by comparing this area with that which I have shown to exist in a pericardial effusion, to determine the differential diagnosis between the two diseases.

According to careful observations which have been made by competent observers on the area of dulness which can be produced by an enlarged heart, whether by hypertrophy or by dilatation, ventricular or auricular, although the relative dulness may extend to the right of the sternum from the second to the sixth rib, and in adults possibly to the distance of 3 or 4 cm. (1⅛ or 1½ inches) on a level with the fourth rib, yet it is rare to find this dulness invading the fifth right interspace more than 2 or 3 cm. (¾ or 1⅛ inches), and still more rare for the absolute dulness to be found in the fifth interspace at all, and even in the fourth interspace for more than 1.5 or 2 cm. (½ or ¾ inch).

This diagram (Diagram 19, page 1056) represents the combined views of authorities on the dulness of an enlarged heart, and will be useful to refer to when we are considering the question of paracentesis. I have myself frequently verified these percussion-outlines, and in my experience it is exceedingly rare, even in extreme cardiac enlargement, to find the relative dulness E E′ E″ of as great an extent as is represented in this diagram. The absolute dulness as represented in the diagram I have met with in most cases of enlarged heart where the enlargement is great and the sternal region is involved.

Diagram 19.



Enlarged heart (Rotch). ■ A, area of absolute dulness caused by an enlarged heart; ▥ B, liver; ▦ B′, that portion of the liver which is covered by the right lung; ⁘ C, lung; E E′ E″, the line marking the area of relative dulness of the enlarged heart; S, sternum; ◎, nipple; 1, 2, 3, 4, 5, 6, ribs; --- (broken line), border of lung.

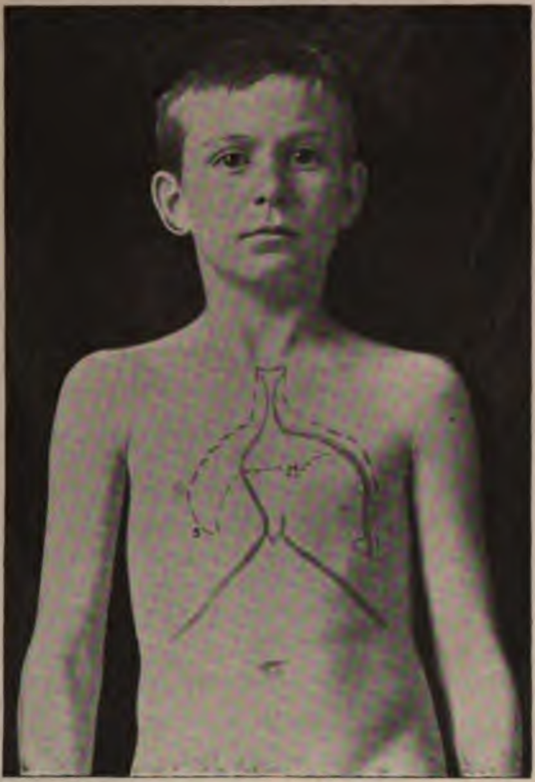
On referring to Diagram 17, we find that the dulness which occurs in a pericardial effusion may correspond to that of an enlarged heart through its whole area, but that the dulness of the effusion is also found in an additional area corresponding to a part of the fifth rib and fifth interspace. Absolute dulness, therefore, in the fifth right interspace 3 or 4 cm. ($1\frac{1}{8}$ or $1\frac{1}{2}$ inches) from the right parasternal line in cases of pericarditis uncomplicated by pleural or pericardial adhesions becomes a valuable means of differential diagnosis from an enlarged heart.

I have found in my experiments on the adult pericardium that the absolute dulness could be detected in the fifth right interspace when from 70 to 80 c.c. of fluid had entered the pericardium.

In order to illustrate to you the difference between the area of dulness produced by an enlarged heart and that produced by a pericardial effusion, I have marked on this boy (Case 508, page 1057), eleven and a half years old, the boundaries of the area of absolute dulness in an enlarged heart, in a small pericardial effusion, and in a large pericardial effusion.

I have indicated the top of the sternum, the boundaries of the enlarged heart, the ensiform cartilage, and the lower border of the ribs by plain black lines, the boundaries of the small effusion by a broken line, and the area of the large effusion by a larger broken line. The figure 5 marks the fifth right interspace; the letter H marks that portion of the heart which has been left uncovered by the small effusion. The small black circle represents the normal position of the apex of the heart, the larger circle the apex of the enlarged heart. You will also notice how the enlarged heart extends beyond the right edge of the sternum at about the fourth rib and fourth interspace, and then returns beneath the lower part of the sternum within or very little outside of the right parasternal line. The outline of the small effusion, as well as that of the large effusion, is, as you see, to the right of the sternum as low as the sixth rib.

CASE 508.



Areas of absolute dulness in enlarged heart, and in distended pericardium. 5, fifth right interspace; H, heart.

The following cases (Table 111, page 1058), taken from a number which have come under my care, illustrate the difficulty of making a differential diagnosis between cardiac and pericardial disease where, as at times happens, we fail to find a friction-sound or murmurs:

TABLE 111.

Differential Diagnosis between Dilated Heart and Pericardial Effusion.

CASE I. Endocarditis; Dilated Heart.	CASE II. Pericarditis; Effusion.	CASE III. Endocarditis; Enlarged Heart; Pericardial Effusion.
Girl, eleven years.	Boy, six years.	Girl, eight years. August 3, 1887.
Attack followed acute articular rheumatism.	Attack followed acute articular rheumatism.	Attack followed acute articular rheumatism.
Orthopnœa; præcordial pain.	Orthopnœa; præcordial pain.	Orthopnœa; præcordial pain.
Heart's impulse feeble, but perceptible a little to left and below left nipple, fifth interspace.	Heart's impulse feeble, but perceptible a little to left and below left nipple, fifth interspace.	Heart's impulse feeble, but perceptible all over cardiac area, with apex-beat a little below and to left of left nipple, fifth interspace.
Vertical absolute dulness not increased.	Vertical absolute dulness not increased.	Vertical absolute dulness not increased.
Absolute dulness under the sternum and to left of sternum; identical with Cases II. and III.	Absolute dulness under the sternum and to left of sternum; identical with Cases I. and III.	Absolute dulness under the sternum and to left of sternum; identical with Cases I. and II.
Absolute dulness did not extend to right of sternum.	Absolute dulness in fifth right interspace two or three centimetres from edge of sternum.	Absolute dulness in fifth right interspace three or four centimetres from edge of sternum.
Systolic murmur at apex.	Pericardial friction-rub at base.	Soft systolic murmur at apex, transmitted to axilla. Pericardial friction-rub at base.
Recovery.	Recovery.	August 6: Less dulness in fifth right interspace; apex murmur much louder and harsh.
		August 11: Dulness only to right edge of sternum.
		August 18: Dulness only to middle of sternum; friction-rub ceased.
		December 1, 1887: Physical examination the same as on August 18, showing enlarged heart and mitral systolic murmur.

You will observe that the symptomatology, both general and local, of these cases was, with the exception of the friction-sounds, murmurs, and percussion, identical, and that where an effusion was present dulness was found in the fifth right interspace, while where it was absent dulness was not found. These typical cases with friction-sounds and murmurs were simply chosen in order that there should be no doubt as to the disease with which I was dealing when testing the value of percussion as a means for differential diagnosis.

I have referred to pericarditis with its accompanying effusion as being likely to occur in the later stages of scarlet fever. According to Steffen, when dilatation of the heart occurs in the later stages of scarlet fever, in cases where from the age of the child, three to eight years, the physiological hypertrophy of the heart is present, the tendency to enlargement is still further promoted by the increased blood-pressure from the diseased kidney, and a differential diagnosis between a pericardial effusion and an enlarged heart thus becomes necessary.

In connection with pericarditis we should consider the possibility of both complete and partial obliteration of the pericardial cavity occurring in children. Where severe cardiac symptoms are present and no valvular murmurs are heard, we should in young children think of degeneration of the heart-muscle itself or of pericardial adhesions. When, again, the absolute area of dulness remains unchanged and there are well-marked systolic retractions, the presence of pericardial adhesions is highly probable.

While in older children and in adults pericarditis is manifested by weakness of the apex-beat, the latter sometimes being imperceptible, and by a friction-sound, in very young children these symptoms are often absent, because the exudation is moderately thick and may not be abundant enough to cause friction-sounds or to mask the apex-beat.

Chronic pericarditis may occur in infancy and in childhood as in adult life, and is the result of acute inflammatory processes which have resulted in adhesions. It is often very latent, as is shown by autopsies.

PROGNOSIS.—In early infancy diffuse pericarditis is a very dangerous disease, and usually soon ends fatally. In later childhood its course and results are much the same as in adults, and in the acute form the disease has a tendency to recovery. Among the unfavorable complications of the disease which render the diagnosis especially serious may be mentioned adhesions of the two layers of the pericardium, which may paralyze the cardiac muscles and from the resulting stasis of blood may lead to extensive dropsy. The principal symptoms of this form of cardiac paralysis are a small and frequent pulse, subnormal temperature, œdema of the cheeks, lids, and lower extremities, and the presence of a small quantity of albumin in the urine.

TREATMENT.—The treatment of pericarditis in infancy and in early childhood does not differ materially from that in later life, and depends upon the various causes which I have referred to in speaking of the etiology of the disease. The tendency to heart-failure, however, which is so pronounced in the child, should be guarded against. Early in the disease absolute physical and mental rest should be enforced. In the acute stage of the disease, before an effusion of any extent has formed, cold can be applied to the cardiac region by means of coils of tubing containing ice-water. An important part of the treatment is the judicious administration of digitalis to aid the heart in the crippled condition in which it is usually left after the early days of the disease. Stimulants should be freely used when there is any indication of heart-failure.

The most important part of the treatment when an effusion of any extent has occurred is paracentesis of the pericardium, which should unhesitatingly be performed, no matter what the cause of the disease may be, when life is in danger from undue distention of the pericardial sac. The pericardium has usually been aspirated to the left of the sternum. The possibility of wounding the heart when the aspiration is made to the left of the sternum should be considered, and, if possible, avoided. An important point both in the diagnosis and in the treatment should be here spoken of. It has been held by certain authorities that the heart's apex is found in effusions to be tilted upward and inward towards the sternal end of the fourth left interspace,—that is, floated up by the effusion. Direct proof of this is wanting, and I believe, from my investigations on this subject, that it is an erroneous view. It would seemingly be impossible for the heart not to sink rather than to be floated up, unless the specific gravity of the effusion was greater than 1050, as I have shown by experiment. It is highly improbable that the specific gravity would be greater than 1050 in an ordinary pericardial effusion, for the specific gravity of a purely purulent fluid is only about 1032. How, then, can we explain the clinical phenomenon of the heart-beat in the region of the fourth left interspace, where it is so frequently found in cases of pericardial effusion? Referring to Diagrams 15 and 16, and to Case 508 (pages 1051, 1052, 1057), it seems plausible to account for this pulsation by the tumultuous action of that portion of the right ventricle which is seen to be free from the effusion in the fourth left interspace when an effusion of any extent is present.

On examining the cacao-butter casts it is also found that this portion of the heart is in the larger effusion, as I have already described to you, covered by a very thin layer of fluid, through which the impulse of the heart can easily be felt and seen. This fact is of especial significance when we consider that both Ludwig and Bowditch have observed that the impulse of the heart as seen normally in the fifth left interspace need not be caused by the heart's apex, but may be caused by a portion of the heart above the apex striking against the thoracic wall. We should also consider that the impulse of the heart in children is often chiefly in the fourth interspace. In Case III. described in this table (Table 111, page 1058) it is recorded that the impulse was found through the whole cardiac area, but that it was still pronounced in the fifth interspace. Now, if in this case there had been a larger effusion, the apex and the lower segment of the right ventricle being surrounded by a mass of fluid, the impulse would have been lost in the fifth interspace, while in the fourth interspace, where the ventricle is covered by only a thin layer of overlying fluid, the impulse would have continued to be both seen and felt, thus simulating an apex-beat. I believe that this is the explanation of what has been called misplaced apex-beats and floating upward of the heart in pericardial effusions.

From the above facts,—namely, that the heart, when an effusion is present, remains in its usual position, and does not, even when much enlarged, impinge on the fifth right interspace, and that the effusion, even when in so small an amount as 100 c.c., is found in the fifth right interspace,—is it not more rational to choose the fifth right interspace as the point for tapping, thus avoiding all question of injuring the heart? When we tap the pleura, we avoid the heart as much as possible: why not carry out the same rule in paracentesis of the pericardium? I have tapped the pericardium in the fifth right interspace a number of times on the cadaver, and have removed the fluid as easily as in the fifth left interspace.

The pericardium has been tapped during life in the fifth right interspace by Ebstein, of Göttingen, and Wilson, of Nashville.

As an illustration of how important it is to tap the pericardium when it is much distended with fluid and when symptoms of failing heart have arisen, I shall report to you a case which occurred a few days since in the wards.

A boy (Case 509), six years old, entered the hospital with a history of having had œdema of the face, hands, feet, and ankles for four weeks. There was no history of rheumatism, and the case was apparently one of acute primary endocarditis with mitral insufficiency. The cardiac area of dulness was increased, and extended from the middle of the sternum to 1.5 cm. (½ inch) beyond the left mammary line, where the impulse of the heart could be felt. The child was kept quiet in bed, and after a few days the œdema lessened and he was very comfortable. While still under treatment, two weeks later, the temperature, which had been normal, rose to 39.1° C. (102.5° F.), the pulse was quickened and somewhat irregular, and the respirations were increased. A few days later a pericardial friction-sound was heard over the upper part of the sternum, and the temperature fell to 37.7° C. (100° F.). There was no change in the cardiac area of dulness, and no evidence of a pericardial effusion.

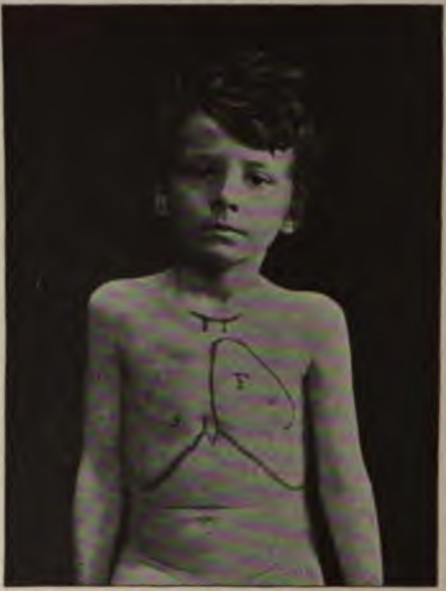
On the following day the cardiac sounds were found to be rather muffled; the child did not seem so well, and was unable to lie on his left side. Two days later the area of præcordial dulness extended farther to the right, and a little beyond the right parasternal line in the fifth right interspace. The attendants were directed to watch the child closely, and warning was given that the necessity for paracentesis of the pericardium might at any time arise. Early the following morning the child began to have marked dyspnœa and became very cyanotic. The house-officer found that the præcordial dulness had extended 2.7 cm. (1 inch) beyond the right edge of the sternum in the fifth interspace, and he therefore got the instruments ready for performing paracentesis. Suddenly the child's pulse became very weak and intermittent, the cyanosis increased very much, the dyspnœa became very marked, and, although stimulants were quickly given, the child suddenly gasped and fell back on its pillow dead. This occurred within three-quarters of an hour from the time when the first serious symptoms arose. The house-officer, Dr. Stickney, immediately introduced the aspirating needle in the fifth right interspace and withdrew some fluid from the pericardium. The child, however, did not revive.

This case of pericardial effusion, as well as the case of pleuritic effusion (Case 481, page 1011), should warn us that whenever a pleural or a pericardial effusion is present the child should be watched with the greatest care, and paracentesis should be performed as soon as any urgent symptoms arise.

Here is a little girl (Case 510), six and one-half years old. She has never had rheumatism, but she had an attack of measles when she was two years old, pertussis when she was two and a half years old, and parotitis when she was three and one-half years old. Four months ago she had an attack of chorea, of so mild a grade, however, that she has been able to go to school until entering the hospital. At that time, although she did not show any especial cardiac symptoms, an examination of the heart showed a latent and insidious endocarditis, represented by an increase of the cardiac area of dulness to the left of the mammary line, but not extending under the sternum, with a systolic murmur transmitted to the axillary line, but not heard in the back. Compensation soon became complete, and she recovered from the chorea.

Two days ago she was attacked with dyspnœa, rapid respirations, and cardiac pain.

Case 510.



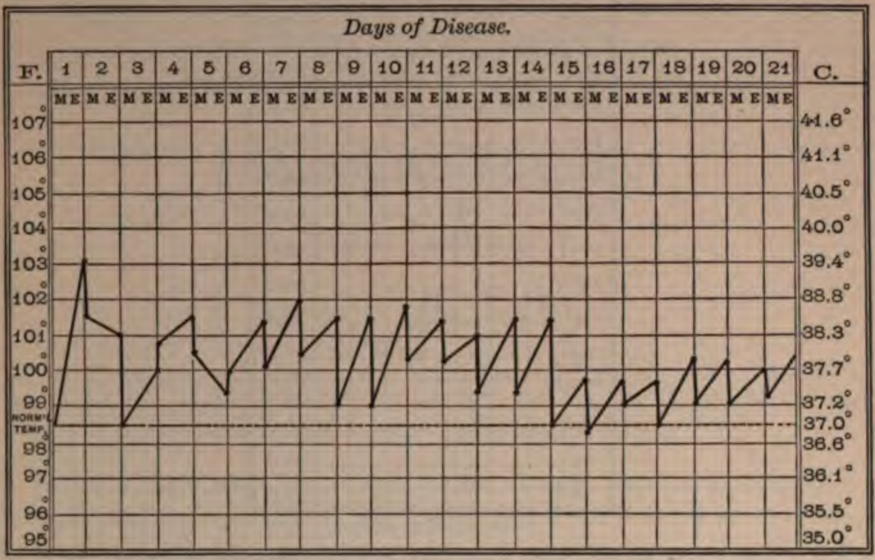
Chronic endocarditis. Mitral insufficiency. Pericarditis sicca. F, pericardial friction-sound; 5, fifth right interspace.

On examining her to-day the child seems very sick, and in addition to the area of cardiac dulness which I have marked in black, and which, as you see, shows the presence of a dilated heart, I find at the junction of the third rib with the sternum a marked præcordial friction-sound.

(Subsequent history.) The præcordial pain, discomfort, and heightened temperature lasted for a few days, and were in the beginning accompanied by orthopnœa and by the friction-sound becoming more intense. There was at no time, however, any evidence of an effusion in the pericardium, and one week afterwards the friction-sound became less distinct, disappearing three days later. The child, however, grew much weaker, and, although she was treated by complete rest in bed and with digitalis, strophanthus, and stimulants, the præcordial pain returned, and she gradually failed and died. The chart (Chart 46, page 1063) shows the temperature during the attack of pericarditis. The pulse varied from 130 to 150, and the respirations from 50 to 80.

The autopsy showed the pericardial sac to be obliterated everywhere by firm fibrinous adhesions. The heart was enlarged. Along the edge of the mitral valve were numerous small grayish-white vegetations. These were also present on the aortic valves and on the portion surrounding the tricuspid valve. The lungs were denser than normal, and were deeply injected and œdematous. The pleura on the inner surface of the right lower lobe was adherent to the pericardium by fibrinous adhesions. The surface of the liver was covered with a thin layer of fibrin. The liver and kidneys were a little denser than normal, but were not noticeably congested.

CHART 46.



Pericarditis sicca.

Anatomical Diagnosis.

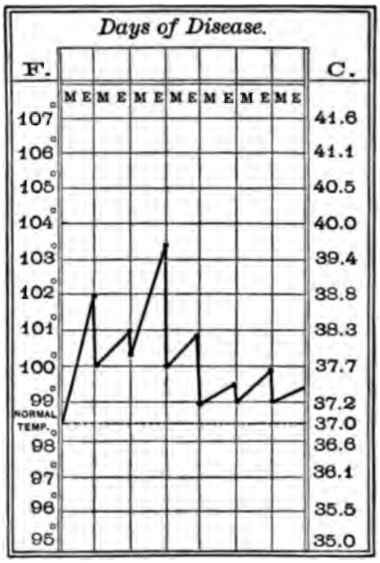
Chronic adhesive pericarditis.
Acute vegetative endocarditis.
Acute fibrinous pleurisy.
Acute fibrinous perihepatitis.
Hypertrophy and dilatation of the heart.

Here is a little girl (Case 511), eight years old, who during the first two years of her life had scarlet fever, varicella, and pertussis. When she was two years old she had an attack of measles, and when she was seven years old an attack of chorea. During the last year she has been fairly well until two weeks ago, when it was noticed that her feet began to swell, she complained of pain in her limbs, and occasionally of headache, she lost in weight, and lately has had orthopnœa with frequent paroxysms of dyspnœa. She has also at times complained of pain in her left chest. Her extremities are apt to be cold. For the past two days she has had a short, dry cough. A physical examination shows the impulse of the heart to be feeble, but it can be felt all over the cardiac area. There is an area of præcordial dulness extending to the right of the sternum almost to the right mammary line, as low as and involving the fifth interspace and as high as the third interspace and to the left a little beyond the left mammary line to the sixth rib. There is a systolic murmur at the apex, which is transmitted to the axilla. The pulmonic second sound is accentuated. There is a præcordial friction-sound heard at the upper part of the sternum. The history of the case and the area of præcordial dulness show us that it is a case of pericarditis with effusion. There may also be some endocarditis, evidence of which is given by the mitral systolic murmur. The child is being treated by rest in bed and by digitalis.

(Subsequent history.) Two weeks later the friction-sound disappeared, and the præcordial dulness grew less, so that it extended only to the middle of the sternum. In the next two weeks the dulness beneath the sternum disappeared, and the mitral murmur lessened, but could still be heard 2 cm. (¾ inch) outside of the mammary line.

This chart (Chart 47) shows the irregular temperature during seven days of the pericardial effusion.

CHART 47.



Pericarditis with effusion.

DIVISION XVIII.

UNCLASSIFIED DISEASES.

LECTURE LIII.

RHACHITIS.—SCORBUTUS.—RHEUMATISM.—PURPURA.—DIABETES.—TUBERCULOSIS.—EPIDEMIC INFLUENZA.—DISEASES OF THE THYROID GLAND.—DISEASES OF THE CERVICAL LYMPH GLANDS.—PAROTITIS.—DISEASES OF THE EAR.

I SHALL now speak of a number of diseases which are not readily classified under the divisions that I have found most useful for teaching, and which will therefore have to be spoken of separately.

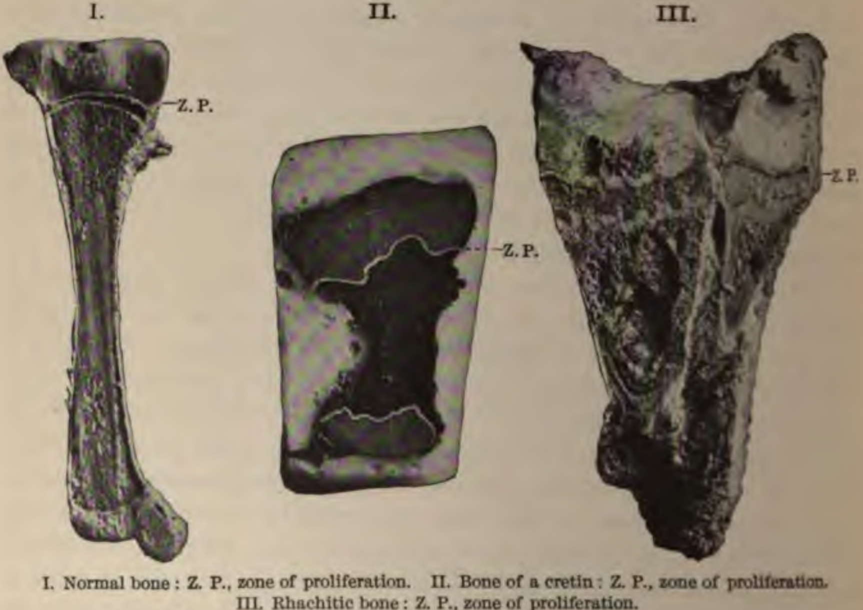
RHACHITIS.—Rhachitis is a disease of infancy, rarely of early childhood, and is closely associated with impaired nutrition. It shows itself mostly in alterations of the growing bones. Its most marked symptoms are met with between the sixth month and the second year, but it can occur at all ages, and may be congenital.

ETIOLOGY.—Although the cause of rhachitis is not yet clearly understood, it is evidently closely connected with interference with the nutrition by improper food and lack of suitable hygienic surroundings. Although it most frequently exists after the first six months of life, yet probably many cases occur earlier, but are so mild in form that the rhachitic lesions do not become sufficiently marked for recognition until the latter part of the first year. It is well known that rhachitis is much more common in its occurrence among all classes of life than was formerly supposed. As a congenital disease it is probably associated with lack of proper intra-uterine nourishment, corresponding to the rhachitis which is met with in cases of prolonged lactation. Like all diseases associated with impairment of nutrition, it is less likely to occur among breast-fed infants than among those who are deprived of their natural food. For the same reason it is more likely to develop in the latter part of the first year than in the early months, since in so many cases the breast-milk deteriorates in quality after the first six or seven months of lactation. The disease seems to occur where the food is not properly adapted to the especial age. Certain races, such as the lower classes in Italy and in England, are notably affected by rhachitis. The disease in a marked form is not common among native-born Americans.

PATHOLOGY.—Although there are lesions of the various organs which seem to be closely connected with rhachitis, such as enlargement of the spleen and of the liver, yet the bones show so markedly the most important changes that practically and clinically, in the present state of our knowledge concerning the disease, these changes in the bones constitute its pathology. We must remember, however, that the nutrition of all the tissues is profoundly affected, and that the equilibrium of the nervous system is very unstable.

According to Delafield and Prudden, the growth of the bones depends upon three conditions. They grow in length by the production of bone in the cartilage between the epiphysis and the diaphysis, and in thickness by the growth of bone from the inner layers of the periosteum. At the same time the medullary canal is enlarged in proportion to the growth of the bone by the disappearance of the inner layer of bone. In rhachitic children these three conditions are abnormally affected. The cartilaginous and subperiosteal cell growth which produces ossification goes on with increased rapidity and in an irregular manner both between the epiphysis and the diaphysis and beneath the periosteum, while the actual ossification is markedly irregular or wanting. At the same time the dilatation of the medullary cavity goes on irregularly and often to an excessive degree. If we examine microscopically the region between the epiphysis and the diaphysis usually called the zone of proliferation, we find that the cartilaginous cells are not regularly arranged in rows around a definite zone in advance of the ring of ossification, as in normal tissue, but that there is an irregular heaping up

FIG. 148.



I. Normal bone: Z. P., zone of proliferation. II. Bone of a cretin: Z. P., zone of proliferation. III. Rhachitic bone: Z. P., zone of proliferation.

of cartilaginous cells, sometimes in rows, sometimes not, covering an ill-defined irregular area. This zone of proliferation also, instead of being narrow and sharply defined, is quite lacking in uniformity. Areas of calcification may be isolated in the region of the proliferating cartilaginous cells, or calcification may be altogether absent over considerable areas.

Here is a section of a normal bone (Fig. 148, I.) taken from an infant which shows the normal zone of proliferation (Z. P.) between the epiphysis and the diaphysis.

Here also is a section of a rhachitic bone (Fig. 148, III.) which shows the broad, irregular, and abnormal zone of proliferation (Z. P.) which I have just described.

Here is a section of another rhachitic bone (Fig. 149), which shows the great enlargement of cartilage at the epiphysis, with the irregular foci of calcification. The diaphysis of the bone shows periosteal thickening to such an extent that it encroaches on the medullary cavity, which, as you see, is much diminished.

An excessive proliferation of cells in the inner layers of the periosteum, the irregular calcification which occurs about them, and the absence of uniformity in the elaboration of the structure of the bone, produce an irregular, spongy bone-tissue instead of the compact lamellated tissue which is so necessary for the uniformity of the structure. The increased cell-growth between the epiphysis and the diaphysis produces the peculiar knobby swellings which are characteristic of rhachitis. At the same time the medullary cavity increases rapidly in size, and the inner layers of the bone become spongy. The medulla may be congested, and fat, if it has formed, may be absorbed, and a species of osteitis ensue. The result of these processes is that the bones do not possess solidity and cannot resist the strain of the muscles or outside pressure. After a time the rhachitic process may stop and the bones may assume a more normal character. The porous bone-tissue becomes compact, and even unnaturally dense, so that in later childhood the rhachitic bone is unusually hard, like ivory, a condition noticed by those who have to operate on these bones.

The swelling at the epiphyses disappears as the disease passes off. Many of the deformed bones may become of a normal shape, but in severe cases the deformity may continue through life, especially if there is a cessation of the growth of the bones in their long axes, so that the children are dwarfed.

FIG. 149.

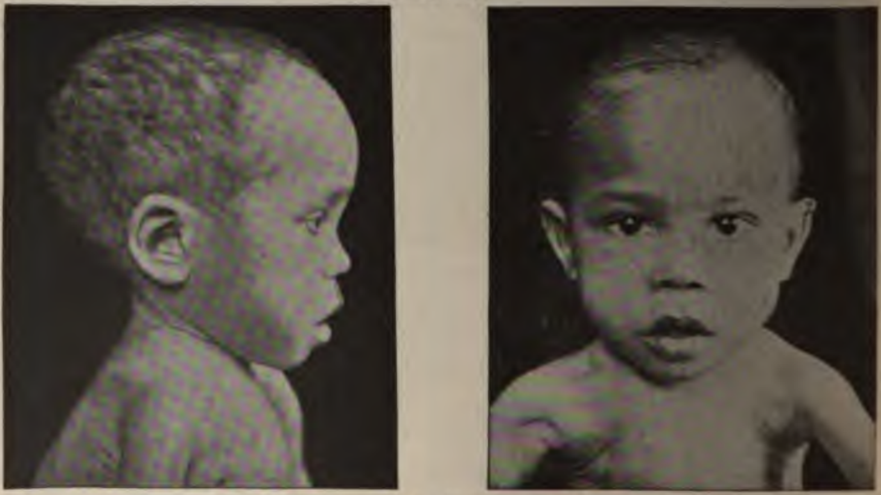
Spindle-shaped rhachitic bone.

The first signs of rhachitic disease are always found in those parts of the bones which are in the most active stage of development. In the early days of extra-uterine life the skull undergoes the most marked changes. The cranium may be unusually large for the size of the face. The fontanelles and sutures are widely open. The bones may be soft, porous, and hyperæmic, while at their edges there may be rough bony projections beneath the periosteum. Sometimes, especially in the occipital bone, there are rounded defects in the bone filled only with a fibrous membrane. This constitutes what is called *craniotabes.* The head itself is usually large and square, in contradistinction to the hydrocephalic head which I showed you at an earlier lecture (Case 286, page 638), and which in consequence of the eversion of the parietal bones has a globular shape. In the rhachitic head the parietal bones are more vertical, thus giving the square appearance.

The forehead is sometimes very prominent, and the normal thickness of the bones is increased by means of a large amount of new periosteal soft growth between the periosteum and the bones, which produces this marked deformity of the forehead.

The forehead looks high and square, and the top of the head is usually depressed. This condition of the bones may be only temporary, but if there is much deposit under the periosteum it will sometimes remain, and where calcification takes place quickly the thickened areas of the bone will remain unabsorbed throughout life. These areas of thickening, however, are often absorbed.

CASE 512.



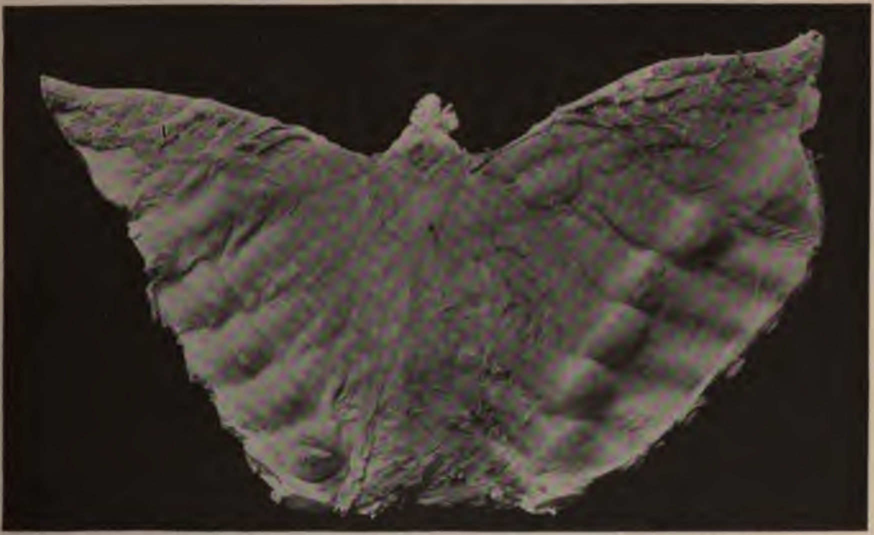
Rhachitic head. Male, 3 years old.

The teeth in rhachitic children are late in developing, and the intervals between the appearance of the different groups are longer than normal. The lower jaw is apt to be short and its angle sharp and prominent.

This infant (Case 512) shows the square rhachitic head.

In striking contrast to the large square head is the narrowed and flattened thorax. There is usually a compression of the chest laterally and a protrusion of the sternum and lower ribs, due to a constriction following the line of the diaphragm. The costal cartilages are frequently enlarged at the junction with the ribs, and can be felt and often seen as a line of rounded prominences. These prominences are called the *rhachitic rosary.* This rosary, although most commonly occurring in the latter part of the first year, has been noticed by Jacobi at the age of two months, and it has also been met with in the early weeks of life. The sternum may be depressed, or with the costal cartilages it may be pushed forward, forming what is known as *pectus carinatum* (pigeon-breast).

FIG. 150.



Inner surface of sternum, with cartilages and portions of ribs attached, showing rhachitic rosary.

Here is a specimen (Fig. 150), taken from a rhachitic child, of the sternum to which are attached the cartilages and portions of the ribs.

It shows on the inner surface a distinct rosary. During life this rosary could not be detected on the outer surface of the thorax.

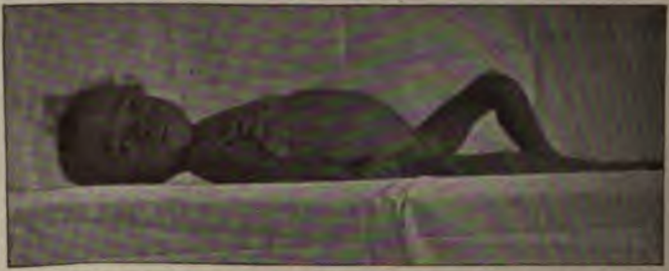
This infant (Case 513, page 1070) shows very markedly a rhachitic rosary, with depression of the lower part of the thorax, and enlarged epiphyses at the wrist.

I have described in previous lectures (pages 71, 1019, 1045) the various deformities of the sternum which arise in connection with a delay in ossification, and which may also occur in such defective ossification as takes place in rhachitis. In addition to these anterior and lateral deformities of the thorax, kyphosis is quite frequently seen in cases of rhachitis at the junction of the lumbar spines when the children begin to stand erect and to walk. Lordosis may be present. Lateral curvature may also occur.

This child (Case 514) shows rhachitic kyphosis to a marked degree.

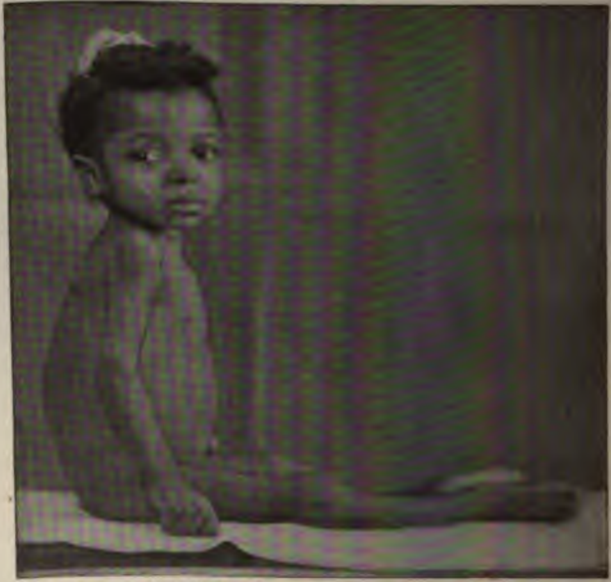
I shall not attempt to describe all the deformities which may arise in rhachitic bones. They are very numerous, and, although exceedingly interesting and important, are in the province of the orthopædic surgeon rather than in that of the physician. A well-marked deformity in connection with the limbs is the enlargement of the epiphyses, which I have just described when showing you this bone (Fig. 149, page 1067). These enlargements are especially noticeable at the wrists and ankles. The legs are apt to be bowed. Knock-knee is also often a result of rhachitis.

CASE 513.



Rhachitic rosary and enlarged epiphysis of the wrist. Female, 25 months old.

CASE 514.



Rhachitic kyphosis. Female, 3 years old.

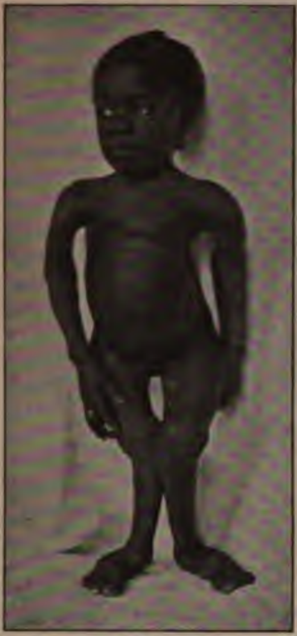
This child (Case 515, page 1071) shows a number of rhachitic deformities.

She has a square head. The thorax is narrow and contracted. The sternum is prominent. The epiphyses of the ankles and wrists are much enlarged. The arms are bowed.

Slight lateral curvature is present. The abdomen is distended. She is bow-legged and knock-kneed, and has flat-foot.

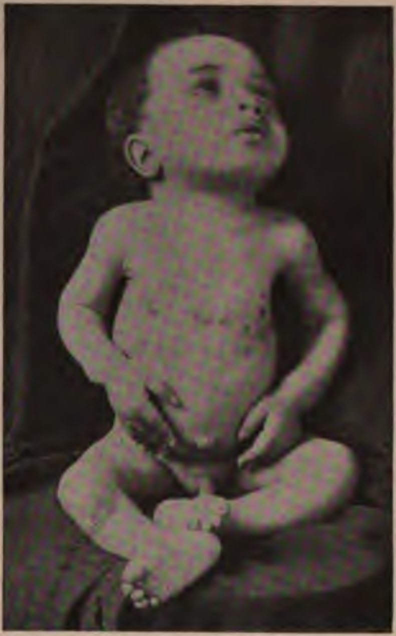
Here is another child (Case 516) who shows markedly the rhachitic deformities of the wrists, the distended abdomen, the rosary, and the rhachitic head.

CASE 515.



Rhachitic deformities.

CASE 516.



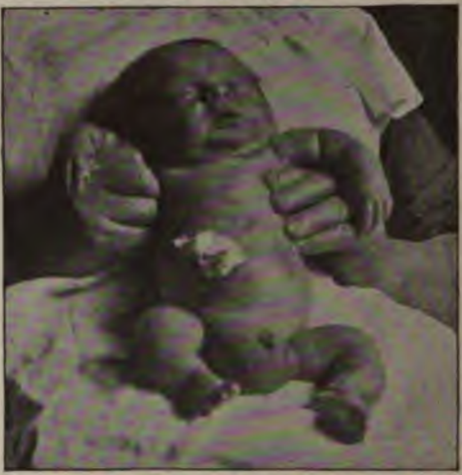
Rhachitic deformities. Male, 2 years old. The enlarged epiphyses of the ribs are marked with black spots.

CONGENITAL RHACHITIS.—Although the occurrence of intra-uterine rhachitis has been disputed, yet there seems to be sufficient evidence of such a disease in new-born infants to warrant the statement that rhachitis may be met with in this early stage of existence: it is, however, a very rare affection. I have seen a case of congenital rhachitis in which the rhachitic process had run its course and the hardening of the bones had apparently been completed before the infant was born.

Another case of congenital rhachitis which has come under my observation was seen by me in consultation with Dr. Townsend (Case 517, page 1072). The parents were young and healthy, and there was no history of syphilis or rhachitis. The father was American, the mother Scotch. There was one other child, three years old, strong and well. The mother during her pregnancy was much worried, and her nourishment was both insufficient and poor. The infant, a male, was one month premature. The labor was easy. The infant weighed seven pounds and was 43.3 cm. (17 inches) in length.

I have here a photograph which was taken on the fourth day of the infant's life. The head, as you see, was square in front, was much flattened behind, and measured 33.8 cm. (13¼ inches). The sutures were all widely open. The ossified portions of all the bones of the skull were small, particularly of the occipital bone, which presented a large area of craniotabes. In the widely-opened sagittal suture just back of the anterior fontanelle was a large Wormian bone 2.7 cm. (1 inch) long. In the squamous and coronal sutures on the right side at least eight small Wormian bones could easily be felt, and on the left side eleven were counted. The thorax was 30 cm. (11¾ inches) in circumference, and was depressed laterally, the depression increasing with each inspiration, owing to an accompanying atelectasis in the lower portions of the lungs. There was considerable cyanosis. No cardiac murmur was detected. A rhachitic rosary was present. The abdomen measured at the level of the umbilicus 28.7 cm. (11¼ inches). There was a large double inguinal hernia. The spleen could not be detected on examination. The liver could be felt below the edge of the ribs, but was apparently not enlarged. There were marked enlargement of all the epiphyses, curvature of all the long bones, and numerous fractures. The humeri showed a slight anterior curvature. The bones of each forearm were also bent anteriorly. The femora were curved outward and forward. The lower legs showed marked angular curvatures forward at the junction of the middle and lower thirds. The fractures were apparently of as recent origin as the birth, as some of them proceeded to unite very quickly. On the eighth day the fracture of the right tibia was quite firmly united; and only a slight crepitus could still be felt over the left tibia. The fracture of the left humerus was firmly united with a ring of callus. The right humerus at birth showed a callus about the middle of the shaft: this was evidently the repair of an intra-uterine fracture. The child died on the ninth day of its life.

CASE 517.



Congenital rhachitis.

SYMPTOMS.—The symptoms of rhachitis are those of a slowly developing constitutional disease. The early symptoms are those which may occur in a number of diseases, and are closely connected with disturbance of the gastro-enteric tract. The children, although they are often quite heavy, are anæmic, and their muscles are soft. The increase in weight depends more on the increase of fat, the normal relative proportion between fat and muscle being altered. Their appetite is capricious; they become fretful, and perspire at night, especially about their heads. They do not learn to walk so early as does the normally developed infant, and they soon show the later and more characteristic signs of rhachitis. I have already spoken of these signs when describing the pathology of rhachitis. As a rule, however, the picture of a rhachitic child is one with a square, prominent forehead, and with an anterior fontanelle remaining open after the age of eighteen months; dentition is delayed; the thorax is narrow and compressed laterally; the rhachitic rosary and enlargement of the epiphyses of the wrists and ankles are present, and the abdomen is distended. The bones of the extremities may be bowed, and the feet may be flat. In some cases there is considerable tenderness of the bones and muscles. The muscles are often so weakened by the depressing effects of the disease that the child has not sufficient strength to walk steadily. There are also a series of nervous phenomena connected with rhachitis which play a very prominent part in the disease. Convulsive attacks are more frequent in rhachitic children than in those whose nervous system is in equilibrium. The condition of laryngospasmus, which I have described to you in previous lectures (pages 747, 949), is at times a prominent feature in the symptoms of rhachitis. Rhachitic children are more liable to die than other children when attacked with such diseases as pneumonia or bronchitis. Attacks of the acute exanthemata are of serious import in rhachitic children, and these children are especially liable to the invasion of the bacillus tuberculosis.

DIAGNOSIS.—The diagnosis of rhachitis should be made from a number of diseases in which the general nutrition of the child is profoundly disturbed. When the disease is fully developed the diagnosis is not difficult. In its early stages, however, the manifestations of rhachitis may be so slight that the diagnosis must often be kept in abeyance. I have already spoken of the diagnosis of rhachitis from hereditary syphilis, and, as a rule, no difficulty arises. You must remember that syphilis and rhachitis have no direct connection with each other, but are both chronic constitutional diseases, and that it is possible to have both diseases occur in the same individual. I have described the syphilitic bone in a previous lecture (page 497). When there is enlargement of the long bones it is not limited to the epiphyses, as in rhachitis, but involves the ends of the diaphysis. It is often accompanied by a condition which closely simulates a callus, and there is a distinct tendency to fracture in syphilis rather than to the bending which is common in rhachitis. The notched teeth and the craniotabes may occur in both diseases, while the lesions of the mouth and lips, which I have already fully described (page 494), are distinctive of syphilis.

The diagnosis from scorbutus I shall speak of presently (page 1077).

The heightened temperature and the acute tenderness and swelling of the joints in acute articular rheumatism are easily distinguished from the subacute or chronic course and the characteristic enlargement of the epiphyses in rhachitis.

Rhachitis, where it causes kyphosis of the spine, may simulate Pott's disease very closely. It occurs at the dorso-lumbar junction, which is a frequent seat of the deformity in Pott's disease. The spine is held rigidly in severe cases, just as in Pott's disease, and the deformity may be angular rather than the usual gradual curve. The coexistence of enlarged epiphyses and other rhachitic conditions makes it very probable that the affection is rhachitic; but both diseases may coexist.

In general, the age of the child, the absence of much pain, and the existence of other signs establish the diagnosis of rhachitis. It is, moreover, in children under two, much more common than Pott's disease. In doubtful cases the diagnosis can be made only after several examinations and a period of two or three weeks of recumbency, under which conditions the rhachitic spine becomes somewhat more flexible.

Cases of rhachitis which do not walk until late, on account either of muscular weakness or of tenderness, may resemble cases of organic nervous disease with true paralysis. The diagnosis must rest on the presence of the general signs of these nervous diseases already described.

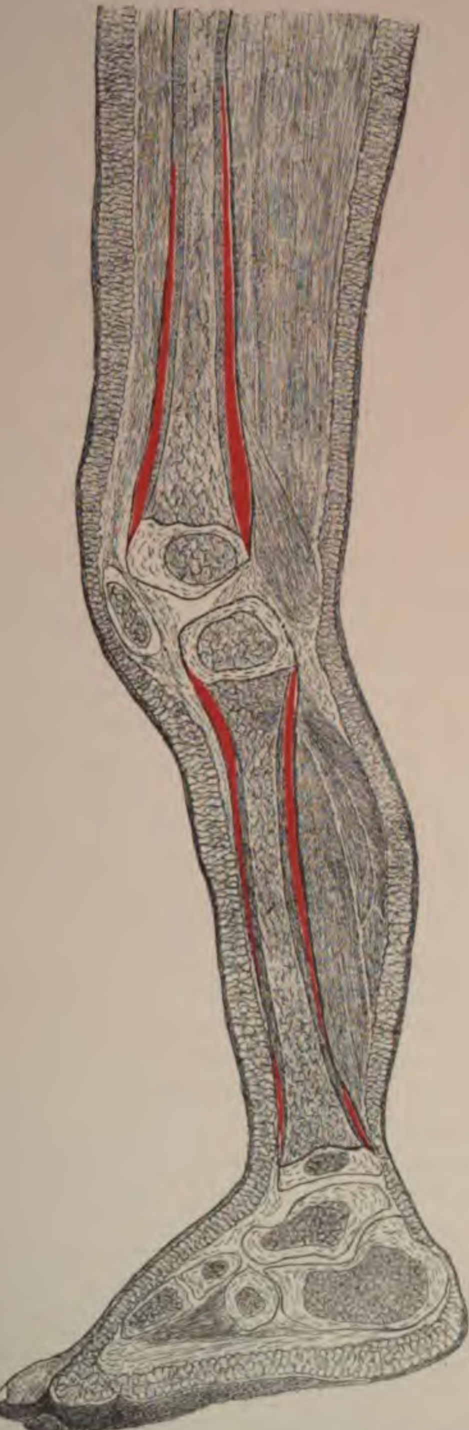
The existence of flat-foot in children over two years old should lead to an examination for knock-knee. The combination of these two conditions will in most cases be found to be dependent upon present or previously existing rhachitis.

PROGNOSIS.—The prognosis of rhachitis is favorable, provided no complications arise. A spontaneous arrest of the disease may take place in any of its stages, but, as a rule, if the affection is at all pronounced, serious deformities are usually produced. If a hydrocephalic condition, which at times appears in rhachitis, is present to any degree, if there is much diarrhœa, or if the infant is subject to frequent attacks of bronchitis, the prognosis is very unfavorable.

When properly treated, the health of these children improves slowly, and, unless the deformities which have occurred in the bones have advanced too far, more or less complete recovery usually takes place in the third or fourth year.

TREATMENT.—The treatment of rhachitis is essentially dietetic and hygienic. The infants should be kept in the open air as much as possible, and should live in rooms accessible to sunlight. The food should be adapted to the age, according to the rules which I have given for the feeding of normal infants during the first two years of life. There does not appear to be any drug which produces a specific effect upon the osseous changes which

CASE 518. FIG. 151.



Vertical section of leg in a case of infantile scorbutus. The red areas around the femur and tibia represent subperiosteal hemorrhages. (Specimen preserved in the Museum of the College of Physicians and Surgeons, New York.)

take place in rhachitis. Phosphorus is considered by some observers to be a valuable adjunct to the general dietetic and hygienic treatment, but, according to our experience at the Children's Hospital, it has not proved to be of any especial benefit.

Where the anæmia is marked, iron in some form should be given, and at times an increase in the fat in the food seems to be beneficial.

There has been much discussion as to whether a form of *acute rhachitis* exists apart from the disease scorbutus, which is now well recognized as occurring in young infants. There are certain cases of rhachitis in which the disease is in the beginning more pronounced and more acute in its development than usual. Again, in the course of an ordinary case of rhachitis acute symptoms may arise. But cases presenting the symptoms to be described under the heading of scorbutus should not be considered necessarily as acute forms of rhachitis on account of the severity of the symptoms, but for the present should be classed as scorbutus supervening on rhachitis.

SCORBUTUS (Scurvy).—Scorbutus is a constitutional disease closely associated with imperfect nutrition and having a definite relation to the deprivation of the individual from fresh food. It is characterized by anæmia and a tendency to hemorrhage, and in most cases is accompanied by the condition of the gums which is present in stomatitis ulcerosa.

ETIOLOGY.—In addition to the view that the cause of scorbutus is of chemical origin, owing to the significant relation which the disease has to a lack of fresh food, it is supposed that there may be a special micro-organism which causes the disease. This, however, has not been proved, and we have no further knowledge regarding the etiology of scorbutus.

PATHOLOGY.—So few post-mortem examinations have as yet been made on infants dying of scorbutus that the pathological lesions have not been finally established. A sufficient number of autopsies, however, has been reported by Barlow and others, notably Northrup, to settle at least the more important features in the pathology of infantile scorbutus.

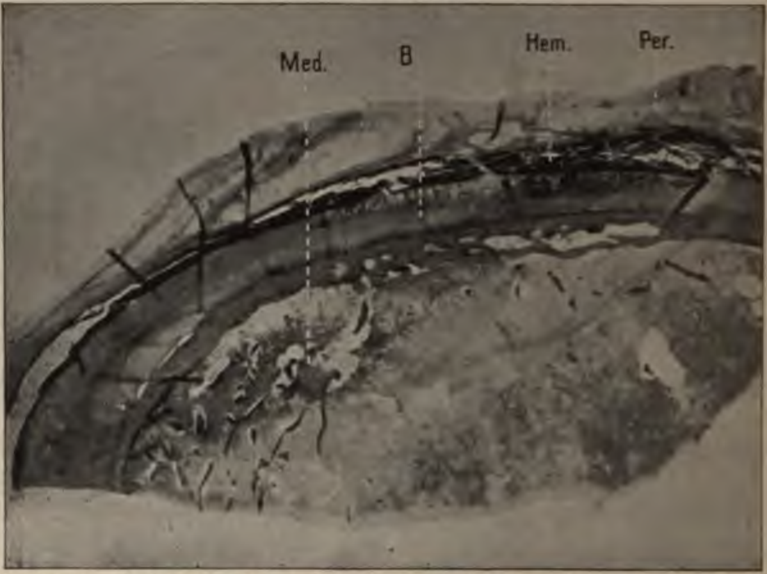
There are no alterations in the blood, either anatomical, chemical, or bacteriological, which can be considered peculiar to scorbutus. There are deep hemorrhages into the muscles and occasionally about or even into the joints, but the hemorrhage in infantile scorbutus is essentially subperiosteal and chiefly of the long bones. The femora are most commonly affected, and there is a tendency to separation of the epiphyses. There may also be a varying amount of interstitial hemorrhage in the lungs, spleen, kidney, and intestinal glands. Hemorrhages into the mucous surfaces are usually present, the gums being chiefly affected and presenting the condition of stomatitis ulcerosa, which I have described in a previous lecture (page 781).

By permission of Dr. Northrup, I have had a section made of the bones of the leg of an infant (Case 518, Fig. 151) who died of scorbutus, under his care.

On examining this specimen you will see that the femur is normal at its upper extremity. The lower half is invested with a black, grumous, subperiosteal layer of blood. The lower epiphysis is detached, and the lower end of the shaft, macerated, eroded, and soft, is lying loose in the black, disintegrating blood-clot. The tibia is surrounded by thin, dark, hemorrhagic layers beneath the periosteum, and the proximal portions are congested. The fibulæ and the bones of the upper extremities were normal.

Here is a microscopic section (Fig. 152, Case 519) of this bone, which shows no syphilitic or rhachitic changes in the bone or the periosteum.

CASE 519. FIG. 152.



Section of scorbutic bone. Med., medulla; B., bone; Hem., hemorrhage; Per., periosteum.

The soft macerated bone gave no evidence of suppuration, but there was a moderate congestion of the femur and the upper extremity of the tibia.

SYMPTOMS.—The symptoms of infantile scorbutus are those of a slow and progressive cachexia. The infants become anæmic, and show more or less gastro-enteric disturbance of a subacute functional type. Profuse sweating, especially about the head, at times slight feverish attacks, and lessened appetite, are among the early symptoms. The temperature may be from time to time slightly raised, but not significantly so. The first symptom, however, which especially attracts the attention is a sensitive condition of the bones. The infant cries when the affected parts are touched. It does not seem to suffer pain when it is allowed to remain quiet, but as the disease advances the expression of its face indicates the fear of being handled. My individual experience with infantile scorbutus has been derived from sixty or seventy cases, all of which, with few exceptions, were from eight to twelve months of age. I have met with no cases later than the first half of the second year, and with none earlier than the first half of the first year.

As the disease progresses, more marked symptoms develop. Swellings of the limbs, usually of the diaphyses just above the epiphyses, appear.

These swellings are most common and most prominent in the legs, but may also appear in the bones of the forearm. They are usually pyriform and symmetrical in shape, the skin over the swelling being more or less tense, but not fluctuating. There is commonly some tenderness on pressure, but, as a rule, no especial heat of the affected part. The pain and swelling do not seem to be in the joint, but in the diaphysis and epiphysis. Signs of hemorrhage may occur in the skin over the affected parts, appearing at first as small blue maculæ and later involving larger areas, as though a deep hemorrhage were coming to the surface. In advanced cases hemorrhage may take place to such an extent in the deeper parts around the eyes that the eyes will be pushed forward (proptosis).

Where the infant has not cut any teeth, the mucous membrane of the gums, according to my experience, has not been affected; but where a tooth is pressing on the gum and is almost through, or even where a small portion of a tooth has penetrated the gum, small areas of congested mucous membrane appear, and are of great aid in the diagnosis. In some cases a few hemorrhagic maculæ appear in other parts of the skin, as of the forehead.

In addition to the symptoms of epiphyseal pain, the infant keeps the affected limb perfectly still, so that, unless it were understood that it is pain which prevents it from moving the limb, it might be supposed that it was paralysis; in fact, this symptom in scorbutus has been termed pseudoparalysis. It has, of course, nothing to do with true paralysis, and corresponds to what is seen in rheumatic affections of the joints.

DIAGNOSIS.—The diagnosis of infantile scorbutus is to be made from rheumatism, rhachitis, purpura, syphilis, and spinal paralysis.

In the diagnosis from rheumatism the absence of heat and tenderness of the joint and of a pronounced rise of temperature is usually sufficient to distinguish the two diseases.

The diagnosis from rhachitis is to be made by the presence of hemorrhages, the intense pain in the region above the epiphyses, the absence of a rhachitic rosary, and the absence of symptoms of rhachitis when it is not coexistent. If teeth are present, the occurrence of stomatitis ulcerosa usually makes the diagnosis clear. Out of all my cases there have been only a small number, perhaps a dozen, that have shown any symptoms whatever of rhachitis. In these cases where rhachitis was present the symptoms of scorbutus appeared to complicate a primary rhachitis, and when the scorbutic symptoms passed away the rhachitic manifestations remained.

Purpura, except in the severe forms in which the joints are affected, is easily differentiated by the absence of the peculiar osseous symptoms of scorbutus.

Scorbutus is differentiated from syphilis by the extreme tenderness, the hemorrhages, and the commonly occurring stomatitis ulcerosa which occur in the former disease, while syphilis has distinctive symptoms which are not found in scorbutus, and which I have already described (page 491).

The differential diagnosis between scorbutus and spinal paralysis is made by the presence in the former of enlargement and tenderness in the neighborhood of the epiphyses. Pain is present only in the initial stage of spinal paralysis, and tenderness is absent. In spinal paralysis, also, the onset is sudden, and there are no premonitory symptoms.

PROGNOSIS.—Scorbutus is very variable in its duration. If left untreated, all the symptoms may become more pronounced and the infant finally die of exhaustion. When properly treated, and uncomplicated by any other disease, the prognosis is very favorable if treatment is begun early in the attack, before the vitality of the infant has been too much reduced.

TREATMENT.—The treatment of infantile scorbutus is essentially by changing the improper food which in most cases is being given, to fresh milk and orange juice. Under this treatment the pain and tenderness of the limbs rapidly disappear, sometimes within a few days, as does also the stomatitis ulcerosa. In the beginning the juice of one orange should be given in the twenty-four hours. If a rapid improvement does not take place, a still larger dose should be given within a few days. These scorbutic infants usually take orange juice with avidity, but they should be forced to take it if they do not like it. The nurse should be cautioned to move the affected limbs as little as possible, and the infant should be kept on a comfortable pillow on which it can be carried about.

In my earlier cases, before I recognized the scorbutic element in the disease, I treated these infants with a number of drugs, none of which appeared to have the slightest beneficial effect. In some of these cases the symptoms grew progressively worse, and the infants died. In one of them, however, where the hemorrhages in the skin were extensive and where proptosis was marked, the infant recovered entirely when a properly modified fresh milk was substituted for the artificial food which it had been taking. In some of the later cases which I have seen in consultation, where infants living in the country with good hygienic surroundings were being fed on one of the many artificial foods, the disease had progressed to such an extent that the infants were extremely anæmic, had hemorrhages in various parts of the skin, were unable to take any food, and were seemingly dying; in fact, they were as much reduced as were the cases which I have just spoken of as having terminated fatally. These infants, after taking orange juice for a few days, invariably improved rapidly, and usually recovered entirely in two or three weeks.

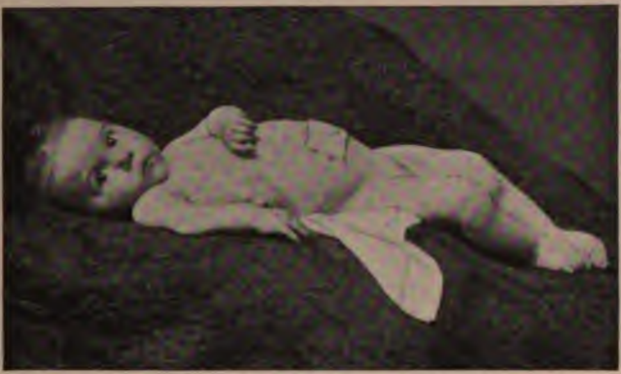
In my experience there is no evidence that sterilized milk is a cause of scorbutus. If the milk is properly modified it can be heated to 75° C. (167° F.), or even to 100° C. (212° F.), without, so far as I am aware, having a deleterious effect upon the osseous system.

All my cases have presented in different degrees the symptoms which I have just described, and which are well represented in this infant whom I have here to show you to-day.

This infant, a female (Case 520), ten months old, was healthy at birth and weighed 3636 grammes (8 pounds). It was nursed at first, but later was fed on a patent food, on which it did not gain. When it was eight months old it lost somewhat in weight, had profuse sweating, and began to have tenderness in its limbs. It has six teeth. On looking at the infant you see an expression of fear on its face, and also that it keeps its arms and legs perfectly motionless.

CASE 520.

I.

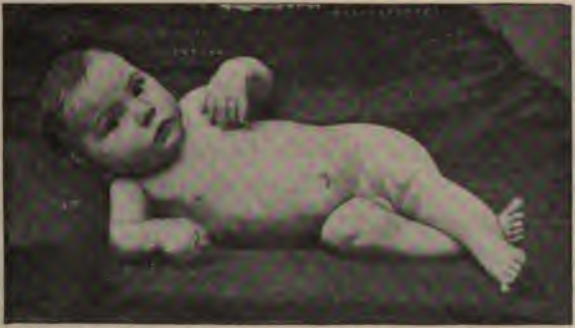


Infantile scorbutus. (Second month of disease.) Female, 10 months old.

Whenever it thinks that I am about to touch the legs or the arms it cries with fear. There is no evidence of rhachitis in this infant. You see that there is a swelling of the diaphysis just above the epiphysis of the bones of the right wrist, and also in the lower part of the femur of each leg and the lower part of the tibia. The swelling does not fluctuate, has a hard, tense feeling, and apparently is not connected with the joints. There is no increased heat of the skin, but there are certain circumscribed areas of hemorrhage in the skin over the swellings. The gums show the condition of stomatitis ulcerosa to so marked a degree that they almost cover the teeth. They are purple, bleed easily, and are very similar to those seen in the case of scorbutus which I showed you at an earlier lecture (Plate VIII., Scorbutus, facing page 781).

CASE 520.

II.



Infantile scorbutus. (One month after treatment.) Female, 10 months old.

(Subsequent history.) The infant's diet was changed to a modified milk, and it was given the juice of one orange daily. Within two days it moved its legs and arms freely, the anxious expression left its face, and in a few weeks it had gained much in weight and was perfectly well (II.).

There was no evidence of rhachitis.

An examination of the blood in this case gave the following result:

BLOOD EXAMINATION 38.

Red corpuscles	4,435,000
Hæmoglobin	35 per cent.
White corpuscles:	
Small mononuclear	8 "
Large mononuclear	44 "
Polynuclear leucocytes	57 "
Eosinophiles	1 "

The blood examinations in two other cases gave the following results:

BLOOD EXAMINATION 39.

Red corpuscles	4,660,000
Hæmoglobin	45 per cent.
White corpuscles:	
Small mononuclear	5 "
Large mononuclear	73 "
Polynuclear leucocytes	22 "

BLOOD EXAMINATION 40.

Red corpuscles	4,602,500
Hæmoglobin	(not obtained)
White corpuscles:	
Small mononuclear	10 per cent.
Large mononuclear	68 "
Polynuclear leucocytes	21 "
Eosinophiles	2 "

RHEUMATISM.—Rheumatism is a non-contagious febrile disease, when affecting children usually subacute, and characterized by pain sometimes in the joints and sometimes in the muscles.

ETIOLOGY.—The cause of rheumatism is not known, although that the disease is incited by exposure to cold and dampness is evident. It is possible that it is microbic in its origin, this view being strongly supported by the intimate relation between rheumatism and endocarditis, since the latter disease has been proved to be of bacterial origin. Acute articular rheumatism is rare in early life, though it may occur at any age. Subacute attacks of rheumatism, characterized by pains in various parts of the body and limbs and a moderate heightening of the temperature, are very common in childhood. The more severe forms of rheumatism which occur in adults, such as arthritis deformans, are very rare in children. The chronic form of rheumatism is also rare in early life. The chief characteristics of rheumatism in young children are that often it does not involve the joints, and that the milder forms of the disease are much more apt to be complicated by endocarditis than is the case in adult life.

PATHOLOGY.—There are no lesions which especially characterize the pathology of rheumatism. The lesions which occur in the course of the disease are those of other diseases, such as endocarditis or pericarditis, which so frequently complicate it. Small subcutaneous fibrous tumors at times appear during an attack of rheumatism, especially in children, and may be found in any part of the body or limbs. They seem to be closely connected with rheumatism, and the cases in which they occur are frequently associated with endocarditis.

SYMPTOMS.—The symptoms of rheumatism when uncomplicated vary according as the disease is the acute articular form or locally affects the muscles of various parts of the body, such as the neck (torticollis); sometimes the disease is simply represented by indefinite pains, which may last for a number of days and then disappear to recur at a later period. The symptoms are, as a rule, not so severe as in later life, even when the joints are affected, and in the few cases of articular rheumatism in children which have come under my care the suffering has been very slight in comparison with what is experienced in adults. In the acute articular form there are swelling, tenderness, and redness of one or more joints, accompanied by a heightened temperature and loss of appetite. A very common accompaniment of rheumatism is anæmia. The disease runs a varying course of three to six weeks, unless complicated by some other disease. The most common complications are endocarditis and pericarditis, and when these diseases appear the symptoms of these complications become prominent. In some cases the endocarditis may appear before the development of the rheumatic symptoms.

PROGNOSIS.—The prognosis of rheumatism in children is very favorable, unless complications arise, in which case it depends upon the severity of the complication.

TREATMENT.—Rheumatism in the articular form is a self-limited disease, and the treatment is purely symptomatic. The child should be kept in bed in a room of an equable temperature, 20° to 21.1° C. (68° to 70° F.). The affected joints should be wrapped in cotton wool. No applications to the joints are, as a rule, indicated. For the alleviation of the pain salicylate of sodium in moderate doses according to the age of the child is valuable; but there is no drug which is in any sense curative of rheumatism, and salicylate of sodium has not been found to lessen the frequency of cardiac complications. The oil of gaultherium can also be used, and has about the same efficacy as salicylate of sodium. Opiates are seldom needed. A careful physical examination should be made every day in these cases of rheumatism, in order to detect the cardiac complications which are so likely to arise. During the acute stage of the attack the diet should be broths and milk. A number of careful observers believe that an alkali, such as citrate of potassium, should be given in conjunction with the salicylate of sodium.

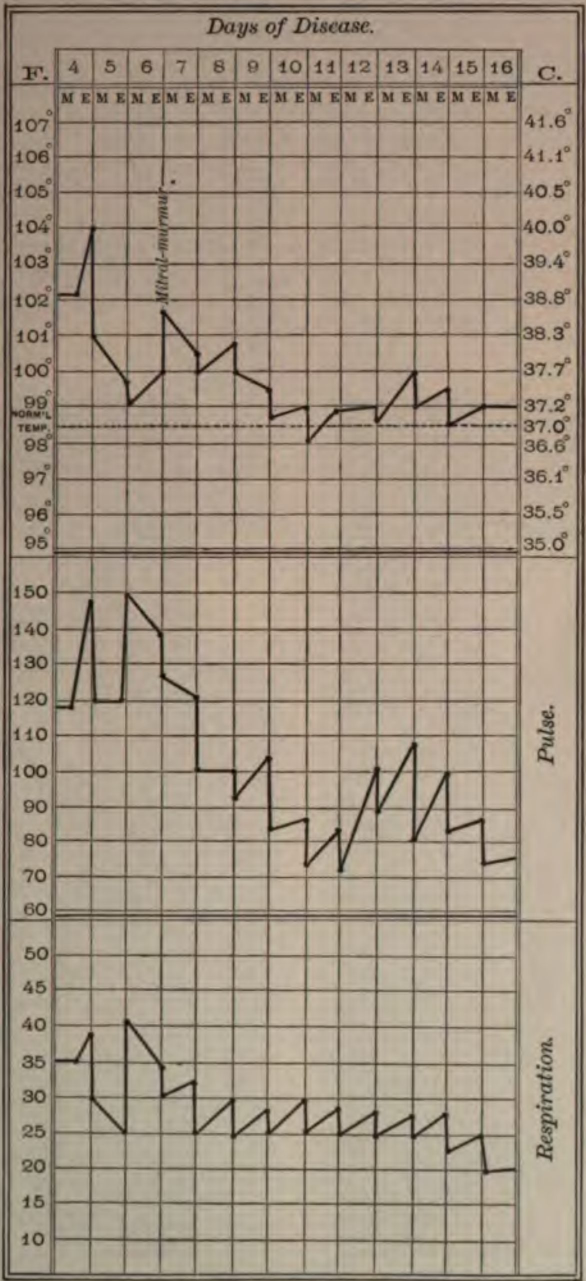
I have some cases here in the wards which illustrate the different forms of rheumatism in children.

Here is a boy (Case 521, page 1082), three years and four months old, who has been treated in the hospital for bronchitis, and when he was convalescent from that disease was attacked with acute articular rheumatism.

There is no rheumatic history in his family, and he has never had rheumatism nor any other disease except the bronchitis for which he was admitted to the hospital. After having

This next child, a girl (Case 522), five and one-half years old, is interesting as illustrating a number of characteristics in connection with the rheumatism of children.

CHART 49.



Acute articular rheumatism. Acute endocarditis on seventh day from beginning of attack.

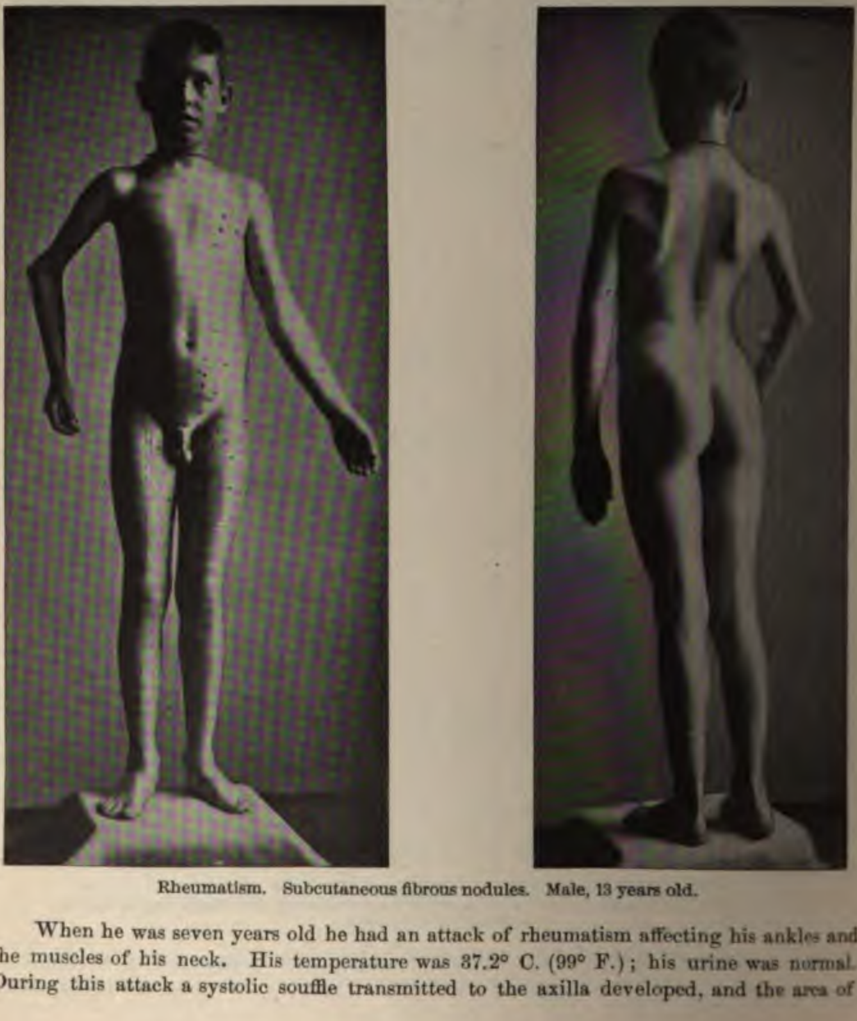
She was attacked eight days ago with pain, swelling, and tenderness in her left ankle. On entering the hospital her temperature was 40° C. (104° F.), her pulse was 145, and her respirations were 40. There was very slight pain in the joints, and, although her appetite was lessened, she otherwise seemed well, and she has not complained of any pain since the beginning of the attack. On the sixth day the temperature fell to 37.2° C. (99° F.). It has been interesting to note the extreme latency of the disease, and how the child has seemed to be perfectly comfortable from the beginning of the attack, except when the ankles, both of which are swollen and tender, were touched. Yesterday, the seventh day of the disease, the temperature rose to 38.6° C. (101.5° F.), and an examination of the chest showed a mitral systolic murmur. To-day the murmur is more marked, and is transmitted to the axilla and the back. The area of absolute cardiac dulness is slightly increased, and extends to the middle of the sternum.

(Subsequent history.) By the end of the second week of the attack the pain and tenderness had left the ankles, and the child seemed quite well. The area of absolute dulness was found to be normal, but the systolic murmur still continued.

The chart (Chart 49, page 1083) shows the rheumatism gradually subsiding up to the seventh day of the disease, when the endocarditis arose as a complication.

I have here a boy (Case 523) who during an attack of rheumatism developed the subcutaneous fibrous nodules which I have just described.

Case 523.

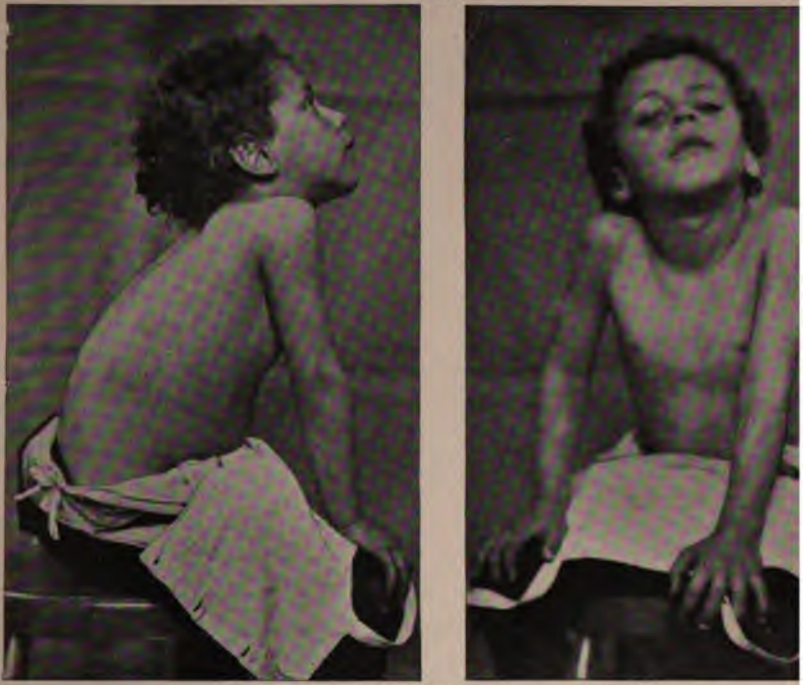


Rheumatism. Subcutaneous fibrous nodules. Male, 13 years old.

When he was seven years old he had an attack of rheumatism affecting his ankles and the muscles of his neck. His temperature was 37.2° C. (99° F.); his urine was normal. During this attack a systolic souffle transmitted to the axilla developed, and the area of

CASE 524.

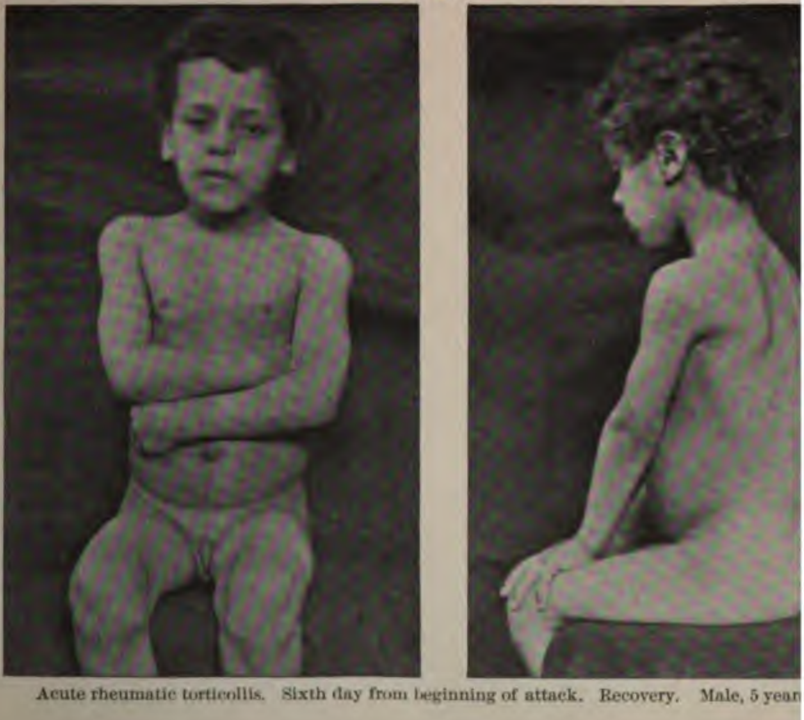
I.



Acute rheumatic torticollis. Fifth day of attack.

CASE 524.

II.



Acute rheumatic torticollis. Sixth day from beginning of attack. Recovery. Male, 5 year

absolute cardiac dulness was increased. This attack lasted eight days. He is now thirteen years old, and ever since his rheumatic attack, six years ago, he has had more or less dyspnœa on exertion, and at times cardiac pain, but he has never had any marked return of the rheumatism. He has lately noticed these small lumps appearing under his skin. When they were first noticed he had indefinite pains in his limbs, severe headache, and malaise. Some of the lumps are slightly tender. You see that they are on the chest, arms, abdomen, and legs, mostly on the anterior surface.

I have here an interesting case of the acute localized form of rheumatism which sometimes occurs in children.

This boy (Case 524) is five years old. Since he was three years old he has been subject to attacks of torticollis, apparently of rheumatic origin. Five days ago he was brought into the hospital in one of these attacks. His temperature is somewhat raised, and he has a slight loss of appetite, but otherwise he is perfectly well, and he does not suffer any pain except when his neck is touched. The head, as you see, is drawn rigidly back. These paroxysmal attacks usually last two or three days, when they pass off as suddenly as they came. The last attack which he had was one year ago.

(Subsequent history.) On the following day the stiffness and the pain in the neck passed off, and the head resumed its normal position.

Various drugs have been given in these attacks, but none with any especial benefit except salicylate of sodium, which seems to control the pain.

I have also had under my care a little boy (Case 525), about four years of age, who was attacked with fever, pain in the region of the spine, and spasm along the entire length of the spinal column. There was no pain or tenderness anywhere except over the vertebral column, and these symptoms were not so marked in the cervical region as lower down. The child had no mental disturbance, but for a number of days was in a condition of continued opisthotonos from the hips upward, and he had to be kept in a reclining chair with pillows under his arched back. The normal functions of the bladder and intestine were not interfered with. The pulse was quick, the temperature was moderately raised, and the respirations were normal. The appetite was lessened.

He remained in this condition for about a week, when the spasm of the back began to disappear. The muscles relaxed for a short time and then stiffened again. Finally complete relaxation took place, and the child recovered entirely.

The attack was acute in its onset, and did not follow any injury. The treatment was with bromide of potassium, 0.3 gramme (5 grains) three or four times in the twenty-four hours.

It seems to me that this case can be classed as one of spasmodic rheumatism.

Although acute articular rheumatism is rare in infancy, I have met with a number of cases at this early period of life.

I have already referred to the little girl (Case 41, page 127), two years old, who, after exposure, was attacked with acute rheumatism in both hip-joints.

I have also met with a case of general acute rheumatism attacking all the joints, in an infant (Case 526) two weeks old, after exposure to a cold draught while being bathed. Any movement of the joints caused the infant to scream. He lost rapidly in weight, his surface circulation was disturbed, and the attack lasted for four months; but when he was six months old he was perfectly well, and no cardiac complication developed during the attack.

Another case of this kind was an infant (Case 527) who was attacked with general acute rheumatism when she was seven months old, the attack lasting until she was fifteen months old, when she recovered without any cardiac complication, and who is a well, strong child to-day.

there were smaller ecchymoses all over the rest of the trunk. On post-mortem examination nothing abnormal was found except a slight atelectasis of the lower lobes of both lungs, with pleuritic adhesions at the base of the right lung and slight granular degeneration of the heart, liver, and kidneys, with hyperplasia of the mesenteric lymph-glands.

In the simple forms of purpura the disease in children is often mild, and is accompanied by a loss of appetite, slight anæmia, a slight degree of fever, and the appearance of petechiæ in various parts of the skin. The prognosis is good, and the duration of these attacks is usually from one to two weeks. They are at times associated with pains located in various places. It is possible for purpura to develop in the course of a rheumatic attack of great severity or where the infant's vitality is much reduced, just as it might appear in any prostrating disease.

The form which has been called *purpura rheumatica* (peliosis rheumatica; Schönlein's disease) probably has no connection with rheumatism beyond the possibility of their both being microbic, and merely simulates rheumatic arthritis from the fact that it affects the joints. The diagnosis is made by the characteristic association of multiple arthritis with purpura and urticaria. Closely simulating and probably representing purpura rheumatica, except that the gastro-enteric symptoms are more prominent, is a form which has been called *Henoch's purpura.* It occurs especially in children between the ages of three and nine years. Its direct cause is not known, although it usually occurs among children who have bad hygienic surroundings and have been ill cared for.

The symptoms are more or less malaise, and pains not especially localized, but chiefly occurring in the extremities and back, sometimes accompanied by slight œdema of the part affected. These early symptoms of pain occur in one or more joints, usually on the outer sides, and sometimes there are swelling and redness simulating articular rheumatism. In this stage there may be a sudden rise of temperature. Accompanying these symptoms there may be a few purpuric spots, but, as a rule, there is a period of several days between the appearance of the pains in the joints and the purpuric appearances on the skin. The purpuric spots may coalesce, and thus form ecchymoses of various sizes and of various colors. They are very apt to begin on the lower leg and spread up to the thighs, genitals, and body. Somewhat later intestinal symptoms develop. While the purpura is spreading there is severe colic, and the pain is very intractable to treatment. The abdomen is retracted and tender. There is obstinate vomiting. The pulse is weak, and the face has an anxious expression. There is more or less diarrhœa, which usually occurs at the end of an attack of colic. The colic and vomiting sometimes last for one or two days. There may be a little blood in the vomitus and in the movements. The vomiting then diminishes, the colic ceases, and later the diarrhœa stops, the pain in the joints passes away, the purpuric spots gradually fade and disappear, and the child, although left in an exhausted condition, is otherwise well.

There are very apt to be relapses, which may appear within a few days or not for several weeks.

These are the symptoms of a typical case; but there are many variations. As a rule, the younger the child the more typical is the case. Sometimes the purpuric spots closely simulate urticaria. They may occur, although rarely, in the mouth. They sometimes simulate the lesion of erythema nodosum. The attacks of colic have a paroxysmal character. There may be swelling of the joints.

The disease is rarely fatal unless it is complicated by some such disease as nephritis or endocarditis.

The treatment is purely symptomatic.

The most severe form of purpura which occurs is that which is called *purpura hæmorrhagica* (morbus maculosus Werlhofii). The hemorrhages in this form are from the mucous membranes as well as into the skin. The disease begins with debility. A few days later purpuric spots appear on the skin, and subsequently hæmaturia and hæmoptysis occur, from which excessive anæmia may result. There is usually slight fever. When recovery takes place it is gradual, usually occupying two or three weeks.

The prognosis is unfavorable in early life, as death may take place from the exhaustion following loss of blood or from hemorrhage into the brain. The diagnosis of purpura hæmorrhagica is to be made from scorbutus by the general history of the case, and by the absence, if teeth are present, of stomatitis ulcerosa.

Very malignant purpura hæmorrhagica may occur, sometimes proving fatal within twenty-four hours. This form of purpura is usually spoken of as *purpura fulminans*. It is most commonly met with in infants and in very young children, and is characterized by cutaneous hemorrhages which develop with great rapidity, death sometimes taking place before there has been any hemorrhage from the mucous membranes. I have met with the reports of only seven or eight cases of this malignant form of purpura.

A case of this kind was placed under my care by Dr. W. L. Richardson.

The infant (Case 529) was seven months old, had always been perfectly healthy, and was being nursed by its mother, who was a healthy, strong woman and had a number of other healthy children. The father was also a remarkably strong and healthy man. This infant, without noticeable previous symptoms, suddenly developed this severe form of purpura. Large ecchymoses appeared upon the buttocks and on the trunk, and the infant rapidly failed in strength, and died in twenty-four hours. There was no hemorrhage from the mucous membranes.

DIABETES MELLITUS.—In connection with other diseases associated with nutrition I shall mention *diabetes mellitus*, a disease in which sugar accumulates in the blood and is excreted in the urine. The origin of the disease is not known. It is a rare disease in early life.

There does not appear to be any especial difference between the symptoms and course of the disease in children and those which are met with in adults. A voracious appetite, marked thirst, progressive anæmia, and sometimes emaciation, with the passage of large quantities of urine of a high specific gravity and containing from five to ten per cent. of sugar, are commonly present. Owing to the irritation from the urine, incontinence is quite frequent.

The prognosis is unfavorable, though cases of recovery have been reported. The duration of the disease varies from a few days to a number of months and even years.

The treatment is to reduce the amount of sugar and starch in the food as much as possible. The diet which is most beneficial is milk. I have not found that there is any especial drug which is useful in the treatment of diabetes mellitus. Codeia, from 0.003 to 0.01 gramme ($\frac{1}{20}$ to $\frac{1}{6}$ grain) three times daily, has been thought to be useful in reducing the amount of sugar in the urine.

I have met with cases in which there was a transient appearance of sugar in the urine in such diseases as nephritis following scarlet fever. In these instances the sugar disappeared from the urine as the disease in which it occurred improved.

DIABETES INSIPIDUS.—Diabetes insipidus is a disease characterized by the passage of large quantities of urine having a low specific gravity and not containing sugar or other abnormal elements.

The etiology and origin of this affection are not known. It is a very rare disease, but is more common in early life than diabetes mellitus, and has been known to be congenital.

Intense thirst, a dry skin, disturbance of the surface circulation, and general nervous symptoms are common in this disease. The children are not apt to show the emaciation which occurs in diabetes mellitus.

Diabetes insipidus is essentially chronic, and so few post-mortem examinations have been made of children dying with this disease that our knowledge concerning it is very limited. There are no drugs which appear to be of benefit in its treatment. The essential part of the treatment is to protect the child from exposure and to see that it is warmly dressed, as sudden changes from heat to cold are liable to increase the general symptoms.

Death usually results from some intercurrent affection. Spontaneous cures have been known to occur.

TUBERCULOSIS.—Tuberculosis is a very prevalent affection in early life. While, according to Osler, it is very rare in the new-born, and uncommon in the first three months of life, after this age the number of cases increases very rapidly, and it is very common in the latter part of the first year and in the second year.

I have already described the cause of tuberculosis when speaking of tuberculosis of the lung (page 993), and therefore I need not again describe the bacillus tuberculosis.

It is now supposed that tuberculosis is hereditary in the sense that the infant inherits tissues which are favorable to the development of the disease, unless in the rare cases where direct intra-uterine infection has taken place.

I have spoken of the manner in which the bacillus tuberculosis gains access to the infant's tissues, either by inhalation or in the food. I have also described the manner in which the bacillus tuberculosis affects the various organs, such as the lung, pleura, pericardium, brain, liver, intestine, and lymphatic glands, especially the mesenteric glands.

I shall not attempt to describe the various lesions which may occur in tuberculosis when localized, but shall in a few words describe the general tuberculosis which occurs so frequently in infancy.

ACUTE MILIARY TUBERCULOSIS.—Acute miliary tuberculosis appears to be more common in the young than in adults. There is always some nidus from which the general infection takes place. The disease occurs as a secondary affection in children who are already tubercular.

After a variable period of loss in weight and general health, which especially occurs in cases where acute miliary tuberculosis is secondary to measles or pertussis, the infant begins to have an irregular type of fever, cough, and general symptoms, such as diarrhœa, capricious appetite, and change of temperament. In some cases the disease advances very rapidly, but often it is of a subacute type, and frequently, unless the tuberculosis markedly affects some organ, such as the lung, the symptoms are very obscure, and cannot be diagnosticated from infantile atrophy, death finally taking place from exhaustion or from the development of some localized tuberculous condition, such as a tubercular meningitis. In my experience, this form, which has been called the *typhoidal type* of the disease, and also the latent forms, are peculiarly difficult to diagnosticate.

CHRONIC DIFFUSE TUBERCULOSIS.—Where instead of the miliary lesions which characterize acute miliary tuberculosis a chronic diffuse form of tuberculosis arises, the symptoms are more marked, and usually are so closely connected with the bronchial lymphatics and the lungs that it is more easily diagnosticated. This latter is one of the more common forms of tuberculosis in children.

The prognosis of general tuberculosis in early life is very unfavorable, and there is no known treatment which is of any benefit.

In order to illustrate how extremely latent and masked may be the symptoms of miliary tuberculosis, I shall show you the results of a post-mortem examination which has just been made on an infant dying of that disease.

This infant (Case 530) was seven months old, and was in the hospital from October until December. During the time that it was in the hospital it became extremely emaciated, diarrhœa occurred from time to time, and there was an irregular and varying temperature, which was never especially high. It had a purulent discharge from the right ear and a serous discharge from the left ear about a week before its death. There were no other symptoms, but it failed rapidly, and died yesterday.

Anatomical Diagnosis.

Miliary tuberculosis of the pleura, spleen, kidney, and liver.
Chronic tuberculosis of the bronchial lymph-glands and of the lung.
Broncho-pneumonia.

Here is an infant (Case 531), one and one-half years old, who was brought to the hospital a few days ago to be treated for an attack of bronchitis. On entrance it was much emaciated, and it has since been rapidly failing. I am able to find no marked signs beyond a subacute bronchitis. There is at times a slight cough, the temperature is moderately raised and of an irregular type, and I suspect that the disease is one of the latent forms of general tuberculosis with a tubercular broncho-pneumonia. On examining the chest, back, and legs, especially the buttocks, you will notice that there are numerous subcutaneous abscesses of various sizes, and there are also a few on the head. These abscesses are probably of a tuberculous nature, and a provisional diagnosis of general tuberculosis, with involvement of the skin and the subcutaneous tissues, can be made.

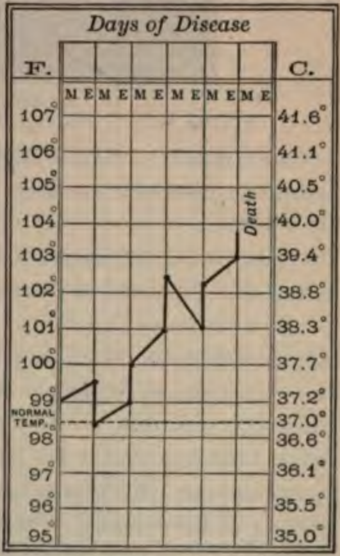
Case 531.

General tuberculosis. Multiple abscess. Infant, 1½ years old.

(Subsequent history.) The temperature in this case had been of an irregular type, and not especially raised until five days before the infant died, when it began to rise, and is as represented in this chart (Chart 50).

The post-mortem examination, made by Dr. Mallory, showed that there was chronic tuberculosis of the bronchial glands, with acute miliary tuberculosis of the pleura, lungs, spleen, kidneys, liver, and meninges of the brain.

CHART 50.



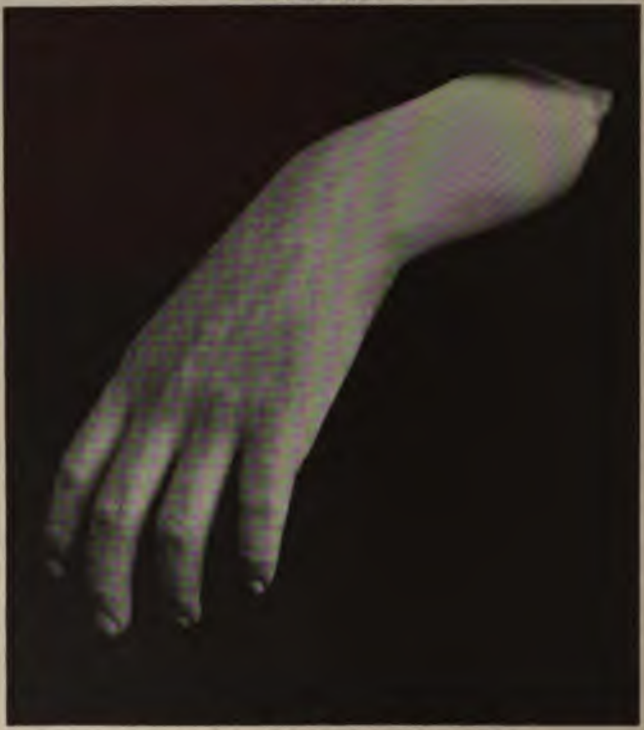
Acute miliary tuberculosis.

In addition to general tuberculosis, there are certain localized forms of the disease. The more important of these I have already spoken of, but I have here a boy (Case 532, page 1092), nine years old, who, when three years old, had a localized tuberculosis of the little finger (tubercular dactylitis) of his left hand, which has recovered entirely.

I have had him brought here to show you how completely these localized forms of tuberculosis may recover, and I shall call your attention to the cases of tubercular and syphilitic dactylitis which I showed you in a previous lecture (pages 502, 509).

The only other important form of tuberculosis which I have not yet dwelt upon is localized tuberculosis of the cervical lymph-glands. These I shall speak of in connection with non-tuberculous adenitis.

CASE 532.



Complete recovery from tubercular dactylitis.

EPIDEMIC INFLUENZA.—Epidemic influenza is an acute, highly infectious disease, caused by a specific organism which has been described by Pfeiffer.

The period of incubation is short, usually a few hours; relapses are common; one attack does not protect from another.

SYMPTOMS.—The symptoms of influenza are very variable. At times they are the same in children as in adults, but in infants and young children the symptoms are often not so severe as in the adult, although they vary in different epidemics, as do those of the adult. It is a characteristic of epidemic influenza that it has no distinct group of symptoms of its own. The symptoms are chiefly a catarrhal affection of the nose and throat, and frequently of the bronchi. These symptoms in young children are accompanied evidently by great discomfort, at times amounting to pain, in the limbs and body, although on account of the age of the patient it is impossible to determine whether much pain is present. Sometimes the only marked symptom is a heightened and irregular temperature, with marked apathy, and the disease may be so slight as to be recognizable only during an epidemic. In older children the symptoms, although, as a rule, not of so severe a type as in adults, are at times quite serious, especially if continuous vomiting occurs. Severe headache and delirium are present in some cases, and extreme emaciation, out of proportion to the fever or to the morbid conditions detectable on physical examination. Severe symptoms connected with the larynx and the lungs may also arise and rapidly disappear.

Diagnosis.—The diagnosis of epidemic influenza is often difficult, unless influenza is present in the community, and is to be made by the careful elimination of other diseases.

Prognosis.—The disease in itself is not dangerous, but complications are especially liable to arise and make the prognosis much more serious. These complications are very numerous. They may be meningitis, otitis, ileo-colitis, broncho-pneumonia, and lobar pneumonia. The most common and dangerous complication of influenza is pneumonia, which is usually a broncho-pneumonia, and is of serious import, especially if the child is debilitated at the time of the attack by some previous disease.

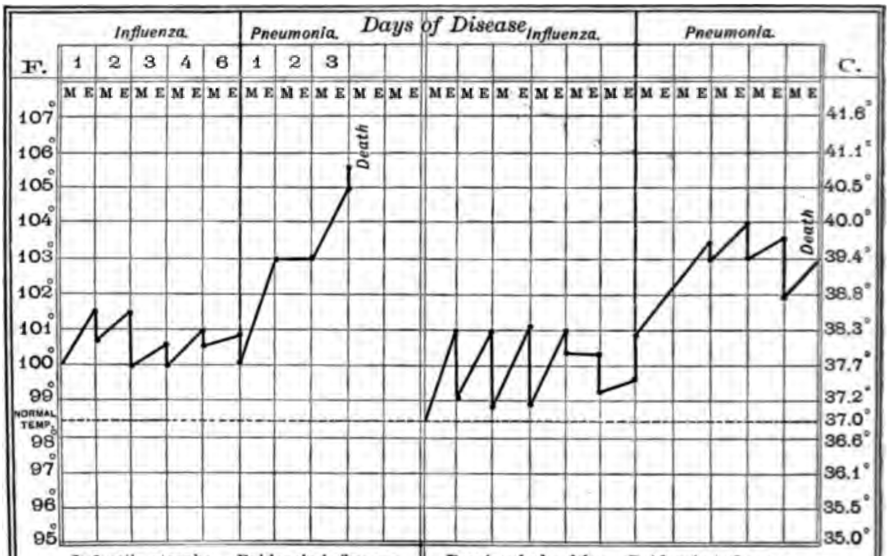
Treatment.—In the treatment of epidemic influenza in infants and children I have found that drugs have very little effect upon the general discomfort caused by the pain. Small doses of phenacetine, 0.06 gramme (1 grain) once in three or four hours, with ten or fifteen drops of brandy, seem to yield as good results as any other mode of treatment. Where there is severe and continuous vomiting, small doses of iced champagne by the mouth and enemata of bromide of potassium, and, if necessary, hydrate of chloral, are indicated. The diet should be milk and beef tea.

During the epidemic of influenza which occurred in Boston in 1891 I had under my care at the Infants' Hospital seven infants, varying in age from a few months to one and a half years, all of whom had epidemic influenza. The symptoms were such as I have described. The infants cried continuously, the temperature was slightly raised, 37.7° to 38.3° C. (100° to 101° F.), and the duration of the attack was about one week. Pneumonia occurred in two of the cases, and in both of these the infants died. Here are the charts (Charts 51 and 52, page 1094) showing the temperature in these cases during the course of the influenza, and the rise when the infants were attacked with pneumonia.

I have in my notes the report of another case, where an attack of influenza was complicated on the eleventh day of the disease by a lobar pneumonia.

The infant (Case 533), sixteen months old, was attacked with catarrhal symptoms of the nose and throat, a slight cough, and a temperature of 40.5° C. (105° F.). The respirations were only slightly increased; the pulse was rapid. Nothing abnormal was found on physical examination. The infant was very fretful, had no appetite, cried incessantly, and seemed to have considerable discomfort. On the ninth day from the onset of the attack the temperature fell to 39.1° C. (102.5° F.), and on the following day to 38.6° C. (101.5° F.). On the evening of this day the infant, who had begun to be brighter and to notice its playthings, again seemed very sick. Its respirations increased in frequency, there was motion of the alæ nasi, and the temperature rose to 40.8° C. (105.4° F.). On the following day the temperature fell in the morning, but began to rise in the evening, and by the next day had reached 41.0° C. (105.8° F.). On this day, the thirteenth from the beginning of the attack and the third from the fresh invasion, marked absolute dulness was

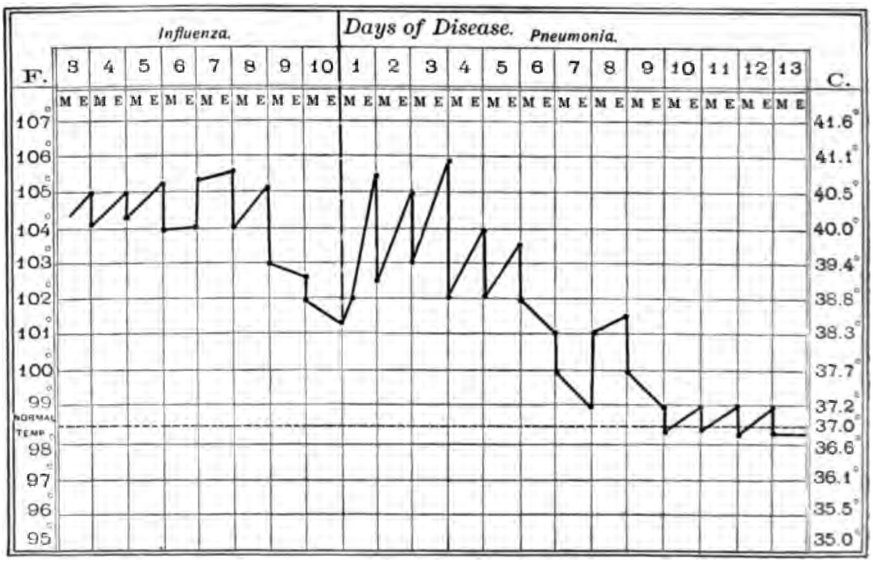
CHARTS 51 AND 52.



Infantile atrophy. Epidemic influenza. Pneumonia. Male, 4 months old.

Previously healthy. Epidemic influenza. Pneumonia. Female, 3 months old.

·CHART 53.



Epidemic influenza. Pneumonia. Recovery. Male, 16 months old.

detected in the right lower back, with bronchial respiration and increased tactile and vocal fremitus. This area of dulness increased, and finally involved the whole lower lobe of the right lung. On the fourth day of this new invasion the temperature fell to 38.8° C.

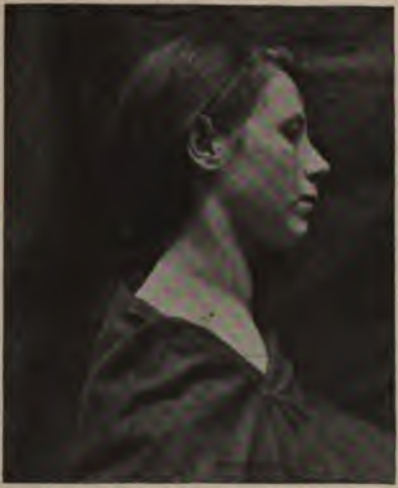
(102° F.); on the following day it rose to 39.7° C. (103.5° F.) in the evening, and in the next two days gradually fell to 37.2° C. (99° F.). On the following day it rose to 38.6° C. (101.5° F.), and in the next forty-eight hours fell gradually to 36.6° C. (98° F.). At this time the dulness began to disappear, moist râles appeared, the infant became much better, and in a few days, although very weak, seemed bright and well, and the physical signs in the lung had entirely disappeared.

Here is the chart (Chart 53), which shows the temperature during ten days of the influenza, when a lobar pneumonia appeared and ran a course of five days, after which the temperature gradually fell to the normal point. It is possible that this case was one of pneumonia from the beginning of the attack, but it showed all the characteristic symptoms of epidemic influenza, and no dulness was found in the lung until the infant had apparently recovered from its influenza.

DISEASES OF THE THYROID GLAND.—The thyroid gland is a highly vascular organ. It covers the front and sides of the upper part of the trachea, and also extends up onto the larynx. Its function is not known. I shall not describe such diseases of the thyroid gland as exophthalmic goitre, which are very rare in early life, but shall refer only to those morbid conditions which you are most likely to meet with,—namely, hyperæmia, inflammation, hypertrophy, and complete absence. Absence of the thyroid gland, disturbance of its function, or actual disease of its tissues, are usually accompanied by peculiar symptoms.

HYPERÆMIA.—A temporary congestion of the rich vascular tissue of the thyroid gland occurs under various conditions, such as the approach of puberty. This condition is usually so transient as to be scarcely noticeable so far as the symptoms are concerned, but sometimes it is sufficient to cause dyspnœa from pressure. This usually trivial condition has been thought, however, in certain cases to lead to the production of one of the forms of goitre.

CASE 534.



Hyperæmia of the thyroid gland. Female, 13 years old.

This girl (Case 534), thirteen years old, was first noticed to have a swelling of the thyroid gland two or three weeks ago. The swelling is becoming more prominent. The catamenia have not appeared. The girl is well and strong, but is somewhat more fretful and capricious than appears to be in accordance with her usual temperament. The tumor is elastic, does not fluctuate, and is not red or tender. She seems to represent one of that class of cases in which continued hyperæmia of the thyroid vessels occurring at puberty leads to enlargement of the gland. It is possible that a spontaneous lessening in the size of the tumor may take place when the catamenia have been established, but we know so little about this class of cases that the prognosis as to complete recovery must be very guarded. The treatment of this case will be the external application of iodine.

THYROIDITIS.—Acute inflammation of the thyroid gland is not very common (Delafield and Prudden), but may occur from a variety of causes. It may result in the formation of abscesses of various sizes or in the production of new connective tissue. According to Osler, acute thyroiditis is rarely primary, being commonly a metastatic affection occurring in the course of some febrile disorder. It has been noticed among children as a complication of measles, and the process in a number of these recorded cases, instead of retrograding spontaneously as it did in others, caused an inflammatory condition in which abscess-formation occurred. On opening the abscesses the pus was found to contain numerous micrococci.

The symptoms of acute thyroiditis are swelling and redness of the gland.

The treatment is essentially expectant, but some previously intractable cases seem to have been benefited by the application of iodine. The patient should be carefully watched, and, if there are indications that suppuration has taken place, an incision should be made at once, as recovery then usually occurs quite quickly.

ENLARGEMENT OF THE THYROID GLAND (Goitre; Bronchocele).—Enlargement of the thyroid gland is commonly called goitre. True goitre consists in the enlargement of the old and the formation of new alveoli in the cells of which a greater or less amount of colloid degeneration takes place. The colloid abnormalities of goitre are rarely present in children (Rex), in whom the thyroid enlargement seems to be little more than a continuation of the natural growth, and a true hypertrophy or an excessive development of normal tissue.

Infants have been born with an enlarged thyroid.

MYXŒDEMA.—Myxœdema is a constitutional affection characterized clinically by a myxœdematous condition of the subcutaneous tissues and by mental failure, caused by a disturbance of the function of the thyroid gland.

There are certain general symptoms which accompany disturbance of the thyroid function, whether from entire or partial absence of the gland, or from disease of its tissues, such as atrophy. These symptoms are hebetude, with a general thickening of the tissues, and in young individuals a great lack of development, both mental and physical. It is probable that it is a difference in degree or in kind of thyroid disturbance which produces the other symptoms so characteristic of myxœdema. These symptoms I shall presently describe to you in connection with the especial cases.

These various disturbances of the thyroid function may be endemic or sporadic. The endemic cases are represented for the most part by symptoms peculiar to disturbance of the thyroid function, and also, where goitre is present, by symptoms of mechanical pressure. Cases of goitre without cretinism may, however, occur sporadically, and the sporadic cretin, as a rule, has no goitre. Atrophy of the thyroid gland may or may not be accompanied by goitre. I shall not speak further concerning endemic cretinism, which occurs in certain localities, such as portions of Switzerland, and is dependent apparently on some unknown endemic cause which is also liable to produce goitre. As a race, cretins are distinguished by their stunted stature, large, deformed heads, sickly-looking countenances, coarse and prominent lips and eyelids, wrinkled and pendulous skin, loose and flabby muscles, and imperfect mental development, to which are often added goitres of all sizes.

In certain individuals there is a congenital absence of the thyroid gland. This is a condition found in sporadic cretinism, in which the function of the thyroid gland is lost, just as its function is disturbed in goitre and in atrophy of the gland.

Where the thyroid gland has been removed surgically there is at times a condition similar to that which is met with in myxœdema. This condition has been called by Horsley *cachexia strumipriva*.

The head in sporadic cretinism is usually brachycephalic; that is, it is contracted in its antero-posterior diameter and increased in its transverse diameter. Virchow was the first to observe that in these cases there is a premature ossification of the spheno-basilar bone. The sphenoid and the basi-occipital bones should remain separate until about the fifteenth year, and their early ossification explains, according to Virchow, the changes which take place in the form of the cretin skull and face. The characteristics of the cretin bone are an enormous overgrowth of cartilage, an arrest of growth at the distal ends of the bones, and a premature ossification of the shaft. Here is the tibia (Fig. 148, II., page 1066) of a cretin child. The section was made by Dr. Whitney, and is distinguished, as you see, anatomically by the almost entire absence of the zone of proliferation. This narrow line (Z. P.) marks the boundary between the broad area of cartilage above and the prematurely ossified bone of the shaft below.

This little girl (Case 535), whom I have had brought to the hospital to show you, and who is just able to stand, and looks as though she were about one and a half years old, seems to be a case of myxœdema.

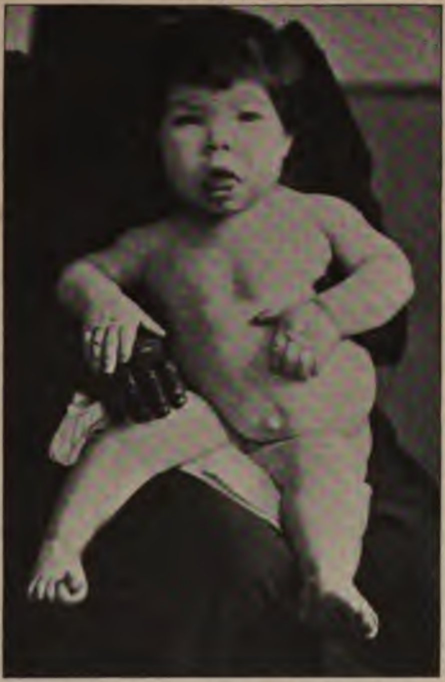
She is five and a half years old. Her parents were healthy Americans, not blood relations, and did not have goitre. She was born after a severe labor: it was a head presentation, and no instruments were used. Nothing especially abnormal was noticed about her until the twelfth month, when she did not seem so bright as is usual at that age. When four years old she was brought to the hospital. She could not speak, and her mental condition was much enfeebled. She had never had any convulsions, but had always had incontinence of urine and of fæces. When seen a year later she appeared to be in good general condition, but her muscles were large and flabby and she had not improved mentally. The circumference of her head is 46.5 cm. (18¼ inches). The measurement

I have under my care a little girl (Case 536), two years old, who appeared to be perfectly well and strong during her first year, but did not learn to sit or creep until the last few months, and who cannot stand alone or walk. There are no signs of rhachitis about the child, except that the anterior fontanelle is not closed. Nothing abnormal is found on physical examination, except that the tissues of the hands and feet are thickened and the skin is dry and cold, with at times a bluish tint. She has always held her mouth open and protruded her tongue, which seems to be thicker than normal. She is phlegmatic, and does not care to play. The bowels are constipated. There is no enlargement of the thyroid. Her case seems to be one of myxœdema.

She has been under treatment two months with the extract of the thymus gland. During this period she has grown much brighter mentally, and now creeps about more, wishes to play, and takes more interest than formerly. The tongue is not so much enlarged as before the treatment was begun, and the bowels are no longer constipated. Several times during the course of the treatment the thyroid extract has had to be omitted, as it seemed to cause digestive disturbance with a rise of temperature, 37.7° or 38.3° C. (100° or 101° F.). Omitting the thyroid extract for twenty-four hours, these symptoms would pass away, and it could then be given again.

This case (Case 537) is one of great interest, as it represents so typically the mental and physical characteristics of sporadic cretinism. I am enabled to show it to you through the courtesy of Professor Northrup, who gives the following account of it:

CASE 537.



Myxœdema. Female, 9 years old. Slight improvement after eighty days' treatment with thyroid extract.

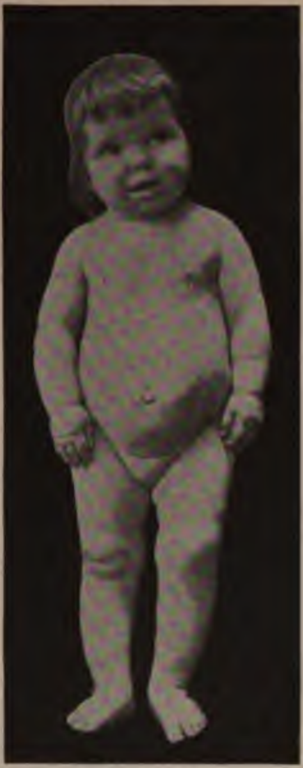
"The parents of the child were healthy Americans from Western Pennsylvania, and they were not consanguineous. The father was 45 years old; the mother was 39 years of age, had had several miscarriages and four healthy children, two of whom had died of some acute disease. This little girl, who is now nine years old, is the fifth child. The mother first noticed that the child could not sit up when it was nine months old, that it practically ceased to grow, and now at nine years it is mentally no older than it was at nine months, and physically it has merely thickened. The first impression one gets on looking at the child is that it is an idiot. Its hands are large and broad. Its color is peculiarly sallow. The hair is thin, long, dry, and without lustre. The eyebrows are present, and are not remarkable in any way. She has the characteristic flattening of the bridge of the nose, diffuse swelling of the under lid and puffiness of the upper lid, and pendulous cheeks. She has thick, pale lips, with a protruding tongue, which is swollen and pale. The lips and tongue have a tendency to dryness. There are fourteen teeth, all of them of the first set. Those in the upper row are eroded, and appear only at the bottom of a series of ulcers in the upper gums. The lower teeth are in nearly the same condition, and the gums are suppurating. An offensive odor is always present in the mouth. The arms, legs, feet, and hands are unnaturally thick. The abdomen is prominent, and there is, as you see, an umbilical hernia. The hand which is resting on its mother's black glove shows the dry, wrinkled condition so characteristic of myxœdema. Perspiration is absent. The skin is pale, and has a peculiar mottled appearance. The soles of the feet and the palms of the hands are dry. There is marked lordosis. The surface of the child does not suggest the feeling of œdema, nor does it pit. The feeling is that of puffiness and flabbiness. The child cannot sit alone. It can, however, stand when once balanced and allowed to grasp some fixed object. The supraclavicular 'pad' of tissue so commonly found in these cases is present. The thyroid gland seems to be present, and is possibly enlarged. Hebetude is shown to a marked degree, and the delayed cerebration is very evident, although the child never speaks except to say, with infinite slowness, 'da—da.'

"(Subsequent history.) The rectal temperature four days before treatment was begun was 36 4° C. (97.5° F.) in the morning, and 37.5° C. (99.5° F.) in the evening. The child was treated with the thyroid extract prepared so that each drachm represented one thyroid gland of a yearling. Of this preparation 0.06 c.c. (1 minim) was given three times a day until the fourth day, when the temperature rose above 37.7° C. (100° F.), and the treatment was stopped for a day. At this time the appetite had improved, and the breath was not so offensive. Two days later the treatment was begun again, and on the eighth day the tongue was found to be considerably smaller. During the next week the temperature remained under 37.7° C. (100° F.). It then rose above 37.7° C. (100° F.), and the treatment was suspended. The first tooth, a canine, was cut at this time. The largest dose which was given during the treatment was 0.24 c.c. (4 minims) three times a day. The child was treated eighty days in this way. The improvement was very slight, but the countenance was brighter, the tongue became much smaller, and the skin less dry. She lost somewhat in weight while under treatment. The constipation, which was marked when the treatment was begun, disappeared, and she was willing to take a much greater variety of food."

Through the kindness of Professor Osler I am enabled to show you this little girl (Case 538), who is four years old.

The parents were healthy, and there was no hereditary taint on either side of the family, none of whom have had goitre. She was the second child: the labor was easy, and she throve well. She has never had any diseases. Nothing especial was noticed about the child until its second year, when it was observed that she did not attempt to walk or talk, and that she seemed unnaturally quiet and dull. She did not cut her first teeth until she was two years old. In her third year her skin became very pale and waxy, and her face and limbs seemed puffy and swollen. She had developed very little mentally, and could say only one or two words. The other symptoms indicative of a disturbance of the function of the thyroid gland gradually appeared, such as the myxœdematous condition of the subcutaneous tissues and the development of the supraclavicular pad. The thyroid gland could not be felt. The examination of the blood showed a moderate increase of leucocytes and some irregularity in the size of the erythrocytes. When three and a half years old she was 75 cm. (29⅜ inches) tall, and her head measured 52.3 cm. (20½ inches). She had been under treatment with tonics for a year, and was reported to take more notice and to look more intelligent. She was then treated with the thyroid extract, and has improved markedly in both her mental and her physical condition. The tongue, which had been thick and protruding, is fast recovering its normal size. She can walk and talk a little, and Dr. Osler thinks that she may be considered to represent a case in which the thyroid extract has produced decided improvement.

CASE 538.



Myxœdema. Female, 4 years old. Marked improvement under thyroid treatment.

DISEASES OF THE CERVICAL LYMPH-GLANDS.—The chain of lymphatics in the neck is so closely connected with the lymphatics of the mouth and throat that infection frequently takes place. I have already spoken of the enlargement of the cervical glands secondary to absorption in cases of diphtheria. Localized enlargement of the cervical glands occurs also in tuberculosis, and sometimes is the only manifestation of that disease. The cervical glands may also be enlarged in lymphatic leucæmia and in multiple sarcoma.

Here is a little girl (Case 539) who has, in addition to marked chronic tuberculosis of the lungs, enlargement of the cervical glands, which is very probably of tubercular origin.

When the tubercular disease is advanced in other organs there is seldom much benefit to be derived from the treatment of these glands. I show you this case more for the purpose of comparison with some other cases having enlarged cervical glands than for anything of especial interest in connection with this class of advanced tubercular cases.

The cervical glands may be enlarged from a number of causes, as well as from direct infection through the throat. Any irritation of the scalp, ears, eyes, nose, throat, gums, or teeth may cause a temporary or permanent

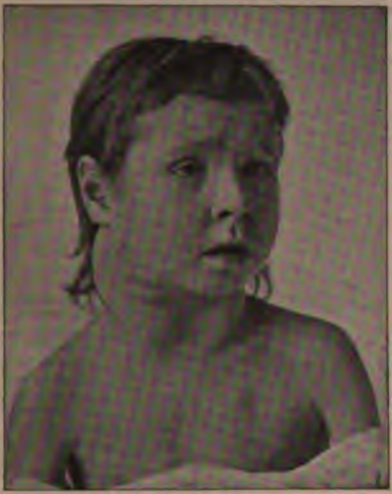
I have here a little girl (Case 540) in whom the cervical lymphatics are enlarged to such an extent that they have become a deformity.

Nothing else abnormal nor any other symptom of tuberculosis can be discovered about the child. These glands should have been removed before they reached such a size as this, as now on their removal a considerable scar will be left.

(Subsequent history.) The glands were removed, and the picture shows what good results can be obtained in these cases by surgical interference.

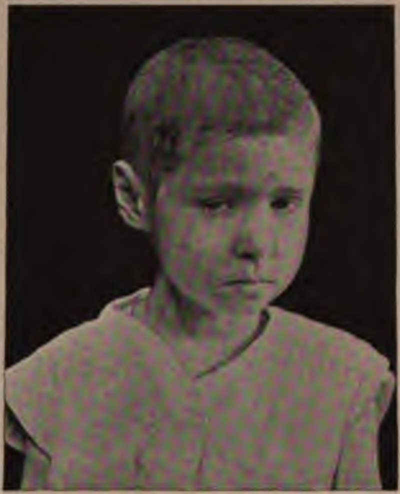
Case 540.

I.



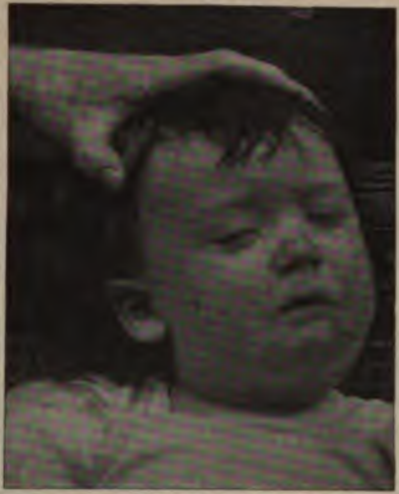
Chronic cervical adenitis.

II.



Chronic cervical adenitis (after treatment).

Case 541.



Enlargement of submaxillary glands.

The submaxillary glands are enlarged in children from various causes, but sometimes from no discoverable cause. At times the enlargement of the glands is accompanied by pain and tenderness, constituting a disease which has been called submaxillary mumps. In the beginning, however, we should not at once make this diagnosis, as the glands may become enlarged and tender from various causes which have no connection with the specific disease mumps.

Here is a little girl (Case 541, page 1103), two years and four months old, who is an illustration of this class of cases. She was suddenly attacked yesterday with a swelling of the submaxillary glands, accompanied by pain and a slight amount of tenderness over the swollen region. She has a history of exposure to parotitis. To-day the swelling has extended under the entire chin and up the left side of the neck to the face and ear.

The diagnosis in a case of this kind must be held in abeyance for a few days, and strict isolation should be enforced, as if the cause of the glandular enlargement proves to be infectious other children should be protected.

(Subsequent history.) The swelling, pain, and tenderness lasted for a number of days and then gradually subsided. Nothing more definite was discovered regarding the case.

PAROTITIS (Mumps).—Parotitis is a highly infectious disease which attacks the parotid gland. Its period of incubation is from two to three weeks. The onset of the attack is usually accompanied by a sense of chilliness, a rise of temperature, and a sensation of stiffness and tenderness about the jaws. This is succeeded by a swelling in the region of the parotid gland, which becomes enlarged and tender, rendering deglutition difficult and often very painful. The disease begins on one side, but the other gland is usually involved in a day or two. As I have just stated, the infection is sometimes confined to the submaxillary glands on one or both sides.

The duration of an attack of parotitis is from a few days to a week, but the infection may last for two or three weeks, and it has been stated in some cases to antedate the appearance of the glandular enlargement. In boys at the age of puberty the complication of orchitis at times arises.

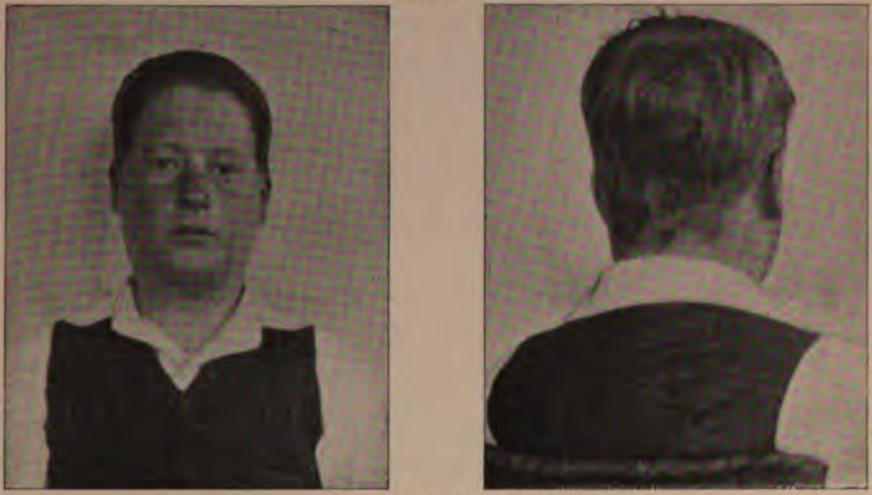
Although the symptoms of parotitis are commonly very mild, unusual cases sometimes occur in which the children are quite sick, and there have been cases in which the orchitis was of so high a grade that acute delirium supervened, and in one case reported by Dukes the boy fainted when the orchitis began.

It is sometimes difficult to differentiate parotitis from a simple non-infectious enlargement of the parotid gland or of the glands in its neighborhood. When the parotid gland is enlarged it usually shows a characteristic swelling under and behind the lobe of the ear, so that the lobe is pushed somewhat upward and forward. This swelling increases rapidly, is very tender, is not especially reddened, does not fluctuate, and is accompanied by constitutional symptoms. The diagnosis is readily made if after a few days the unilateral swelling is followed by corresponding symptoms on the opposite side.

Here is a boy (Case 542, page 1105), twelve years old, who was attacked five days ago with swelling and tenderness in the region of the parotid gland, followed in a short time by stiffness and pain at the angle of the jaw, and accompanied by symptoms of loss of appetite and a heightened temperature.

Two days later the parotid of the opposite side was involved. You will notice how the neck in the part which corresponds to the position of the parotid is swollen on both sides, and how the characteristic swelling which pushes the ear upward and forward is seen on looking at the child from behind.

CASE 542.



Parotitis. Male, 12 years old.

There is no especial treatment for the disease, as it is self-limited and runs a definite course. The children should be carefully isolated, in order that there may be no further spread of the infection. As deglutition is painful, their diet is usually milk and soups. They should be carefully protected from exposure, and should be confined to their rooms. Older children should be confined to bed, as orchitis in boys and trouble with the mammæ in adolescent girls are less likely to arise under these conditions. It is usually better to apply some soft cotton wool to the painful swelling, and to protect it from any irritation.

DISEASES OF THE EAR.—We have, gentlemen, studied the normal infant at birth, and have followed it through its various stages of development into childhood, up to the age of puberty. I have also endeavored to make you familiar with the various morbid conditions which are most likely to arise during these early periods of life.

Before closing this course of lectures, however, I wish again to call your attention to the great importance of bearing in mind, in examining infants and children, the common occurrence of some morbid process in the ear. In many cases where the more pronounced aural symptoms are not evident, symptoms which appear obscure, but really are due to some latent disturbance in the neighborhood of the ear, reflex or otherwise, are readily explained when in addition to the presence of some other disease the unusual symptoms are found to arise from the aural complication. The question of diseases of the ear in infancy and childhood has not received from the general practitioner, nor indeed from those who devote themselves especially to children, the attention that it deserves. Even leaving out of

INDEX.

A.

Abdomen in the new-born infant, 44.
  of the premature infant, 291.
Abdominal band, 183.
  organs, normal development of, 77.
Abnormal effects of vaccination, 152.
Abscess, cerebral, 648.
    prognosis of, 648.
    treatment of, 648.
  peritonsillar, 815.
  retro-pharyngeal, 817.
Acquired disease, definition of, 19.
Acute catarrhal gastritis, 854.
  corrosive gastritis, 856.
  coryza, 801.
  cystitis, 942.
  fatty degeneration of the new-born, 440.
  follicular pharyngitis, 816.
  gastric indigestion, 844.
  gastritis, 854.
  laryngitis, 951.
  miliary tuberculosis, 1090.
  nephritis, 932.
  peritonitis, 920.
  rhinitis, 801.
  simple pharyngitis, 815.
  tonsillitis, 809.
  tubercular bronchò-pneumonia, 994.
  tuberculosis of the lung, 993.
  yellow atrophy of the liver, 915.
Adenoid growths, 806.
Adolescence, urine of, 114.
Air-passages, upper, syphilis of the, 490.
Albuminuria, physiological, 927.
Alopecia areata, 461.
  treatment of, 462.
Amnesia, temporary, 739.
Amyloid infiltration of the kidney, 938.
  liver, 916.
Anæmia infantum pseudo-leukæmica, 359.
  progressiva perniciosa, 356.
  rhachitic, 368.
Anæmias, primary, 355.
  secondary, 365.
Anatomy, topographical, of the early periods of life, 120.
Anencephalia, 409.
Anencephalocele, 409.
Animal heat of premature infants, 295.
  parasites, 908.
Anterior poliomyelitis, 609.
Antitoxin, 829.
Anuria, 946.
Anus, fistula of the, 880.
  imperforate, 433.
  syphilitic condylomata of the, 494.
Apparatus for feeding, 232.
  for feeding at home, 262.
Appendicitis, 888.
  diagnosis of, 889.
  etiology of, 888.
  prognosis of, 889.
  symptoms of, 888.
  treatment of, 890.
Appendix vermiformis, anatomy and development of, 92.
Arrested physical development, 740.
Arteries, umbilical, 20.
Artery, common carotid, 43.
  pulmonary, 43, 75.
Arthritis, acute, of infants, 1086.
Artificial foods, 154.
Ascaris lumbricoides, 910.
  symptoms of, 911.
  treatment of, 911.
Asiatic cholera, 887.
Asphyxia, 440.
Asthma, etiology of, 1004.
  pathology of, 1005.
  prognosis of, 1005.
  symptoms of, 1005.
  treatment of, 1005.
Ataxia, hereditary, 689.
    pathology of, 689.
    prognosis of, 689.
    symptoms of, 689.

- Ataxia, hereditary, treatment of, 689.
  - locomotor, 689.
    - diagnosis of, 689.
    - pathology of, 689.
- Atelectasis, 979.
- Athetosis, 661.
  - diagnosis of, 661.
  - pathology of, 661.
  - prognosis of, 661.
  - treatment of, 661.
- Atrophic rhinitis, 804.
- Atrophy, acute yellow, of the liver, 915.
  - infantile, 869.
    - diagnosis of, 869.
    - pathology of, 869.
    - prognosis of, 870.
    - symptoms of, 869.
    - the blood in, 377.
    - treatment of, 870.
  - myopathic progressive muscular, 763.
    - diagnosis of, 765.
    - pathology of, 764.
    - prognosis of, 765.
    - symptoms of, 764.
    - treatment of, 765.
  - neuropathic progressive muscular, 768.
  - of the intestine, 869.
  - progressive muscular, 763.
- Auscultation of the sick child, 822.
- Autumnal catarrh, 1005.

B.

- Babcock milk-tester, 249.
- Back, care of the, 143.
- Bacteriological examination of human milk, 180.
- Barley jelly, 282.
  - water, 281.
- Basin as used in bathing the infant, 129.
- Bath, temperature of the, 129.
- Bathing, 128.
- Bed for a child, characters of, 126.
- Bile in the new-born infant, 52.
  - normal development of the, 111.
- Bile-ducts, congenital obstruction of the, 438.
  - pathology of, 438.
  - symptoms of, 438.
  - treatment of, 440.
- Bilious attack, 862.
- Birth-paralysis, 438.
- Bladder at end of term, 24.
  - in the new-born infant, 48.
  - normal development of the, 78.
  - reflex of the, 752.
- Blood, bibliography of the, 398.
  - chemistry of the, 334.
  - fœtal, 337.
  - in individual diseases, 370.
    - broncho-pneumonia, 373.
    - chorea, 376.
    - diphtheria, 372.
    - empyema, 373.
    - hydrocephalus, 375.
    - icterus neonatorum, 379.
    - infantile atrophy, 377.
    - malaria, 380.
    - measles, 371.
    - miliary tuberculosis, 374.
    - nephritis, 376.
    - parasites of the, 380.
    - periostitis, 378.
    - peritonitis, 377.
    - pneumonia, 372.
      - and empyema, 373.
    - scarlet fever, 371.
    - sclerema neonatorum, 379.
    - scorbutus, 379.
    - tubercular meningitis, 374.
    - typhoid fever, 370.
    - variola, 371.
  - in infancy and childhood, 330.
  - in premature infants, 348.
  - in the fœtal circulation, 19.
  - in the new-born infant, 52, 349.
  - key, 331.
  - literature of the, 329.
  - nomenclature of the, 330.
  - normal, at birth, 339.
    - color of, 339.
    - development of, 111.
    - hæmoglobin of, 340.
    - reaction of, 339.
    - specific gravity of, 339.
  - origin of the, 336.
- Blood-vessels, malformations of the, 440.
- Bone, normal development of, 107.
- Bone marrow in the new-born infant, 51.
- Brain, diseases of the, 594.
  - dura mater of the, 65.
  - growth of the, 65.
  - in the new-born infant, 37.
  - normal development of the, 64.
  - subarachnoid space of the, 65.
- Branchial fistula, 417.
- Breast-pump, 162.
- Broken cells, 345.
- Bronchitis, 954.
  - acute, 955.
    - diagnosis of, 957.
    - pathology of, 955.

Bronchitis, acute, prognosis of, 957.
        symptoms of, 955.
        treatment of, 957.
    chronic, 961.
    fibrous, 962.
Broncho-pneumonia, 962.
        complications of, 972.
        diagnosis of, 972.
        etiology of, 962.
        pathology of, 963.
        symptoms of, 969.
        the blood in, 373.
        treatment of, 973.
    chronic, 979.
Brooder, 309.
    apparatus connected with the, 313.
Broth, chicken, 287.
    mutton, 287.
Buhl's disease, 440.

C.

Cæcum in the new-born infant, 47.
    normal development of, 92.
Cancrum oris, 788
Capacity of the stomach, 79.
Caput succedaneum, 404.
Caries of the spine, paralysis caused by, 688.
Carpet, the, in the nursery, 126.
Catalepsy, 735.
Catarrh, acute gastric, 854.
    autumnal, 1005.
    chronic gastric, 856.
    periodic, 1005.
Catarrhal ophthalmia, 415.
    stomatitis, 776.
Ceiling, the, in the nursery, 126.
Cephalhæmatoma, 404.
Cerebral abscess, 648.
    paralysis, 648.
    sinuses, thrombosis of, 626.
Cerebro-spinal meningitis, 692.
        diagnosis of, 694.
        etiology of, 693.
        pathology of, 693.
        prognosis of, 695.
        symptoms of, 694.
        treatment of, 696.
    sclerosis, multiple, 691.
Cervical glands, effect of scarlet fever upon, 537, 555.
Chicken broth, 287.
Chicken-pox, 524.
Child, distinction of, from infant, 18.
Chlorosis, 355.
Cholera Asiatica, 887.
Cholera infantum, diagnosis of, 886.
    etiology of, 885.
    pathology of, 885.
    prognosis of, 886.
    symptoms of, 885.
    treatment of, 886.
Chorea, 711.
    etiology of, 712.
    pathology of, 712.
    prognosis of, 713.
    symptoms of, 713.
    the blood in, 376.
    treatment of, 714.
Chronic bronchitis, 961.
    cystitis, 942.
    diffuse tuberculosis, 1090.
    gastric catarrh, 856.
    interstitial nephritis, 937.
    laryngitis, 952.
    parenchymatous nephritis, 933.
    peritonitis, 921.
    tonsillitis, 812.
    tuberculosis of the lungs, 996.
Chyluria, diagnosis of, 941.
    etiology of, 941.
    symptoms of, 941.
    treatment of, 941.
Circulation in the fœtus, 19.
    in the premature infant, 295.
    post-natal changes in the, 21.
Cirrhosis of the liver, 917.
Cleft palate, 412.
Clock, the, in the nursery, 132.
Closets and drawers, 127.
Clothing, 132.
Club-foot, treatment of, 436.
Club-hand, treatment of, 436.
Colon, ascending, normal development of, 92.
    descending, normal development of, 94.
    dilatation of the, 875.
Color of fæcal dejections, 274.
Colostrum, 166.
Common carotid artery, 43.
Concussion, 738.
Condylomata, syphilitic, of the anus, 494.
Congenital disease, definition of, 19.
    form of hernia, 428.
    hydrocephalus, 409.
    malformations of the stomach, 440.
    obliteration of the intestine, 440.
    obstruction of the bile-ducts, 438.
    umbilical hernia into the cord, 425.
Constipation, atonic, 868.
    spasmodic, 868.

Contraction of the stomach, 848.
Contra-indications to maternal feeding, 159.
Convulsions, 754.
  prognosis of, 757.
  treatment of, 758.
Cord, spinal, 676.
  umbilical. *See* Umbilical cord.
Corpuscles, nucleated red, 342.
Coryza, acute, 801.
Cough, reflex, 751.
Cow, the, as a source of milk-supply, 218.
Cranium of the new-born infant, 31.
  normal development of the, 64.
Cricoid cartilage, normal development of the, 599.
Curtains in the nursery, 127.
Curvatures of the spine, 148.
Curves of the spine, 56.
Cystitis, acute, 942.
    etiology of, 942.
    prognosis of, 942.
    symptoms of, 942.
    treatment of, 942.
  chronic, 942.
    prognosis of, 943.
    symptoms of, 943.
    treatment of, 943.

D.

Dactylitis syphilitica, 496.
Defects of posture, 142.
Degeneration, acute fatty, of the new-born, 440.
Dejections, fæcal, color of, as influenced by the percentage of fat in the food, 274.
  odor of, 274.
Dental reflexes, 746.
Dentition, difficult, 794.
Dermatitis exfoliativa neonatorum, 464.
Descent of the testicle, 432.
Developmental diseases of the intestine, 858.
Diabetes insipidus, 1089.
  mellitus, 1088.
Diaphragm in the new-born infant, 41.
Diarrhœa, acute, 860.
    symptoms of, 861.
    treatment of, 861.
  prophylaxis of, 858.
Difficult dentition, 794.
Digestive disturbances in hereditary syphilis, 493.
Dilatation of the colon, 875.
  of the stomach, 848.
Diphtheria, complications and sequelæ of, 826.
Diptheria, etiology of, 821.
  incubation of, 823.
  pathology of, 823.
  prognosis of, 827.
  prophylaxis of, 828.
  symptoms of, 823.
  the blood in, 372.
  treatment of, 828.
  variations in type of, 824.
Disinfection in scarlet fever, 549.
Draughts in the nursery, 127.
Dress, 135.
Drugs in lactation, 184.
  use of, 326.
Duodenum in the new-born infant, 45.

E.

Early periods of life, topographical anatomy of, 120.
Eczema, 470.
Eliminative disturbance of the intestine, 872.
Elongation of the uvula, 816.
Empyema, 1013.
Encephalocele. 408.
Endocarditis, diagnosis of, 1033.
  etiology of, 1031.
  pathology of, 1031.
  prognosis of, 1034.
  symptoms of, 1032.
  treatment of, 1034.
Enlargement of the thyroid gland, 1096.
Enuresis, etiology of, 946.
  prognosis of, 948.
  symptoms of, 948.
  treatment of, 948.
Eosinophiles, mononuclear, 344.
  polynuclear, 344.
Epididymitis, 944.
Epiglottis, normal development of the, 59.
Epilepsy, diagnosis of, 726.
  etiology of, 724.
  prognosis of, 727.
  symptoms of, 725.
  treatment of, 727.
Epispadias, 435.
Epistaxis, 805.
Erysipelas, 512.
  ambulans, 512.
  migrans, 512.
  of the new-born, 513.
  of sucklings, 513.
  pathology of, 512.
  treatment of, 514.

Erythema, 465.
        symptoms of, 465.
        treatment of, 466.
    intertrigo, 466.
    neonatorum, 108.
    nodosum, 466.
Erythrocytes at birth, 340.
    in fœtal blood, 338.
Eustachian tube in the fœtus, 20.
    normal development of the, 66.
    post-natal changes in the, 21.
Evolution of vaccination, 151.
Exanthemata, 517.
Eye at time of birth, 24.
    in the new-born infant, 37.

F.

Face in the new-born infant, 31.
    normal development of the, 64.
Fæcal dejections, color of, 274.
    odor of, 274.
Fatty degeneration, acute, of the new-born, 440.
        diagnosis of, 440.
        etiology of, 441.
        pathology of, 441.
        prognosis of, 441.
        symptoms of, 440.
        treatment of, 442.
    infiltration of the liver, 915
Feeding, amount at each, 234.
    apparatus for, 232, 262.
    contra-indications to maternal, 159.
    difficulties of, 154.
    direct substitute, 209.
    general principles of. 153.
    general remarks on substitute, 230.
    indirect substitute, 214.
    intervals of, 181.
    maternal, 158, 159.
    method of, 285.
    mixed, 205.
    of average infants born at term, 272.
    of premature infants, 299.
Feet, care of the, 138.
    club, 436.
    in the new-born, 49.
    malformations of the, 436.
    normal development of the, 105.
    of premature infants, 293.
Femoral hernia, 430.
Fender, 130.
Fermental diseases of the intestine, diagnosis of, 881.
    etiology of, 880.
Fermental diseases of the intestine, prognosis of, 881.
    symptoms of, 880.
    treatment of, 882.
Fibrous bronchitis, 962.
Fingers, malformations of the, 436.
Fissures, 879.
Fistula, branchial, 417.
    of anus, 880.
Floor, the, in the nursery, 126.
Fœtus, blood of the, 337.
    circulation in the, 19.
    post-natal changes in the, 21.
Fontanelle, anterior, 30.
    posterior, 31.
Fontanelles, normal development of the, 63.
Food, amount of, taken, 90.
    management of, and increase in weight, tables showing, 264.
Foramen ovale in the fœtus, 20.
    post-natal changes in the, 21.
Foreign bodies in the larynx, 950.
    in the œsophagus, 834.
Formation of milk, the, 164.
Freckles, 481.
Friedreich's disease, 689.
Fruits, 286.
Functional diseases of the intestines, 860.
Functions in the new-born infant, 51.
    normal development of the, 110.
Fungus of the umbilicus, 425.
Funicular form of hernia, 428.
Furniture of the nursery, 127.
Furunculosis, treatment of, 459.

G.

Gall-bladder, normal development of the, 77.
Gastric indigestion, acute, 844.
    chronic, 845.
Gastritis, acute, 854.
    catarrhalis acuta, 854.
        pathology of, 854.
        symptoms of, 854.
        treatment of, 855
    catarrhalis chronica, 856.
        pathology of, 857.
        prognosis of, 857.
        symptoms of, 857.
        treatment of, 857.
    corrosiva acuta, 856.
    pseudo-membranosa, 856.
Glands, diseases of the cervical lymph-, 1101.
    mammary, 155.
Glossitis, 793.
Goitre, 1096.

Granules occurring in human blood, 345.
Gums in the new-born infant, 32.
  lancing of the, 796.

H.

Hæmatoma of the sterno-cleido-mastoid muscle, 416.
Hæmaturia, 940.
Hæmoglobin at birth, 340.
Hæmoglobinæmia, infectious, of the new-born, 443.
  etiology of, 445.
  literature of, 446.
  pathology of, 444.
  symptoms of, 444.
  treatment of, 446.
Hæmoglobinuria, 940.
  etiology of, 940
Hæmophilia, 451.
Hair at birth, 24.
Hand, club, 436.
Hands in the new-born infant, 49.
  malformations of the, 436.
Harelip, 409.
Hay fever, 1005.
Head at birth, 404.
  circumference of the, 60
    of, relative to the thorax, 60.
  of the premature infant, 291.
Headache, 741.
Heart, 42.
  acquired diseases of, 1027.
  congenital diseases of, 1020.
    diagnosis of, 1026.
    prognosis of, 1027.
    symptoms of, 1025.
    treatment of, 1027.
  diseases of, 1019.
  effect of scarlet fever on, 541.
  functional diseases of, 1027.
  inflammatory lesions of, 1030.
  in the new-born infant, 42.
  malformations of, 440.
  mechanical conditions of, 1030.
  normal development of, 74.
  organic diseases of, 1028.
  position of, at term, 24.
  reflexes of, 751.
  syphilitic, 490.
  weight of, 74.
Heat, animal, of premature infants, 295.
Heat-stroke, 736.
Heating the nursery, 127.
Height in the new-born infant, 49.
  normal development of, 96.
Hemorrhage from the umbilical cord, 54.
  in early life, 446.
Hemorrhoids, 880.
Hepatitis, interstitial, 917.
Hereditary ataxia, 689.
  syphilis, digestive disturbances in, 493.
    insomnia a symptom of, 494.
Hernia, 879.
  congenital form of, 428.
    umbilical, into the cord, 425.
  femoral, 430.
  funicular form of, 428.
  infantile form of, 428.
  inguinal, 427.
  treatment of, 428.
  umbilical, 426.
Herpes zoster, 478.
  diagnosis of, 479.
  symptoms of, 478.
  treatment of, 479.
Hip, congenital dislocation of the, 437.
Hives, 467.
Hydrocele, 430.
  encysted, of the canal of Nuck, 431.
    of the cord, 430.
  treatment of, 431.
Hydrocephalus, acquired internal, 642.
    diagnosis of, 643.
    prognosis of, 643.
    symptoms of, 643.
    treatment of, 643.
  congenital, 409.
  congenital internal, 634.
    diagnosis of, 636.
    operation for, 637.
    pathology of, 634.
    prognosis of, 636.
    symptoms of, 635.
    treatment of, 636.
  external, 630.
  internal, 634.
  the blood in, 375.
Hydronephrosis, 941.
  etiology of, 941.
  prognosis of, 942.
  symptoms of, 942.
  treatment of, 942.
Hygiene of the nursery, 125.
Hypertrophic rhinitis, 804.
Hypertrophy of the pharyngeal tonsil, 806.
Hypnotism, 735.
Hypospadias, 435.
Hysteria, 732.
  diagnosis of, 734.
  prognosis of, 734.

Hysteria, symptoms of, 733.
  treatment of, 734.

I.

Ichthyosis, fœtal, 482.
    prognosis of, 483.
    symptoms of, 483.
    treatment of, 483.
  neonatorum, 483.
    treatment of, 484.
Icterus, 914.
  neonatorum, 107.
  the blood in, 379.
Idiocy, 670.
  diagnosis of, 671.
  pathology of, 670.
  symptoms of, 670.
  treatment of, 671.
Ileo-colitis, 891.
    treatment of, 898.
  amœbic, 900
  chronic, 906.
  tubercular, 906.
    pathology of, 907.
    symptoms of, 907.
  typhoidal, 900.
    diagnosis of, 902.
    pathology of, 901.
    prognosis of, 903.
    symptoms of, 901.
    treatment of, 903.
Imperforate anus, 433.
Impetigo contagiosa, 458.
Impression, maternal, 404.
Incontinence of fæces, 867.
  of urine, 946.
Incubator, 301.
  Dr. Worcester's, 306.
Indigestion, 844.
  acute gastric, 844.
    diagnosis of, 844.
    symptoms of, 844.
    treatment of, 844.
  chronic gastric, 845.
    symptoms of, 845.
    treatment of, 845.
  intestinal, 862.
Infant, anatomical points of, 25.
    abdomen, 44.
    bile, 52.
    bladder, 48.
    blood, 52.
    bone-marrow, 51.
    brain, 37.
    cæcum, 47.
Infant, anatomical points of: common carotid artery, 48.
    cranium, 31.
    diaphragm, 41.
    duodenum, 45.
    ear, 37.
    Eustachian tubes, 35.
    eye, 37.
    face, 31.
    feet, 49.
    functions, 51.
    gums, 32.
    hands, 49.
    hard palate, 37.
    head, 30.
    hearing, 51.
    heart, 42.
    height, 49.
    intestines, 47.
    jaws, 32.
    kidney, 44.
    lachrymal glands, 51.
    liver, 44.
    lung, 43.
    lymphatic system, 52.
    lymph-vessels of the pharynx, 35.
    meconium, 53.
    mouth, 36.
    naso-pharynx, 32.
    neck, 30.
    pancreas, 52.
    pelvis, 48.
    petro-squamosal suture, 38.
    pulmonary artery, 48.
    pulse, 48.
    respiration, 40, 48.
    ribs, 38.
    salivary glands, 51.
    sebaceous glands, 51.
    sight, 51.
    smell, 51.
    spine, 27.
    sternum, 38.
    stomach, 45.
    supra-renal capsule, 44.
    sweat-glands, 51.
    taste, 51.
    teeth, 37.
    temperature, 48.
    thorax, 38.
    thymus gland, 42.
    tonsils, 35.
    touch, 51.
    uric acid infarction, 44.
    urine, 48, 53.
    veins, 48.

- Infant, anatomical points of: vitality, 49.
        - voice, 51.
        - weight, 49.
    - at term, 23.
    - distinction of, from child, 18.
- Infantile atrophy of the intestine, 869.
    - paralysis, pseudo-syphilitic, 496.
    - tubercular meningitis, 612.
- Infantilism, 505.
- Infectious hæmoglobinæmia of the new-born, 446.
- Inflammatory diseases of the intestines, 887.
- Influenza, epidemic, 1092.
    - diagnosis of, 1093.
    - prognosis of, 1093.
    - treatment of, 1093.
- Insanity, 731.
- Insolation, 736.
    - diagnosis of, 737.
    - prognosis of, 737.
    - treatment of, 737.
- Insomnia a symptom of hereditary syphilis, 494.
- Inspection of the mouth, 325.
    - of the sick child, 320.
- Interstitial hepatitis, 917.
    - keratitis, 506.
- Intertrigo, 132.
- Intestinal contents, 858.
        - in premature infants, 294.
    - discharges, 117.
    - indigestion, 862.
        - chronic, 863.
- Intestine, chronic functional diseases of the, 863.
    - congenital obliteration of the, 440.
    - developmental, 858.
    - diseases of the, 836.
    - eliminative disturbances of the, 872.
    - fermental, 880.
    - functional, 860.
    - infantile atrophy of the, 869.
    - inflammatory, 887.
    - in the new-born infant, 47.
    - mechanical, 875.
    - new growths of the, 887.
    - normal development of the, 91.
    - organic, 875.
    - polypi of the, 880.
    - prolapse of the, 879.
    - tubercular, 861.
- Intra-cranial syphilis, 668.
    - tumors, 662.
- Intussusception, diagnosis of, 877.
    - prognosis of, 877.
- Intussusception, symptoms of, 877.
    - treatment of, 878.

J.

- Jaws in the new-born infant, 32.

K.

- Kaposi's disease, 481.
- Keratitis, interstitial, 506.
- Kidney, acute hyperæmia of the, 930.
    - amyloid infiltration of the, 938.
    - diseases of the, 927.
        - acquired, 927.
        - congenital, 927.
    - effect of scarlet fever on the, 537, 580.
    - in the new-born, 44.
    - malignant growths and enlargements of the, 939.
    - normal development of the, 78.
    - of premature infant, 295.
    - passive hyperæmia of the, 931.
    - syphilitic, 490.
    - tumors of the, 939.
- Killian's observations on the tonsils, 36.
- Knee, congenital dislocation of the, 487.

L.

- Laboratory, milk, 245.
- Lachrymal glands in the new-born infant, 51.
    - normal development of the, 110.
- Lactation, cases of disturbed, 190.
    - diet of, 182.
    - disturbed, 184.
    - drugs during, 184.
    - exercise during, 184.
    - management of disturbed, 188.
    - menstruation, effect of, on, 185.
    - pregnancy, effect of, on, 186.
    - prolonged, 205.
    - regimen of, 182.
- Lancing the gums, 796.
- Laryngitis, acute, 951.
        - diagnosis of, 952.
        - symptoms of, 951.
        - treatment of, 952.
    - chronic, 952.
    - pseudomembranous, 953.
- Laryngospasmus, 949.
- Larynx, diseases of the, 949.
    - foreign bodies in the, 950.
    - new growths of the, 949.
    - normal development of the, 59.

Larynx, œdema of the, 950.
  reflex phenomena of the, 748.
Legs, 146.
Leiter's coil, 599.
Length of spine, normal development of, 55.
Lentigo, 481.
Leptomeningitis, 595.
Lesions of the mouth, syphilitic, 494.
Leucæmia, 351.
Leucocytes at birth, 340.
  in fœtal blood, 338.
  large mononuclear, 343.
  percentages of various, in normal blood, 346.
  polynuclear, 343.
  small mononuclear, or lymphocytes, 343.
  transitional forms of, 343.
  varieties of, 343.
Leucocytosis, 350.
Lichen, 481.
Liver, acute yellow atrophy of the, 915.
  amyloid, 916.
  at end of term, 24.
  cirrhosis of the, 917.
  diseases of the, 914.
  fatty infiltration of the, 915.
  icterus, 914.
  in the new-born infant, 44.
  normal development of the, 77.
  syphilitic, 489.
  tuberculosis of the, 915.
Lobar pneumonia, 980.
Locomotor ataxia, 689.
Lungs, acute tuberculosis of the, 993.
  chronic tuberculosis of the, 996.
  diseases of the, 954.
  in the new-born infant, 43.
  normal development of the, 75.
  reflex of the, 750.
  syphilitic, 490.
Lymphatic system in the new-born infant, 52.
  normal development of the, 111.
Lymph-glands, diseases of the cervical, 1101.

M.

Macroglossia, 794.
Malaria, diagnosis between, and meningitis, 609.
  diagnosis of, 387.
  pathology of, 386.
  prognosis of, 388.
  symptoms of, 388.
  the blood in, 380.
    method of examination of, 380.
  treatment of, 390.
Malformations about the rectum, 433.
  congenital, of the stomach, 440.
  of the heart and blood-vessels, 440.
Malignant growths and enlargements of the kidney, 939.
Mammary gland, relation of micrococci to inflammation of the, 162.
Marasmus (infantile atrophy), 869.
Mastitis, 162, 418.
Maternal feeding, contra-indications to, 159.
  impression, 404.
Mattress, 127.
Maxillary bones, normal development of the, 67.
Measles, complications and sequelæ of, 584.
  desquamation of, 578, 582.
  diagnosis of, 578.
  efflorescence of, 577, 582.
  incubation of, 576.
  pathology of, 574.
  prodromata of, 576.
  prognosis of, 578.
  symptoms of, 576.
  the blood in, 371.
  treatment of, 578.
  variations in type in, 581.
Mechanical diseases of the intestines, 875.
Meconium, 23, 53.
Meckel's diverticulum, 426.
Melanoderma lenticularis progressiva, 481.
Meningitis, cerebro-spinal, 692.
  infantile tubercular, 612.
    prognosis of, 612.
    treatment of, 612.
  non-tubercular, 595.
    pathology of, 596.
    symptoms of, 598.
    treatment of, 599.
  tubercular, 603.
    diagnosis of, 608.
    differential diagnosis of, 609.
    pathology of, 605.
    symptoms of, 605.
    the blood in, 374.
Meningo-myelocele, 420.
Menstruation during lactation, 185.
Method of examining a sick child, 318.
Microcephalus, 672.
Micrococci, relation of, to inflammation of the breast, 162.
Microglossia, 794.
Migraine, 742.
Milk, analysis of bad, 187.
    cow's, 237.
    normal, 187.
    over-rich, 187.

Milk, analysis of poor, 187.
    ash of, 174, 241.
    ass's, 178.
    average analysis of human, 172.
    bacteriological examination of cow's, 237.
        of human, 180.
    bacteriology of cow's, 242.
    chemical analysis of, 172.
    clinical examination of human, 169.
    constituents and properties of, 165.
    cow's, 178.
    cows producing suitable, 225.
    estimate of the proteids of, 171.
    fat of, 173.
        and proteids of, 170.
    -house, the, 225.
    human, 167.
        quality of, 168.
        quantity of, 167.
    -laboratory, the, 245.
    methods of examination of, 170.
    microscopic examination of, 169.
    nervous disturbances affecting the, 165.
    peptonized, 282.
    production of, 155, 164.
    reaction of cow's, 223, 286.
        of human, 172.
    -room, 247.
    salts of, 169.
    specific gravity of, 169, 170, 172.
    sugar of, 170, 174.
    tester, Babcock, 249.
    transportation of, 263.
    variations in human, 177.
    water of, 173.
Mind, normal development of the, 110.
Mirror writing, 673.
Miscarriage caused by syphilis, 503.
Modifying-room, 249.
Molluscum contagiosum, 459.
Mononuclear eosinophiles, 344.
    neutrophiles, 344.
Mother, the nursing, 160.
Mouth, diseases of the, 774.
    in the new-born infant, 36.
    inspection of the, 325.
    normal development of the, 67.
    syphilitic lesions of the, 494.
Mucous polypus, 805.
Multiple cerebro-spinal sclerosis, 691.
    neuritis, 704.
Mumps, 1104.
Mutton broth, 287.
Myelitis, 676.
Myelocytes, 344.
Myopathic progressive muscular atrophy, 763.
Myopathies, 763.
Myotonia congenita, 773.
Myxœdema, 1096.

N.

Nævus, 454.
Napkins, 135.
Naso-pharynx, diseases of the, 801, 805.
    in the new-born infant, 32.
    normal development of the, 65.
Neck in the new-born infant, 30.
    normal development of the, 59.
Nephritis, acute, 932.
    chronic interstitial, 937.
    parenchymatous, 933.
    the blood in, 376.
Nervous disturbances affecting the milk, 165.
    system, diseases of the, 590.
        functional, 732.
        presumably organic, 711.
Nettle-rash, 467.
Neuralgia, 709.
Neuritis, multiple, 704.
    diagnosis of, 705.
    etiology of, 704.
    pathology of, 704.
    prognosis of, 705.
    symptoms of, 704.
    treatment of, 705.
Neuropathic progressive muscular atrophy, 763.
New-born, abdomen in the, 44.
    acute fatty degeneration of the, 440.
    cæcum in the, 47.
    cranium of the, 31.
    diaphragm in the, 41.
    duodenum in the, 45.
    feet in the, 49.
    functions in the, 51.
    gums in the, 32.
    hands in the, 49.
    head in the, 30.
    height in the, 49.
    infectious hæmoglobinæmia of the, 446.
    intestine in the, 47.
    jaws in the, 32.
    kidney in the, 44.
    lachrymal glands in the, 51.
    liver in the, 44.
    lungs in the, 43.
    lymphatic system in the, 52.
    mouth in the, 36.
    naso-pharynx in the, 32.
    neck in the, 30.

New-born, pancreas in the, 52.
  paralysis of the, 706.
  pelvis in the, 48.
  petro-squamosal suture in the, 38.
  pulse in the, 48.
  respiration in the, 40, 48.
  ribs in the, 38.
  salivary glands in the, 52.
  sebaceous glands in the, 28, 51.
  sight in the, 51.
  smell in the, 51.
  sternum in the, 38.
  stomach in the, 45.
  supra-renal capsules in the, 44.
  sweat-glands in the, 51.
  taste in the, 51.
  teeth in the, 37.
  temperature in the, 48.
  thorax in the, 38.
  thymus gland in the, 42.
  touch in the, 51.
  uric acid infarction in the, 44.
  urine in the, 53.
  uterus in the, 48.
  veins in the, 48.
  vitality in the, 49.
  voice in the, 51.
  weight in the, 49.
New growths of the intestine, 887.
  of the larynx, 949.
  of the stomach, 854.
Night-dress, 136.
Night-terrors, 745.
Nipples, 161.
  artificial, 233.
Noma, 788.
Non-tubercular meningitis, 595.
Normal development: abdomen, 77.
  bile, 111.
  bladder, 78.
  blood, 111.
  blood-vessels, 75.
  bone marrow, 107.
  brain, 64.
  cæcum and ascending colon, 92.
  cord, 54, 110.
  descending colon, 94.
  ear, 65.
  Eustachian tube, 66.
  face and cranium, 64.
  feet, 105.
  functions, 110.
  gall-bladder, 77.
  hard palate, 67.
  head, 60.
  heart, 74.
Normal development: height, 96.
  infantile skeletons, 118.
  intestinal discharges, 117.
  intestine, 91.
  kidney, 78.
  lachrymal glands, 110.
  liver, 77.
  lungs, 75.
  lymphatic system, 111.
  maxillary bone, 67.
  mental impressions, 110.
  mouth, 67.
  naso-pharynx, 65.
  neck, 59.
  pancreas, 77, 111.
  petro-squamosal suture, 65.
  pharyngeal tonsil, 66.
  pulse, 94.
  respiration, 72, 96.
  salivary glands, 111.
  sigmoid flexure, 93.
  skin, 107.
  spine, 55.
  spleen, 77.
  sternum, 70.
  stomach, 79.
  supra-renal capsules, 78.
  sweat-glands, 110.
  teeth, 68.
  temperature, 94.
  thorax, 70.
  thymus gland, 72.
  thyroid, 111.
  topographical anatomy of the early periods of life, 120.
  urine, 111.
  vermiform appendix, 92.
  voice, 110.
  weight, 97.
Northrup on the lung in the new-born infant, 43.
Nose, diseases of the, 801.
  normal development of the, 65.
Nuck, encysted hydrocele of the canal of, 431.
    treatment of, 431.
Nucleated red corpuscles, 342.
Nursery, hygiene of the, 125.
Nursery-maids, 141.
Nursing mother, the, 160.
Nutritive period, second, 284.
  third, 287.

O.

Oats, 281.
Obstruction, congenital, of the bile-ducts, 438
Occlusion of the vagina, 435.

Pharynx, diseases of the, 801, 809.
Phimosis, 946.
Physiological albuminuria, 927.
Phlebitis and arteritis umbilicalis, 425.
Pillow, the, 127.
Pityriasis maculata circinata, 480.
  rosea, 480.
  rubra, 480.
Placenta the lung of the fœtus, 19.
Pleura, diseases of the, 1007.
Pleurisy, 1007.
    diagnosis of, 1009.
    pathology of, 1007.
    prognosis of, 1011.
    symptoms of, 1008.
    treatment of, 1012.
  purulent, 1013.
Pneumonia, acute tubercular broncho-, 994.
    diagnosis of, 995.
    prognosis of, 995.
    symptoms of, 995.
    treatment of, 995.
  lobar, 980.
    complications of, 986.
    diagnosis of, 985.
    etiology of, 980.
    gangrene in, 986.
    pathology of, 981.
    prognosis of, 986.
    symptoms of, 981.
    treatment of, 987.
  the blood in, 372.
Poliomyelitis, 676.
    diagnosis of, 678.
    differential diagnosis of, 679.
    pathology of, 676.
    symptoms of, 677.
    treatment of, 681.
  anterior, diagnosis between, and meningitis, 609.
Polynuclear eosinophiles, 344.
Polypi of the rectum, 880, 887.
Polypus, mucous, 805.
Porencephalia, definition of, 650.
Port-wine mark, 454.
Position of the heart at term, 24.
Posture, defects of, 142.
Powder, 130.
Pregnancy during lactation, 186.
Premature infants, 288.
  amount of food at each feeding of, 299.
  amylolytic function of, 294.
  animal heat of, 295.
  circulation of, 295.
Premature infants, cleansing and clothing of, 301.
  composition of food for, 299.
  digestion of sugar of, 295.
  Dr. Worcester's incubator for, 306.
  fat and proteid digestion of, 295.
  feet of, 293.
  gastric capacity of, 293.
  head, thorax, and abdomen of, 291.
  incubators for, 301.
  intervals of feeding of, 299.
  intestinal contents in, 294.
  kidney of, 295.
  pathology of the blood in, 348.
  pulse, temperature, and respiration of, 297.
  skin of, 292.
  sweat-glands of, 292.
  weighing of, 301.
  weight of, 290.
Primary anæmias, 355.
Progressive muscular atrophy, 763.
    myopathic, 763.
    neuropathic, 763.
  pernicious anæmia, 356.
Prolapse of the rectum, 879.
Prolonged lactation, 205.
Proteids, coagulation of, 239.
  estimation of the, 171.
  in human milk, 174.
    and cow's milk, 238.
Prurigo, 478.
    treatment of, 478.
  mitis infantilis, 477.
    symptoms of, 477.
    treatment of, 478.
Pseudo-hypertrophic muscular paralysis, 768.
    diagnosis of, 769.
    treatment of, 771.
  -leukæmic anæmia of infants, 359.
  -membranous gastritis, 856.
    laryngitis, 953.
  -syphilitic infantile paralysis, 496.
Psoriasis, 475.
  treatment of, 476.
Puberty, age of, 18.
Pulmonary artery, 43, 75.
Pulse in the new-born infant, 48.
  normal development of the, 94.
  of premature infant, 297.
Purpura, 1086.
Purulent ophthalmia, 415.
  pleurisy, 1013.
Pyelitis, 938.
Pyelo-nephritis, 938.

R.

Ranula, 414.
Rectum, malformations about the, 433.
        imperforate anus, 433.
    prolapse of the, 879.
    reflex of the, 752.
Red corpuscles, nucleated, 342.
Reflex cough, 751.
    of the bladder, 752.
    of the heart, 751.
    of the rectum, 752.
    of the stomach, 752.
    of the vagina, 752.
    phenomena of the larynx, 748.
        of the lungs, 750.
Reflexes, dental, 746.
Respiration in the new-born infant, 40, 48.
    normal development of, 72, 96.
    of the premature infant, 297.
    of the sick child, 320.
Retarded speech, 740.
Retro-pharyngeal abscess, diagnosis of, 817.
    pathology of, 817.
    prognosis of, 818.
    symptoms of, 817.
    treatment of, 818.
Rhachitic anæmia. 368.
Rhachitis. congenital, 1071.
    diagnosis of, 1073.
    etiology of, 1065.
    pathology of, 1066.
    prognosis of, 1073.
    symptoms of. 1073.
    treatment of, 1073.
Rheumatism, diagnosis between, and tubercular meningitis, 611.
    etiology of. 1080.
    pathology of, 1080.
    prognosis of. 1081.
    symptoms of. 1081.
    treatment of. 1081.
Rhinitis. acute, 801.
    atrophic, 804.
    hypertrophic, 804.
    purulent, 803.
Ribs in the new-born infant. 38.
    movement of the, 39.
Ringworm. 460.
Rose cold, 1005.
Rötheln, 588.
Rubella, 588.
Rubeola, 573.
Rugs in the nursery. 126.

S.

Salivary glands in the new-born infant. 52.
    normal development of the. 111.
Scabies, 456.
    treatment of, 457.
Scales, 128.
    systematic and frequent use of. 99.
Scarlet fever, 532.
    cervical glands in. 537.
    complications of, and their treatment, 555.
    contagium of, 533.
    desquamation of. 544.
    diagnosis of, 545.
    ear in, 536.
    efflorescence of, 542.
    heart in, 541.
    incubation of. 542.
    isolation and disinfection in, 549.
    kidney in, 537.
    malignant form of, 572.
    pathology of. 534.
    prodromata of, 542.
    prognosis of, 545.
    symptoms of. 542.
    the blood in. 371.
    throat in. 535.
    treatment of, 545.
    urine of. 544.
    variations in the benign form of. 552.
School. 142.
Sclerema neonatorum. 453. 484.
    blood in. 379.
    symptoms of. 454.
    treatment of. 453.
Scleroderma. 484.
Sclerosis. definition of. 650.
    multiple cerebro-spinal. 691.
        diagnosis of. 692.
        etiology of. 691.
        pathology of. 691.
        prognosis of. 692.
        symptoms of. 691.
        treatment of. 692.
Scorbutus. blood in. 379.
    diagnosis of. 1077
    etiology of. 1075.
    pathology of. 1075
    prognosis of, 1078.
    symptoms of, 1076.
    treatment of. 1078.
Sebaceous glands in the new-born infant. 23, 51.
Seborrhœa capitis, 132.
Second nutritive period. 284.

Secondary anæmias, 365.
Sensitive spine, 744.
Separating-room, 248.
Separator, 248.
Shirt, 135.
Shoes, 138.
Sight in the new-born infant, 51.
Sigmoid flexure, normal development of the, 93.
Simulated diseases, 735.
Sinuses, cerebral, thrombosis of, 626.
Skeletons, infantile, 118.
Skin, 24.
  diseases of the, 455.
  normal development of the, 107.
  of premature infants, 292.
Sleep, 14
Smell in the new-born infant, 51.
Soap, 129.
Speech, retarded, 740.
Spina bifida, 419.
Spinal curves, 27.
  meningocele, 420.
Spine at time of birth, 26.
  curvatures of the, 143.
  curves of the, 56.
  flexibility of the, 56.
  length of the, 55.
  normal development of the, 55.
  paralysis caused by caries of the, 688.
  prominent processes of the, 58.
  sensitive, 744.
  surface anatomy of the, 58.
Spleen, diseases of the, 918.
  normal development of the, 77.
  position of the, at birth, 25.
  syphilitic, 489.
Sponges, 129.
Statistics of vaccination, 147.
Sterilizer, 252.
  home, 276.
Sterno-cleido-mastoid muscle, hæmatoma of the, 416.
Sternum, depressed, 418.
  in the new-born infant, 38.
  normal development of the, 70.
  ossification of the, 39, 71.
  prominent, 419.
Still, 249.
Stockings, 136.
Stöhr's observations on the tonsils, 36.
Stomach, capacity of the, 79.
  congenital malformations of the, 440.
  contraction of the, 848.
  developmental diseases of the, 840.
Stomach, diagnosis between diseases of the, and meningitis, 609.
  dilatation of the, 848.
    diagnosis of, 850.
    pathology of, 849.
    prognosis of, 851.
    symptoms of, 850.
    treatment of, 851.
  diseases of the, 609, 836.
    diagnosis of, 838.
    etiology of, 837.
    general bacteriology of, 837.
    pathology of, 837.
    symptomatology of, 838.
    treatment of, 839.
  functional diseases of the, 840.
  inflammatory diseases of the, 854
  in the new-born infant, 45.
  nervous diseases of the, 840.
  new growths of the, 854.
  normal development of the, 79.
  of the premature infant, 293.
  organic diseases of the, 848.
  reflex of the, 752.
  ulcers of the, 853.
Stomatitis catarrhalis, 776.
    pathology of, 777.
    prognosis of, 777.
    symptomatology of, 777.
    treatment of, 778.
  gangrænosa, 788.
    diagnosis of, 789.
    etiology of, 788.
    prognosis of, 789.
    symptoms of, 789.
    treatment of, 789.
  herpetica, 779.
    etiology of, 779.
    pathology of, 780.
    prognosis of, 780.
    symptomatology of, 780.
    treatment of, 780.
  hyphomycetica, 785.
  mycetogenetica, 784.
    diagnosis of, 786.
    pathology of, 785.
    prognosis of, 786.
    symptoms of, 786.
    treatment of, 786.
  ulcerosa, 781.
    diagnosis of, 783.
    etiology of, 781.
    pathology of, 782.
    prognosis of, 783.
    symptoms of, 782.
    treatment of, 783.

Sun and windows, 125.
Supra-renal capsules, affections of the, 940.
  in the new-born infant, 44.
  normal development, 78.
Sweat-glands in the new-born infant, 51.
  normal development, 110.
  of premature infants, 292.
Sweet whey, 282.
Syphilis, congenital, with enlarged spleen, 867.
  dentition in, 493.
  diagnosis between, and meningitis, 611.
  diagnosis of, 497.
    of hereditary, 496.
  digestive disturbances in, 493.
  early manifestations of hereditary, 491.
  heart in, 490.
  hæmorrhagica neonatorum, 494.
  hereditary, 487.
  insomnia in, 494.
  intra-cranial, 668.
    diagnosis of, 669.
    pathology of, 668.
    prognosis of, 670.
    symptoms of, 669.
    treatment of, 670.
  kidney in, 490.
  later manifestations of hereditary, 504.
  liver in, 489.
  lungs in, 490.
  miscarriage in, 503.
  mouth in, 494.
  nose in, 494.
  osseous system in, 490.
  pancreas in, 490.
  pathology of, 489.
  spleen in, 489.
  teeth in, 506.
  testicle in, 490.
  throat in, 490.
  thymus gland in, 490.
  treatment of, 498.
    of later symptoms of, 506.
  upper air-passages in, 490.
Syphilitic dactylitis, 496.
  iritis, 493.
  kidney, 490.
  lesions of the mouth, 494.
  liver, 489.
  lung, 490.
  onychia, 506.
  osteochondritis, 495.
  pancreas, 490.
  spleen, 489.
Syphilitic teeth, 508.
  testicle, 490.
  throat, 490.
  thymus gland, 490.
Syringomyelia, 690.
Syringo-myelocele, 420.

T.

Tables showing management of food and increase in weight, 264.
Tæniæ, 911.
Tapeworms, 911.
Taste in the new-born infant, 51.
Teeth, effect of syphilis on the, 493, 506.
  in the new-born infant, 37.
  normal development of the, 68.
  syphilitic, 508.
Temperature in the new-born infant, 48.
  normal development of, 94.
  of premature infant, 297.
  of the sick child, 320.
Temporary amnesia, 739.
Testicles at end of term, 24.
  descent of the, 432.
  syphilitic, 490.
  tubercular disease of the, 945.
  tumors of the, 432, 946.
Tetanus neonatorum, 452.
  etiology of, 452.
  symptoms of, 452.
  treatment of, 452.
Tetany, 744.
Third nutritive period, 287.
Thorax in the new-born infant, 38.
  normal development of the, 70.
  of the premature infant, 291.
Throat, examination of the, 323.
  in scarlet fever, 535, 556.
  syphilitic, 490.
Thrombosis of the cerebral sinuses, 626.
Thrush, 785.
Thymus gland in the new-born infant, 42.
  normal development of the, 72.
  syphilis of the, 490.
Thyroid gland, diseases of the, 1095.
  enlargement of the, 1096.
  hyperæmia of the, 1095
  normal development of the, 111.
Thyroiditis, 1096.
Tinea, 460.
  circinata, 460.
    treatment of, 461.
  favosa, 461.

Tinea favosa, treatment of, 461.
    tonsurans, 460.
    trichophytina, 460.
Tongue-tie, 414.
Tonsil, pharyngeal, 35, 66.
    hypertrophy of the, 806.
        diagnosis of, 806.
        etiology of, 806.
        pathology of, 806.
        prognosis of, 808.
        symptoms of, 806.
        treatment of, 809.
    Killian's observations on the, 36.
Tonsillitis, acute, 809.
        diagnosis of, 811.
        prognosis of, 811.
        symptoms of, 810.
        treatment of, 811.
    chronic, 812.
        pathology of, 812.
        prognosis of, 813.
        symptoms of, 813.
        treatment of, 813.
Tonsils, faucial, 35.
Touch in the new-born infant, 51.
Toys, 128.
Trachea, diseases of the, 953.
Tremor, 762.
Tub, 129.
Tubercular diseases of the intestines, 861.
        of the testicle, 945.
    meningitis, 603.
        infantile, 612.
    peritonitis, 921.
Tuberculosis, acute miliary, 1090.
    chronic diffuse, 1090.
    chronic, of the lung, 996.
        diagnosis of, 996.
        prognosis of, 997.
        symptoms of, 996.
        treatment of, 997.
    of the liver, 915.
    of the lung, 993.
        etiology of, 993.
        pathology of, 993.
    of the skin, 485.
        treatment of, 486.
    the blood in miliary, 374.
Tumors, intra-cranial, 662.
        pathology of, 662.
        symptoms of, 663.
    of the kidney, 939.
    of the testicle, 946.
Typhoid fever, diagnosis between, and meningitis, 610.
    the blood in, 370.

U.

Ulcers of the stomach, 858.
Umbilical arteries in the fœtus, 20.
        post-natal change in the, 21.
    cord, hemorrhage of, 54.
        normal condition of, 24, 54.
        normal development of, 110.
    hernia, 426.
        congenital, into the cord, 425.
    vein, post-natal change in the, 21.
Umbilicus, fungus of the, 425.
Uric acid infarction in the new-born infant, 44.
Urine in the new-born infant, 53.
    in scarlet fever, 544.
    normal development of the, 111.
    of adolescence, 114.
Urticaria, treatment of, 467.
Uterus in the new-born infant, 48.
Uvula, elongation of the, 816.

V.

Vaccination, abnormal effects of, 152.
    efflorescence of, 528.
    evolution of, 151.
    method of, 150.
    statistics of, 147.
Vaccinia, 152, 528.
    efflorescence of, 528.
Vagina, occlusion of the, 485.
    reflex of the, 752.
Varicella, 524.
    complications of, 526.
    diagnosis of, 527.
    efflorescence of, 525.
    gangrenous, 526.
    incubation of, 525.
    pathology of, 525.
    prodromata of, 525.
    prognosis of, 526.
    symptoms of, 525.
    treatment of, 531.
Variola, 516.
    blood in, 371.
    confluent, 521.
    complications of, 522.
    desquamation of, 521.
    diagnosis of, 522.
    discrete, 520.
    efflorescence of, 521.
    hemorrhagic, 521.
    incubation of, 520.
    modified form of, 522.
    pathology of, 518.

Variola, prodromata of, 520.
  symptoms of, 520.
  treatment of, 524.
Veins in the new-born infant, 43.
Ventilation in the nursery, 127.
Ventilator, 248.
  window, 127, 130.
Vermiform appendix, normal development of the, 92.
Vernix caseosa, description of, 23.
Verruca, 480.
Vertebræ, position of, in the new-born infant, 37.
Vertigo, 748.
Vitality in the new-born infant, 49.
Voice in the new-born infant, 51.
  normal development of the, 110.
Volvulus, 876.
Vomiting, 840.
  diagnosis of, 842.
  etiology of, 841.
  prognosis of, 842.
  symptoms of, 841
  treatment of, 842.
Vulvo-vaginitis, 943.
  pathology of, 943.
Vulvo-vaginitis, prognosis of, 944.
  symptoms of, 944.
  treatment of, 944.

W.

Walls in the nursery, 126.
Warts, 480.
Wash-room, 253.
Weaning, 206, 284.
Weighing of premature infants, 301.
Weight in the new-born infant, 49.
  normal development of, 97.
  of premature infant, 290.
Wet-nurses, 209, 211.
  diet of, 212.
Wheat, 282.
Whey, sweet, 282.
Whooping-cough, 998.
Windows, sun, and in the nursery, 125.
Worcester, Dr. Alfred, brooder, 309.
  incubator, 306.
Writing, mirror, 673.

Y.

Yellow atrophy, acute, of the liver, 915.

THE END.

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

OCT 4 1990
SEP 2[illegible] 1990

OCT - 1 1990
PC SEP 19 1990

P45 Rotch, Thomas Morgan
R842 Pediatrics
1896 17852

NAME	DATE D